Orbital Fractures in the Pediatric Population

12

Peter J. J. Gooris, Maarten P. Mourits, Gertjan Mensink, and J. Eelco Bergsma

Learning Objectives

- Orbital fractures in children exhibit different features in children as compared to the adult population: children are no small adults.
- A white eye after blunt trauma in a child can be deceiving. A trapdoor fracture may betray itself only by a limitation of elevation. An attempt to elevate the eyes may result in a cardiac arrest.
- Such a trapdoor trauma requires instant intervention.
- Nausea caused by trapdoor fractures may be confused with concurrent head injury i.e., concussion.
- A growing skull fracture can be a late complication in case of orbital roof fracture involvement.

P. J. J. Gooris (✉)
Department of Oral and Maxillofacial Surgery, Amphia Hospital Breda, Breda, The Netherlands

Amsterdam University Medical Centers, Amsterdam, The Netherlands

Department of Oral and Maxillofacial Surgery, University of Washington, Seattle, WA, USA

M. P. Mourits
Amsterdam University Medical Centers, Location AMC, Amsterdam, The Netherlands

G. Mensink
Department of Oral and Maxillofacial Surgery, Amphia Hospital Breda, Breda, The Netherlands

University Medical Center Leiden, Leiden, The Netherlands

J. E. Bergsma
Department of Oral and Maxillofacial Surgery, Amphia Hospital Breda, Breda, The Netherlands

Amsterdam University Medical Centers, Amsterdam, The Netherlands

Acta dental school, Amsterdam, The Netherlands

Introduction

Epidemiology

In children, a traumatic injury to the craniofacial skeleton can result in facial fractures but may also affect and disturb facial growth [1, 2]. Fortunately, facial fractures in children are relatively rare and are less common than in the adult population [2, 3]. The incidence of facial fractures varies with age. Only approximately 1% of all facial fractures occur in children under the age of 1 year. The majority of fractures are observed in children within the age group of 13–18 years of age; boys are twice as much involved in facial fractures as girls are [1–6]. In children, under the age of 16, the overall incidence of orbital wall fractures is 5–25% of all facial fractures, which is lower than the incidence in the adult population

P. J. J. Gooris et al. (eds.), *Surgery in and around the Orbit*, https://doi.org/10.1007/978-3-031-40697-3_12

(around 50%) [1–5]. As explained further on, a skull fracture may occur in the very young and can include a fracture of the anterior skull base resulting in intracranial injury [1, 2].

A contiguous orbital roof fracture is more common in very young children [7–9]. However, the incidence of orbital roof fracture is likely to be underreported [8, 9]. Although fractures of the orbital floor and medial wall are diagnosed at any age, there is an increase of isolated fractures of the orbital floor with age and development [3, 5]. In case of an orbital floor fracture in children, more than 50% entrapment of orbital soft tissue is encountered [1–6, 10]. Not surprisingly, the mechanism of the craniofacial impact, differs with age (Table 12.1):

Growth and Development and Subsequent Different Kind of Patterns of Injury

In the pediatric population, there is a relatively high proportion of cancellous, richly vascular bone and growing sutures of cartilaginous structure which is responsible for the characteristic of elasticity of the young growing bone. In adults, the bone becomes more compact, dense and rigid. The craniofacial skeleton undergoing mineralization, most profoundly at the age 2–3 years, changes from an elastic to a rigid structure with age. The elastic structure still has the intrinsic capacity to deform or buckle instead of fracture when force is applied resulting in less fractures.

Depending on age, the size and shape, the anatomy-proportion of the skull changes. The orbital floor itself deepens and becomes less steep from lateral to medial with age. The lowest point along the orbital floor shifts posteriorly [3, 4]. Neurocranial growth is continuous and stimulated by the enlarging brain. There is a preponderance of this growth mainly in the first 2 years, after which it gradually decreases over the following years. Facial skeletal growth is discontinuous, is multifactorial and varies in location and direction [2, 3]. Orbital depth reaches 90% of adult dimensions at age 6 and 95% at age 12, which is analogous to the cranial growth [3, 11, 12]. The fastest growth of the orbit is within 12–24 months; after age 6, the rate of expansion declines (Table 12.2) [11, 12].

Pneumatization of the paranasal sinuses develops during childhood. While in utero, the sinuses and nasal cavity are in fact mucosal tissue within cancellous bone and form one single structure. During development, the ethmoid, frontal and maxillary sinus subdivide in a predictable sequence [2] (Table 12.3).

The *frontal sinus* pneumatization evolves at age 7, completes before adulthood at around age 16 [1, 2, 7, 9]. Radiographically, this pneumatization becomes "visible" at age 8. The lack of pneumatization in young children allows for more direct transmission of force to the supraorbital rim which extends directly posterior to the anterior cranial base and orbital roof with subsequent an increased risk of an orbital roof fracture as a result [2, 3, 6, 9]. Once frontal sinus pneumatization has been completed, less force is directly transmitted to the anterior cranial base and impact forces are dissipated.

Table 12.1 Type of impact related to age

Young child	Impact as a result of daily activities, like a fall. More skull fractures and orbital roof fractures
Older child	Impact as a result of sport, traffic or violence and sport related injuries, results in orbital floor and medial wall fractures

Table 12.2 Average orbital volume (mL) [12]

At birth	9–15
6 years	20
Mature age	25–28

Table 12.3 Growth and development [1, 2]

	Years of age of development	Adult size reached at (year)
Maxillary sinus	0–3 and 7–12	16
Ethmoid sinus	1–12	12
Frontal sinus	7–16	16

Ethmoid air cells already present at birth gradually grow to an adult size at age 12 (Fig. 12.1). During the continuous pneumatization and subsequent expansion, the medial orbital wall becomes progressively thinner as the lamina papyracea and thus more susceptible for orbital wall fracture in adulthood [2, 3].

Maxillary sinus development is biphasic. Its growth peaks at age 0–3 and at age 7–12. The maxillary sinus is initially located medial to the orbit, by age 4, it develops more infero-laterally, expands at age 12 and reaches its adult size at age 16. Eventually, both the changes in bone morphology of the craniofacial skeleton and the sinus development during growth will affect how the force of impact will be transmitted and how this will result in a variable fracture pattern (Table 12.4).

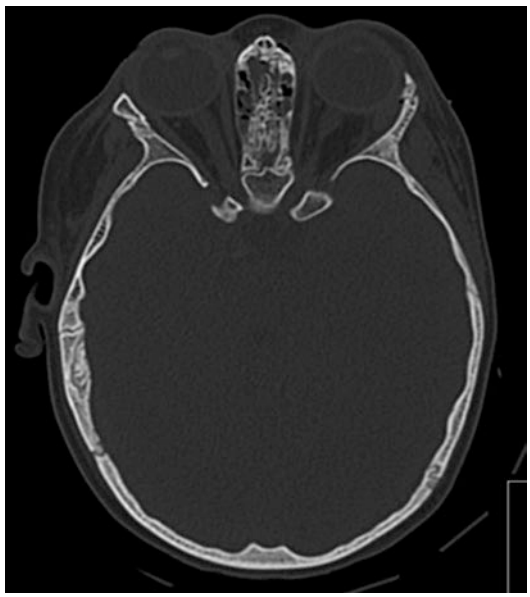


Fig. 12.1 The early presence of ethmoid air cells, axial view

In the group till the age of 7, due to a higher cranial to face ratio (Table 12.5) which results in a proportionately larger neurocranium i.e., more exposure of the frontal bone, head trauma will more often result in a skull and orbital roof fracture rather than into a fracture of the facial complex [2, 5, 7–9] (Figs. 12.2 and 12.3).

With exceeding age (older than 7), isolated fractures of the lateral wall are declining in frequency because of its increase in thickness and non-sinus boundary.

The probability of fractures of the orbital floor does not exceed that of the orbital roof until age 7 [1, 2, 4, 7, 10]. It is said that the orbital floor fracture only "starts" at age 3–4 because of the pneumatization of the maxillary sinus [2, 9, 10]. Unerupted maxillary dentition in the undeveloped maxillary sinus also resists orbital floor fractures in young children, especially under the age of 7 [2] (Figs. 12.4, 12.5 and 12.6).

Table 12.4 Relation between age and facial fracture pattern

Birth—till age 7	Orbital roof > orbital floor	Incomplete pneumatization frontal sinus High cranium-face ratio presence unerupted maxillary dentition
Age 7—adulthood	Orbital floor > orbital roof Increase medial wall	Completion maxillary sinus pneumatization Eruption maxillary dentition completion ethmoid sinus pneumatization

Table 12.5 Anatomical changes during maturation "size ratio" in growth and adult size

	Ratio neurocranium:face	% of Adult size neurocranium	% of Adult size face
Birth	8:1	35%	25%
2 years		75%	70%
5 years	4:1		80%
10 years		95%	
Mature	2:1	100%	100%

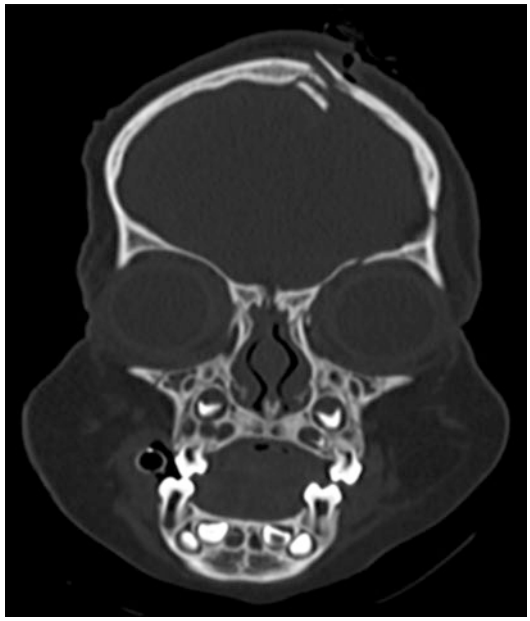


Fig. 12.2 Fracture of the anterior skull and orbital roof in the very young child after fall, coronal view

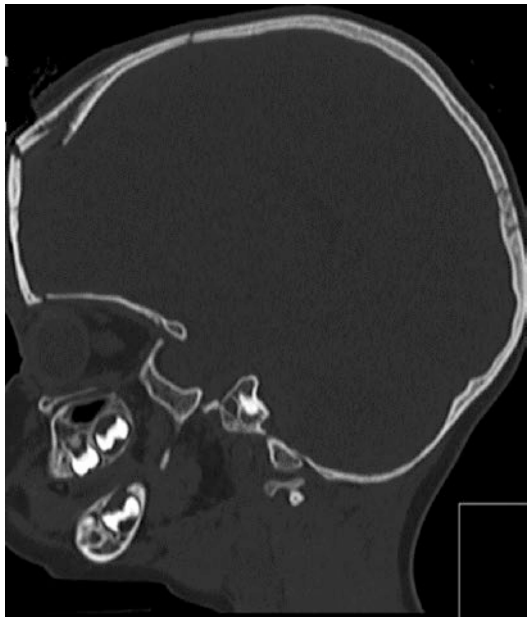


Fig. 12.3 Fracture of the anterior skull and orbital roof in the very young child after fall, sagittal view

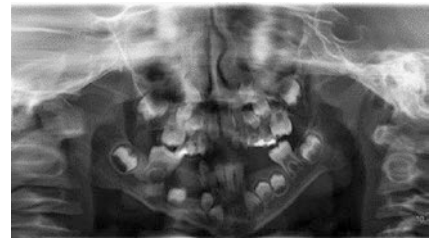


Fig. 12.4 The high maxillary cuspid location during mixed dentition: eye–teeth. Panoramic view

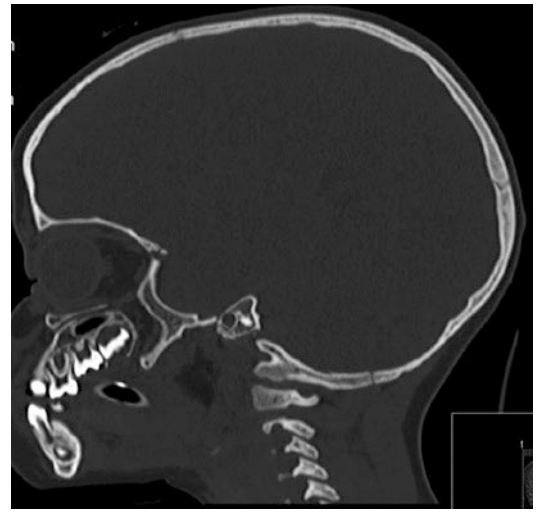


Fig. 12.5 The small maxillary sinus at early age and the high position of the mixed dentition just below the orbital floor, sagittal view

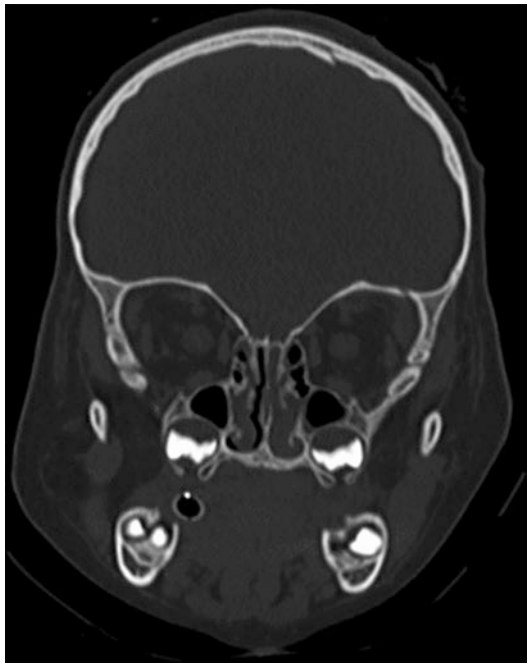


Fig. 12.6 View of early, developing stage ethmoid air cells and maxillary sinus, high position mixed dentition, coronal view

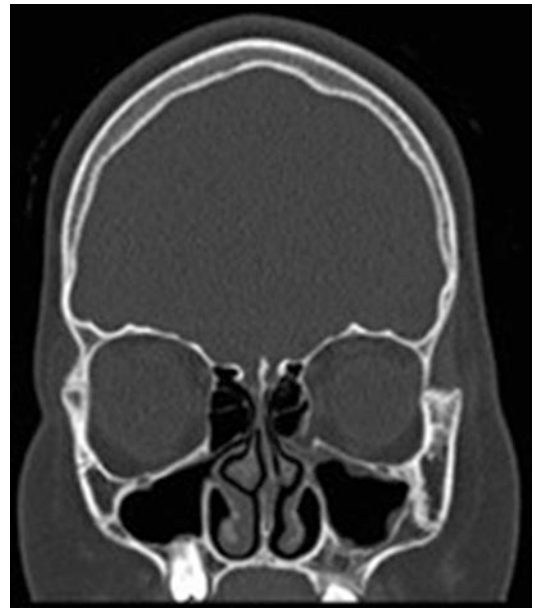


Fig. 12.7 Coronal view trapdoor fracture orbital floor OS; subtle tear-drop sign present; small accompanying low medial wall fracture

The Orbital Floor Fracture in Children: "An Evolving Pattern"

Fracture patterns and susceptibility of orbital fractures change with age. As stated above, this is the combined result of a change in anatomy on the one hand, and a physiological change due to growth and development during maturation on the other. Besides these anatomical changes, physiology during growth will affect the mechanical properties of the craniofacial skeleton. Cancellous immature elastic bone develops into rigid mature bone. Elastic bone will absorb energy differently compared to rigid bone. The elastic, flexible bone that comprise the immature orbital floor is able to deform more than adult compact bone when traumatic force is applied. Because of the flexibility, the orbital floor may bend rather than fracture and if a fracture does occur, the intrinsic elastic property allows for the tendency to recoil. The thick and elastic periosteum may also contribute to the trapdoor mechanism of the orbital wall involved [13]. But before the fracture recoils, soft tissue may herniate and remain entrapped after the hinge-fracture returns to its original position [1, 3, 4, 6]. Mature bone in these cases is much more prone to fracture without subsequent recoil. In the adult case, we are mostly dealing with an open floor fracture with downward displacement of the orbital content, clearly visible on the coronal image of the CT scan. However, in the younger still growing pediatric population, when the child presents with clinical symptoms of an orbital floor fracture, often hardly any findings of displacement of the orbital floor are diagnosed on the CT scan (Fig. 12.7). The thin not fully mineralized bone may be hard to recognize on the scan images or only a tear-drop sign may be present, suggesting orbital soft tissue to prolapse (Figs.12.7 and 12.8). This *trapdoor phenomenon* causes an acute mechanical failure in vertical gaze. Apart from periosteal lining and orbital fat, the inferior rectus- and inferior oblique muscle may become entrapped, the muscle component causing more pronounced inability of vertical globe motility [1–4, 6, 7, 13–19].

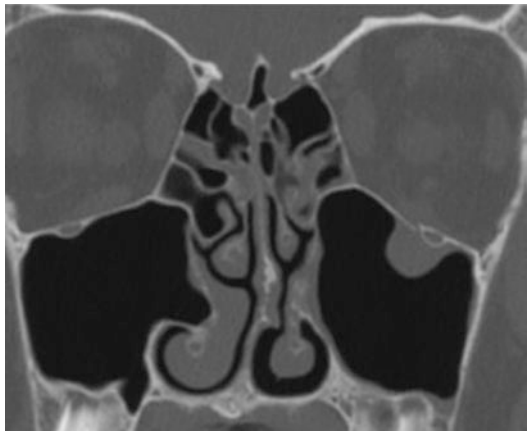


Fig. 12.8 Teardrop sign as a result of an orbital floor trapdoor fracture OS, coronal view

Trapdoor Fracture of the Orbital Floor: Findings

Early Findings

Clinical symptoms:

- Few/absence of peri-orbital signs of facial trauma (ecchymosis), subconjunctival hemorrhage (white eyed orbital fracture)
- Pain/decreased sensation infraorbital nerve supply region
- Lack of enophthalmos
- Marked impaired ocular mobility: limited vertical gaze (Fig. 12.9)
- Head posture (torticollis) to counteract double vision (Fig. 12.10)
- More rarely: oculo-cardiac/oculo-vagal reflex as a result stimulation of the ophthalmic division NV—afferent reticular formation—visceral motor nuclei N Vagus: efferent limb to cardiac system [7, 16–19]. There also may be traction on baroreceptors potentially present in the orbital soft tissue
- Nausea-vomiting-vertigo/bradycardia-hypotension/syncope (watch for potential arrhythmias)

As explained above, due to the elasticity of the bone in children and the ability to recoil, impact to the orbit results in a pure and linear fracture of the orbital floor and peri-orbital content may become entrapped resulting in an acute restriction of eyeball elevation: the patient experiences double vision.

Once the peri-orbital lining is disrupted, extraconal fat and the highly organized connective tissue septa, an accessory locomotor system can herniate in pathologic circumstances like blow-out fractures and can account for the motility disturbances in these cases [20]. As a result, upward gaze is severely restricted and can luxate the oculocardiac reflex. Posturing of the head will reduce the diplopia. Traction on the orbital soft tissue, the extra ocular muscles (EOM) or peri-orbital fat lining stimulates the afferent ophthalmic division of NV resulting in nausea, vomiting and a vaso-vagal (including bradycardia-syncope) as a response [3, 6, 16–19]. *The nausea may be confused with concurrent head injury* i.e*., concussion*. Spontaneous resolution is highly unlikely, surgical intervention should be employed preferably within 12–24 h [1, 3, 19, 21–24]. As stated above, a typical "finding" is the just subtle or absence of skeletal radiological CT findings which may lead to misdiagnosis (Fig. 12.7). Despite multislice (1.0 mm thickness, 1.0 mm increment) computed tomography, CT images are restricted in revealing orbital soft tissue entrapment [18, 24]. In the examination of the patient, the clinical presentation often with marked limitation of globe elevation should outweigh the radiographical (non)findings (Figs. 12.7 and 12.9a, b). When in doubt, an additional MRI can depict more precisely the extent and differentiation of the injured orbital soft tissue.

Late Findings

When no proper surgical intervention is carried out within time, as a result of persisting ischemia, necrosis of herniated, incarcerated orbital soft tissue may develop [4, 18, 21, 23–25]. This is especially true for connective tissue septae, orbital fat and fascial muscle sheet. Fibrosis and finally scarring result in persisting or potentially permanent vertical motility restriction of the globe [21, 23, 24, 26, 27]. Ischemia of developing orbital

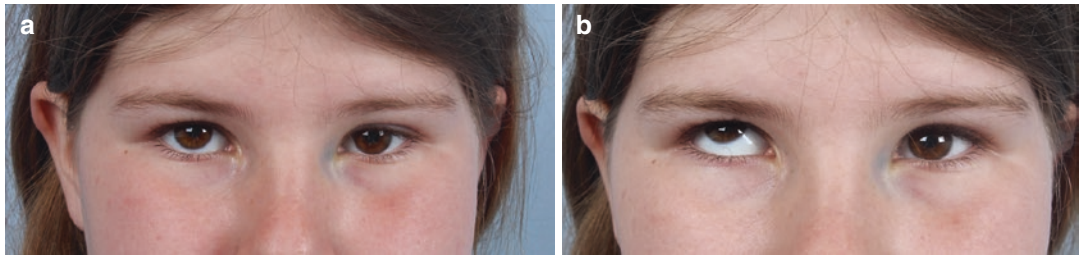


Fig. 12.9 (**a**) Preoperatively clinical view: primary gaze. (**b**) Preoperatively clinical view: limited upward gaze OS

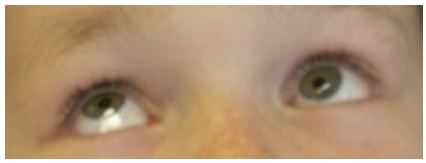


Fig. 12.10 Young boy with torticollis, compensatory head tilt to limit double vision

soft tissue and EOM tissue is more likely to result in incapacitating ocular motility: one can assume that the growing potential to an adult structure is irreversibly interrupted by the longer standing incarceration or strangulation of especially inferior oblique-inferior rectus muscle resulting in a complication very difficult to correct. It is reported that, in the younger patient group, diplopia takes more time to resolve and that they have more persistent problems [6, 28]. So, if left untreated, permanent restriction in ocular motility may result.

In some cases, patients are referred after several weeks; meanwhile, the patient may develop a torticollis to compensate for the double vision (Fig.12.10).

Variations in Orbital Wall Fractures

Blow-Up Fracture, Blow-In Fracture

A blow-up fracture involves the superior displacement of the orbital roof into the anterior cranial fossa [4]. A blow-in fracture describes an inferior displacement of the orbital roof [4]. In case of an orbital roof fracture, watch for displacement and possible accompanying dural tearing resulting in a possible leptomeningeal cyst (encephalocele) or (progressive) pulsatile exophthalmos; proptosis, vertical dystopia may develop. Rarely, a progressive, growing orbital roof skull fracture is seen, which may still develop months after head injury has occurred [2, 8, 9, 29, 30].

Pure Versus Impure Orbital Floor Fracture

A distinction can be made between a pure or indirect (solely orbital floor fracture) and impure or direct (orbital floor in conjunction with other fractures) orbital floor fracture [2].

Open Door Fracture Versus Trapdoor

Opposite of the trapdoor fracture is the "open door" fracture, a floor fracture without entrapment which is more common in de adult population [2]. When the children grow older, chances of an open door, blowout fracture increase and enophthalmos may result. The term "blow-out" fracture had already been introduced by Smith and Regan in 1951 [14].

Medial Wall Fracture

The medial wall fracture in older children is similar to adults and is described in Chap. 10.

Complex (Multi) Fracture

Orbital fractures can of course also be part of a complex Naso-Orbito-Ethmoid (NOE) fracture, a midface Lefort fracture (<5%) or a relatively simple zygomatic complex fracture (16%), fortunately rare in younger children [2, 7].

Tests and Treatment Principles

The management of growing individuals who present with an orbital fracture requires a customized approach, adjusted according to the growing individual. In the evaluation of the patient, apart from a change in vision, critical aspects consist of globe motility disturbances and enophthalmos or hypoglobus.

Thorough examination by the OMF surgeon, the ophthalmologist and orthoptist should be carried out (Fig. 12.11a). However, a complete examination can be difficult to obtain in the young, sometimes obstinate uncooperative patient. A "white-eye" orbital fracture may even lead to denial diagnosis and doctor's delay in adequate treatment [25, 26]. A CT scan should be obtained in case of suspicion of an orbital wall fracture. Displacement of bone structures provides a simple diagnosis but often the findings in children are limited to a tear-drop sign (Fig. 12.8). Moreover, CT images may also incorrectly deny the existence of a fracture (Fig. 12.7). A 3D reconstruction can support a more accurate diagnosis. Nonetheless, the clinical findings are in the lead when it comes to a treatment plan.

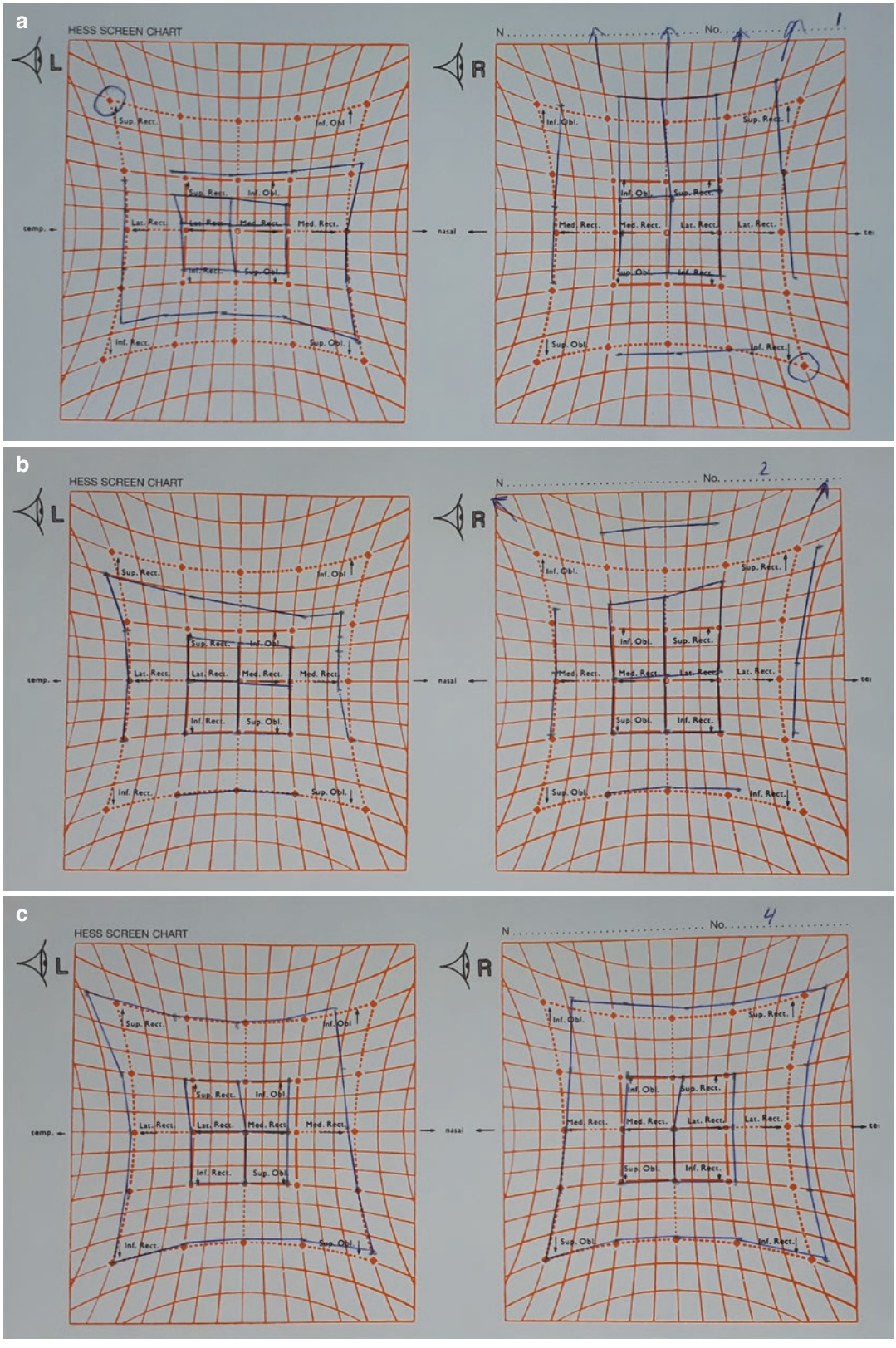
When no acute enophthalmos, hypoglobus, diplopia or entrapment is present, these fractures can be treated conservatively, closely monitored during surveillance. When diplopia is present, orthoptic evaluation is mandatory prior to surgery to classify the extent of the motility disorder and compare these initial findings with future recovery development (Fig.12.11a–c). When on initial presentation, there is double vision in *many or all* directions, this is most likely to be caused by swelling i.e., oedema instead of entrapment of orbital soft tissue. Allow some time to recover and follow closely. When double vision is seen in just a *few or one* direction, entrapment is the most obvious diagnosis which warrants immediate intervention [24, 29]. Once entrapment is diagnosed, surgical intervention of especially the incarcerated tissue should be done within 12–24 h [1–4, 7, 14, 15, 19, 21, 24, 25, 27]. The primary goal is to release the entrapped orbital soft tissue (Figs. 12.12 and 12.13). Because of the recoil of the linear trapdoor fracture, there is hardly any need to restore the orbital floor in such cases (Fig. 12.13). If intervention is carried out instantly, a complete recovery within days is very likely (Figs. 12.11c and 12.14a, b). Another indication for immediate or early intervention is the presence of acute enophthalmos >2 mm and hypoglobus. The Hertel exophthalmometer is used to measure the extent of the enophthalmos.

When during follow-up the initial (peri)orbital swelling has subsided and clinical signs and symptoms of limited recovery of ocular motility or enophthalmos >2 mm and hypoglobus become apparent, *early intervention* (2–14 days) may be indicated. Again, the definition, measurement and reduction of double vision i.e., diplopia should always be objectivated by careful orthoptic evaluation [31].

Delayed intervention (2–3 weeks) may be performed when there is insufficient recovery of double vision. However, a delayed or late intervention will generally result in a poorer outcome, especially in the younger generation. Moreover, the high bone and soft tissue turnover in the younger patient group challenges the intervention at a later stage.

Late enophthalmos can be an indication for *late intervention* (>3 weeks).

Fig. 12.11 (**a**) Orthoptic evaluation: Hess preoperatively: severe limitation of upward gaze OS, confined limitation of depression OS, near (30 cm distance) little exophoria, small right—hyperphoria (no double vision), far (5 m distance) little right—hypertropia (double vision), overshoot upward gaze OD. (**b**) Orthoptic evaluation: Hess 2 weeks postoperatively, restore eye motility, however not yet complete recovery upward gaze OS, recovery limitation of depression OS, still some overshoot OD. (**c**) Orthoptic evaluation: Hess 3 months postoperatively, full recovery of limitation of elevation OS


a
HESS SCREEN CHART
L
R
N
No. 1
Sup. Rect.
Inf. Obl.
Lat. Rect.
Med. Rect.
Inf. Rect.
Sup. Obl.
temp.
nasal
b
HESS SCREEN CHART
No. 2
c
HESS SCREEN CHART
No. 4

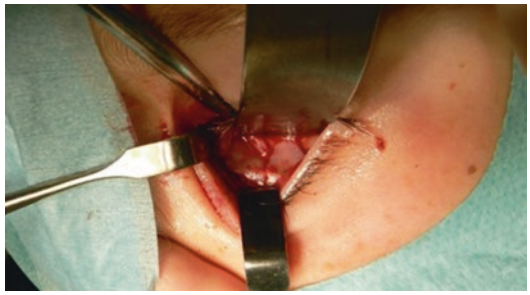


Fig. 12.12 Intraoperative view orbital floor OS: herniated, incarcerated orbital soft tissue in linear trapdoor orbital floor fracture (patient Fig. 12.9)

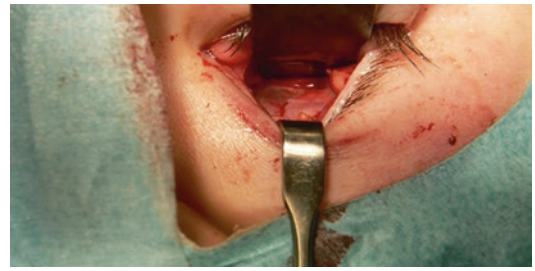


Fig. 12.13 Intraoperative view orbital view OS: retrieved orbital soft tissue from linear trapdoor orbital floor fracture (patient Fig. 12.9)

Fig. 12.14 (**a**) Postoperatively 2 weeks: primary gaze: binocular single vision. (**b**) Postoperatively 2 weeks: recovery limitation elevation OS

When treatment is indicated, even in children, a transconjunctival approach gives ample access to the orbital floor. Nevertheless, when surgically intervening in the growing orbit, we should always keep in mind that the orbital wall morphology is different in children and further development is still to come [1, 32].

Releasing the entrapped soft tissue will often be enough treatment; orbital floor reconstruction is seldom necessary. If a larger floor defect needs coverage to prevent recurrence of herniation, we preferably use an autologous graft. The autologous grafts are instantly available, have ideal mechanical properties, revascularization potential and adaptation to orbital tissue with a minimal immune response. In the growing individual, we are hesitant to use foreign or non-degradable materials. We tend not to use alloplastic materials, there is an increased rate of infection and possible migration of the reconstruction material. Screw fixation of Med Por or titanium plates (mesh) should be avoided in the growing individual [4]. Resorbable materials are more suitable but may cause an inflammatory response during the resorption process which negatively affects the surrounding orbital soft tissue [33]. Intra-operatively, a presurgical and postsurgical forced duction test is conducted (Fig.12.15). Postoperatively, neurologic, ophthalmologic and orthoptic follow-up are necessary. Instructions are given not to blow the nose and patients are cautioned to avoid sneezing: both can cause subcutaneous or intra-orbital emphysema. Generally, if surgical intervention is carried out in time, a good final outcome can be expected (Fig. 12.16a–g).

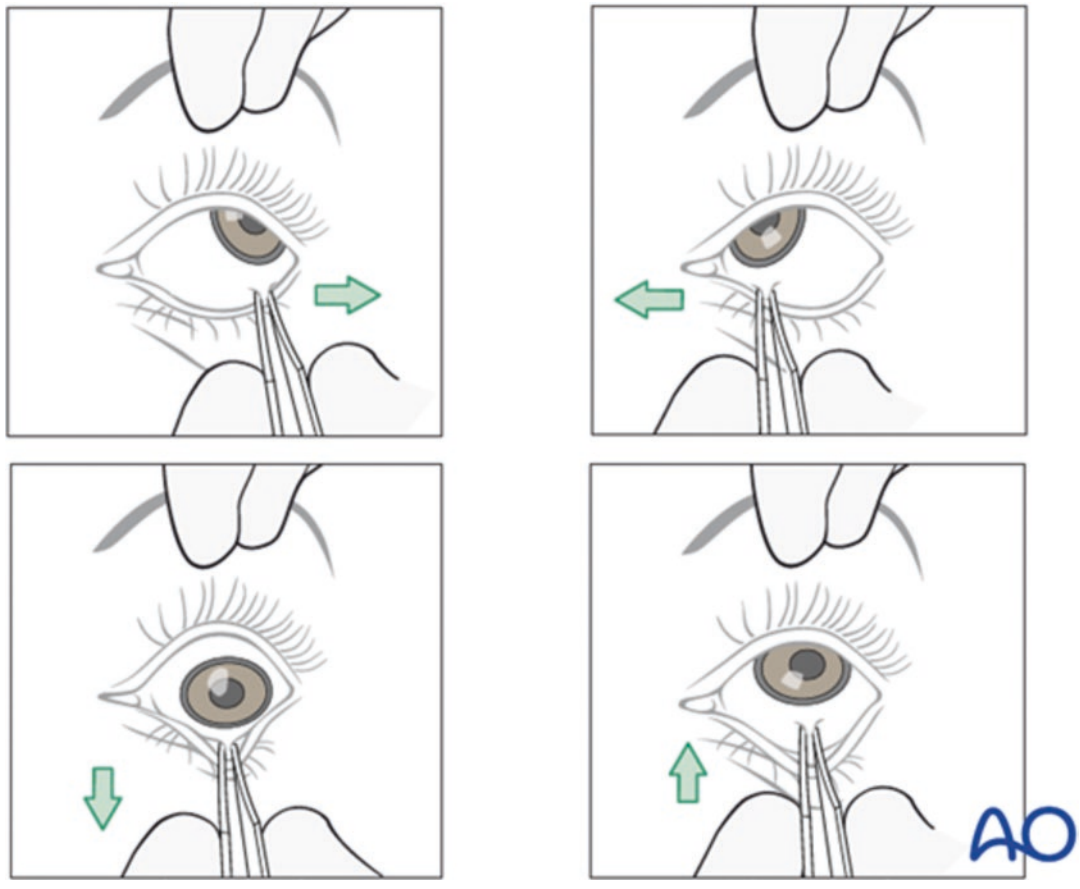


Fig. 12.15 AO illustration of forced duction test (with permission from AO Foundation)

Post-operative Warning Signs

Increasing pain or reduced visual acuity or inadequate pupil reaction require instant re-examination and exploration. A compartment syndrome of the orbital apex or retrobulbar hemorrhage is a serious threat for vision. Orbital roof fractures can be associated with a fracture of the anterior skull base, the dura lining may be torn resulting in CSF leakage. Thus, in the long term, the development of a growing orbital roof fracture is exceptional [29].

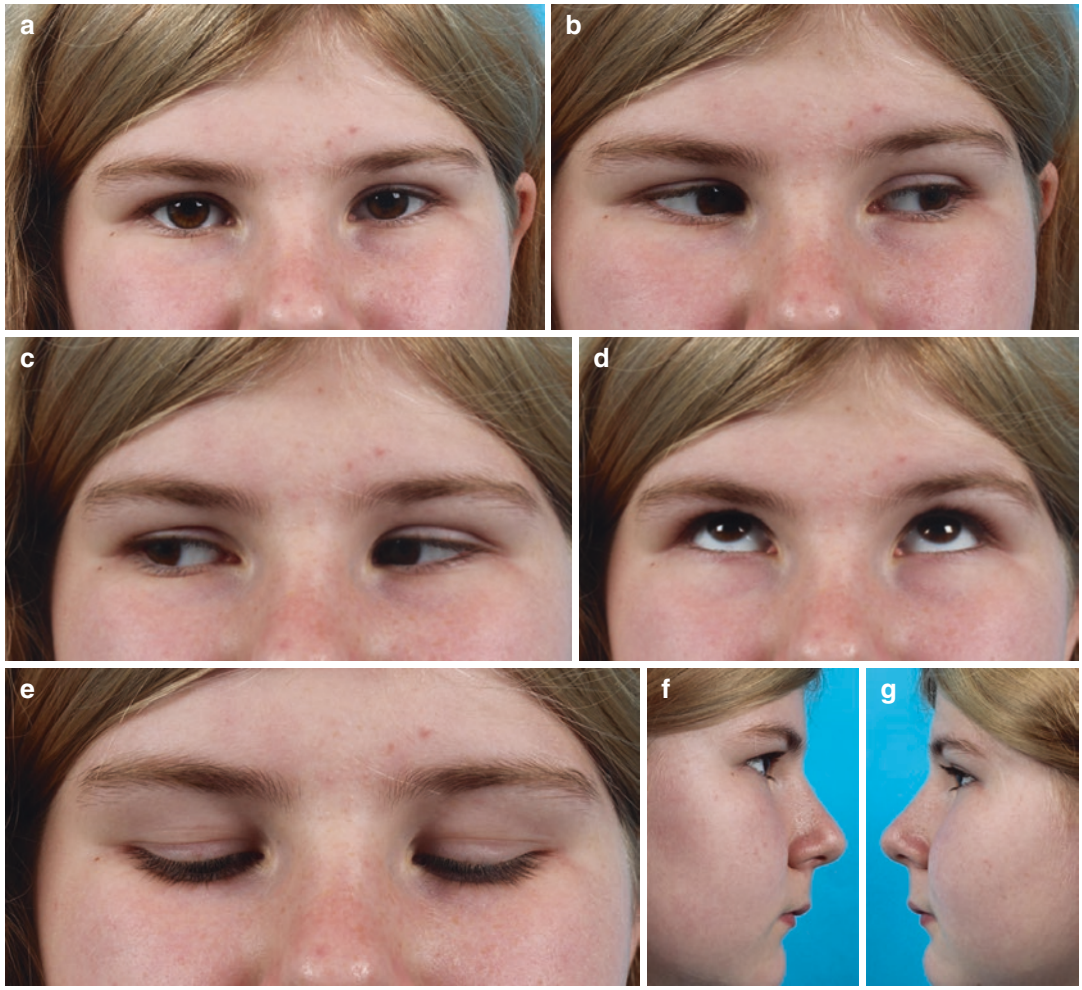


Fig. 12.16 (**a**) Postoperatively 2 years: primary gaze: binocular single vision. (**b**) Postoperatively 2 years: abduction, single vision. (**c**) Postoperatively 2 years: adduction, single vision. (**d**) Postoperatively 2 years: elevation, unlimited. (**e**) Postoperatively 2 years: depression, unlimited. (**f**) Postoperatively 2 years: A-P globe position 17 mm Hertel OD. (**g**) Postoperatively 2 years: A-P globe position 17 mm Hertel OD

References

1. Stotland MA, Do NK. Pediatric orbital fractures. J Craniofac Surg. 2011;22:1230–5.
2. Oppenheimer AJ, Monson LA, Buchman SR. Pediatric orbital fractures. Craniomaxillofac Trauma Reconstr. 2013;6:9–20.
3. Gerber B, Kiwanuka P, Dhariwal D. Orbital fractures in children: a review of outcomes. Br J Oral Maxillofac Surg. 2013;51:789–93.
4. Joshi S, Kassira W, Thaller SR. Overview of pediatric orbital fractures. J Craniofac Surg. 2011;22:1330–2.
5. Sirichai P, Anderson PJ. Orbital fractures in children: 10 years experience from a tertiary centre. Br Oral Maxillofac Surg. 2015;53(10):938–42.
6. Heggie AA. Isolated orbital floor fractures in the paediatric patient: case series and review of management. Int J Oral Maxillofac Surg. 2015;44:1250–4.
7. Cobb ARM, Jeelani NO, Ayliffe PR. Orbital fractures in children. Br Oral Maxillofac Surg. 2013;51:41–6.
8. Greenwald MJ, Boston D, Pensler JM, Radkowski MA. Orbital roof fractures in childhood. Ophthalmology. 1989;96(4):491–6.
9. Messinger A, Radkowski MA, Greenwald MJ, Pensler JM. Orbital roof fractures in the pediatric population. Plast Reconstr Surg. 1989;84:213–6.
10. Koltai PJ, Amjad I, Meyer D, Feustel PJ. Orbital fractures in children. Arch Otolaryngol Head Neck Surg. 1995;121:1375–9.
11. Chang JT, Morrison CS, Styczynski R, Mehan W, Sullivan SR, Taylor HO. Pediatric orbital depth and

growth: a radiographic analysis. J Craniofac Surg. 2015;26(6):1988–91.
12. Escaravage GK, Dutton J. Age-related changes in the pediatric human orbit on CT. Ophthalmic Plast Reconstr Surg. 2013;29:150–6.
13. Guyot L, Lari N, Benso-Layoun C, Denis D, Chossegros C, Thiery G. Orbital fractures in children. J Fr Ophthalmol. 2011;34(4):265–74.
14. Smith B, Regan WF Jr. Blow-out fracture of the orbit; mechanism and correction of internal orbital fracture. Am Ophthalmol. 1957;44:733–9.
15. Losee JE, Afifi A, Jiang S, Smith D, Chao MT, Vecchione L, Hertle R, Davis J, Naran S, Hughes J, Paviglianiti J, Deleyiannis FW-B. Pediatric orbital fractures: classification, management, and early follow up. Plast Reconstr Surg. 2008;122:886–97.
16. Sires BS, Stanley RB, Levine LM. Oculocardiac reflex caused by orbital floor trapdoor fractures: an indication for urgent repair. Arch Ophthalmol. 1998;116:955–6.
17. Cohen S, Garret C. Paediatric orbital floor fractures: nausea/vomiting as signs of entrapment. Otolaryngol Head Neck Surg. 2003;129:43–7.
18. Bansagi Z, Meyer D. Internal orbital fractures in the paediatric age group: characterization and management. Ophthalmology. 2000;107:829–36.
19. Egbert JE, May K, Kersten RC, Kulwin DW. Paediatric orbital floor fracture. Ophthalmology. 2000;107:1875–9.
20. Koornneef L. Orbital septae: anatomy and function. Ophthalmology. 1979;86(5):876–80.
21. Smith B, Lisman R, Simonton J. Volkmann's contracture of the extraocular muscles following blow out fractures. Plast Reconstr Surg. 1984;74:200–5.
22. de Man K, Wijngaarde R, Hes J, de Jong PT. Influence of age on the management of blow-out fractures. Int Oral Maxillofac Surg. 1991;20:330–6.
23. Wei LA, Durairaj VD. Pediatric orbital floor fractures. J AAPOS. 2011;15:173–80.
24. Parbhu KC, Galler KE, Li C, Mawn LA. Underestimation of soft tissue entrapment by computed tomography in orbital floor fractures in the pediatric population. Ophthalmology. 2008;115:1620–5.
25. Jordan DR, Allen LH, White J, Harvey J, Pashby R, Esmaeli B. Intervention within days for some orbital floor fractures: the white-eyed blowout. Ophthalmic Plast Reconstr Surg. 1998;14:379–90.
26. Lane K, Penn RB, Bilyk JR. Evaluation and management of pediatric orbital fractures in a primary care setting. Orbit. 2009;26(3):183–91.
27. Karthik R, Cynthia S, Vivek N, Saravanan C, Prashanthi G. Intraoperative findings of extraocular muscle necrosis in linear orbital trapdoor fractures. J Oral Maxillofac Surg. 2019;77(6):1229.e1–8. https://doi.org/10.1016/j.joms.2019.02.033.
28. Cope MR, Moos KF, Speculand B. Does diplopia persist after blow out fractures of the orbital floor in children. Br J Oral Maxillofac Surg. 1999;37:46–51.
29. Suri A, Mahapatra AK. Growing fractures of the orbital roof. A report of two cases and a review. Pediatr Neurosurg. 2002;36(2):96–100.
30. Coon D, Yan N, Ones D, Howell LK, Grant MP, Redett RJ. Defining pediatric orbital roof fractures: patterns, sequelae, and indications for operation. Plast Reconstr Surg. 2014;134(3):442e–8e.
31. Jansen J, Dubois L, Maal T, Mourits MP, Ellema HM, Neomagus P, Lange de J, Hartman LJC, Gooris P, Becking AG. A nonsurgical approach with repeated orthoptic evaluation is justified for most blow-out fractures. J Craniomaxillofac Surg. 2020;48:560–8.
32. Nagasao T, Hikosaka M, Morotomi T, Nagasao M, Ogawa K, Nakajima T. Analysis of the orbital floor morphology. J Craniomaxillofac Surg. 2007;35:112–9.
33. Bergsma JE. Poly(L-lactide) implants in repair of the orbital floor. A 5 years animal study. Cells Mater. 1994;4:31–6.