Pediatric Considerations in Craniofacial Trauma

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KEYWORDS

• Pediatric craniofacial trauma • Pediatric facial fractures • Pediatric normal skull base
• Pediatric craniofacial development • Pediatric facial trauma • Toppled furniture
• All-terrain vehicle pediatric injuries • Impalement injuries

KEY POINTS

- Mechanism of injury and growth and development of the pediatric face play a role in the type and pattern of injury in pediatric craniofacial trauma.
- Normal variant lucencies in the pediatric skull base are important to recognize, so as not to misdiagnose fractures.
- Lack of complete ossification of the anterior skull base, before the age of 4 years, should not be mistaken as a posttraumatic or congenital anomaly.
- Trapdoor orbital floor fractures are more common in children than adults, and can result in entrapment of orbital soft tissues, without significant displacement of fracture fragments.
- Beware of toppled furniture, especially the television, as a cause of significant craniofacial and skull base trauma in children.
- Most pediatric craniofacial impalement injuries are treated conservatively. However, imaging is very helpful to define the extent of injury and assess for retained foreign bodies.

INTRODUCTION

Craniofacial trauma in children is in many respects very similar to that in adults. The patterns of fractures and associated injuries in older children and adolescents are frequently identical to those found in adults. However, the patterns of facial injury in younger children differ from those in adults, primarily reflecting changes in anatomy and physiology of the developing face, extent of paranasal sinus pneumatization, and phase of dentition. The frequency of different types of fractures is, therefore, also variable depending on the age of the child. In addition to understanding how normal growth and development of the pediatric skull base and craniofacial structures affect the patterns of injury in children, it is important for the imager to recognize multiple normal variant lucencies in the pediatric skull base that may mimic fractures. Furthermore, a few types of injury deserve special attention in children, including injuries related to toppled furniture, nonaccidental trauma, all-terrain vehicle (ATV) accidents, and impalement injuries.

NORMAL GROWTH AND DEVELOPMENT

Growth and development play a role in the types of craniofacial fractures that occur at differing ages. Because many of the structures are still in the process of growing and maturing, and dentition may be incomplete, pediatric maxillofacial injuries carry with them the risk of altering the function and ultimate growth of the affected structures. Therefore, timely diagnosis and prompt management are important to prevent disturbances in future growth that may affect function, dental occlusion, and cosmetic appearance. By the end of the first year of life, the mandibular halves are fused at the sypmphysis. The condyle contributes to the

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vertical growth of the mandible. Most growth of the zygoma and maxilla is complete by 7 years, most orbital growth is completed by 5 to 7 years of age, but cranial vault and craniofacial structures typically do not achieve growth maturity until 14 to 16 years of age.[1] The bones of the craniofacial skeleton grow and develop by remodeling and displacement throughout young life. Remodeling occurs secondary to local factors that result in change in size and shape of each component, and displacement occurs secondary to bones moving apart at joints, sutures, and articular surfaces. The cranium and orbits grow in response to the growth of the brain and globes early during the first year of life and growth of the zygoma and maxilla is initially slower than the cranio-orbital region. Therefore, the cranio-orbital complex is larger than the maxilla-mandibular complex in infancy. Over time, the young child's craniofacial development is altered by central nervous system, optic pathway, and speech/swallowing development and use and development of muscles of facial expression and mastication, paranasal sinus pneumatization, and normal phases of dentition. Deciduous teeth begin to erupt at approximately 6 months of age, mixed dentition is noted at about 6 years of age, and adult dentition is reached by 12 or 13 years of age.

Features unique to the young pediatric face that affect outcome of injury

Cranio-orbital complex is larger than the maxilla-mandibular complex in infancy

Incomplete development of the paranasal sinuses: increases stability and decreases incidence of midface fractures

Incomplete dentition: increases stability and decreases incidence of mandible fractures, rare in infants

NORMAL VARIANT LUCENCIES IN THE SKULL BASE

The postnatal development of the anterior and central skull base is complex, and beyond the scope of this article. The central skull base (chondrocranium) is composed of at least 25 separate ossification centers in the embryo that ultimately contribute to the mature sphenoid and occipital bones.[2] Throughout childhood, there are many normal skull base sutures, fissures, synchondroses, vascular channels, and clefts that can routinely be identified on head and neck computed tomography (CT) imaging in children. Knowledge of the normal developmental anatomy of the skull base is important to prevent misinterpretation of these findings as fractures, osseous lesions, and cephaloceles.

A large number of normal lucencies are identified in the central skull base, including but not limited to the spheno-occipital synchondrosis, olivary eminence, craniopharyngeal canal, canalis basilaris medianus, median raphe of the basiocciput, and coronal clefts of the basiocciput. In addition, there are normal variant lucencies in the occiput that should not be confused with fractures. These include remnants of the anterior intraoccipital synchondrosis, and posterior lucencies related to variant fusion of Kerckring ossicle.

At birth, there are multiple separate ossification centers that ultimately form the mature sphenoid bone, all of which are initially separated from the adjacent centers by a nonossified synchondrosis. The most commonly visualized synchondrosis related to the sphenoid bone on postnatal CT is the spheno-occipital synchondrosis. Most skull base growth occurs at the spheno-occipital synchondrosis, which separates the postsphenoid ossification center from the basiocciput and remains patent until teenage years (**Fig. 1**). During closure, small ossified bodies may be identifiable within the spheno-occipital synchondrosis (**Fig. 2**). After closure is complete, there are frequently small divots, clefts, or fissures on one or both sides of the spheno-occipital synchondrosis.

In infants, the sphenoid body frequently contains two visible midline foramina, an anterior triangular-shaped lucency and a round posterior foramen. The anterior cartilage-containing structure is called the olivary eminence (**Fig. 3**) and is not identifiable in most older children, but may be visible as a sclerotic remnant in 11.2% of children older than 9 months of age.[2] The round posterior foramen, the craniopharyngeal canal, is a tubular lucency extending from the floor of the sella turcica to the roof of the nasopharynx (**Fig. 4**). The craniopharyngeal canal is visible on CT in 8.5% of children, and as a partial canal or sclerotic remnant in 20% of children. Rarely, this canal is pathologically widened secondary to the presence of cephaloceles that frequently contain ectopic adenohypophysis (**Fig. 5**).[3]

Most normal-variant lucencies in the occipital bone involve the basiocciput or the region of the Kerckring ossicle. Occasionally, midline lucency in the basiocciput, called the canalis basilaris medianus, is identified posterior to the spheno-occipital synchondrosis (**Fig. 6**). This structure

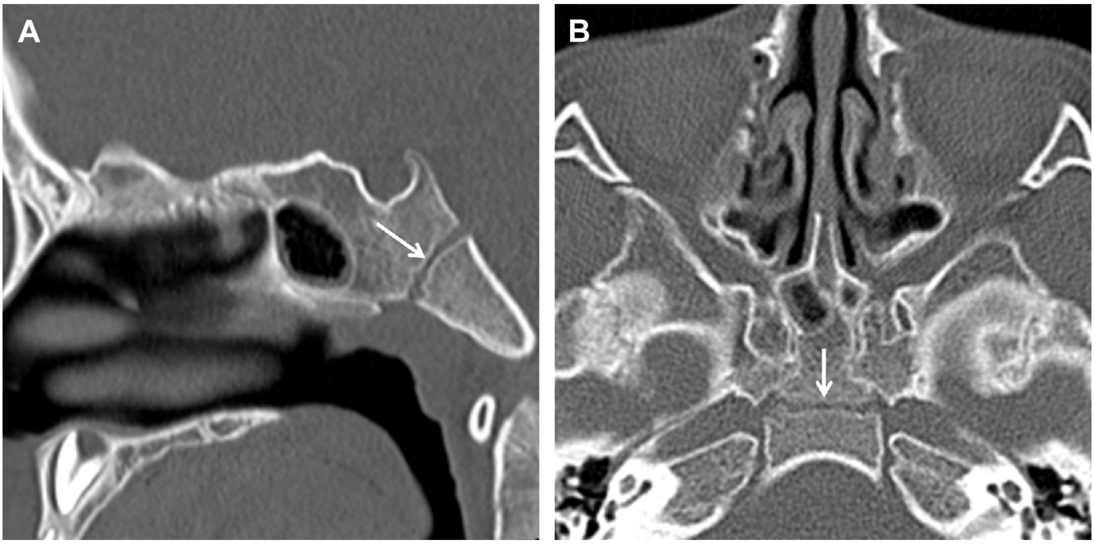

Fig. 1. Spheno-occiptal synchondrosis. (*A*) Sagittal reformatted CT image in a 4 year old demonstrates a patent spheno-occipital synchondrosis (*arrow*). (*B*) Axial bone window CT image in the same child shows the horizontal lucency between the postsphenoid and the basiocciput (*arrow*).

may be variable in shape, and complete or incomplete.[4,5] The canalis basilaris medianus is thought to represent a remnant of the cephalic end of the notochordal canal, most frequently is an incidental finding, but is rarely associated with nasopharyngeal cysts (**Fig. 7**).[5,6] The anterior intraoccipital synchondrosis has a variable appearance over time, and during fusion may progress from a somewhat cross-shaped appearance to a small well-corticated round lucency (**Fig. 8**). Coronal clefts involving the basiocciput may also occur. Finally, lucencies related to variant fusion of Kerckring ossicle include unfused and partially fused Kerckring ossicles (**Fig. 9**), both of which, if not recognized as normal variants, may be misinterpreted as fracture. When fracture is suspected on axial imaging, three-dimensional reconstructions in these children are frequently very helpful to better define the lucencies as normal variants related to Kerckring

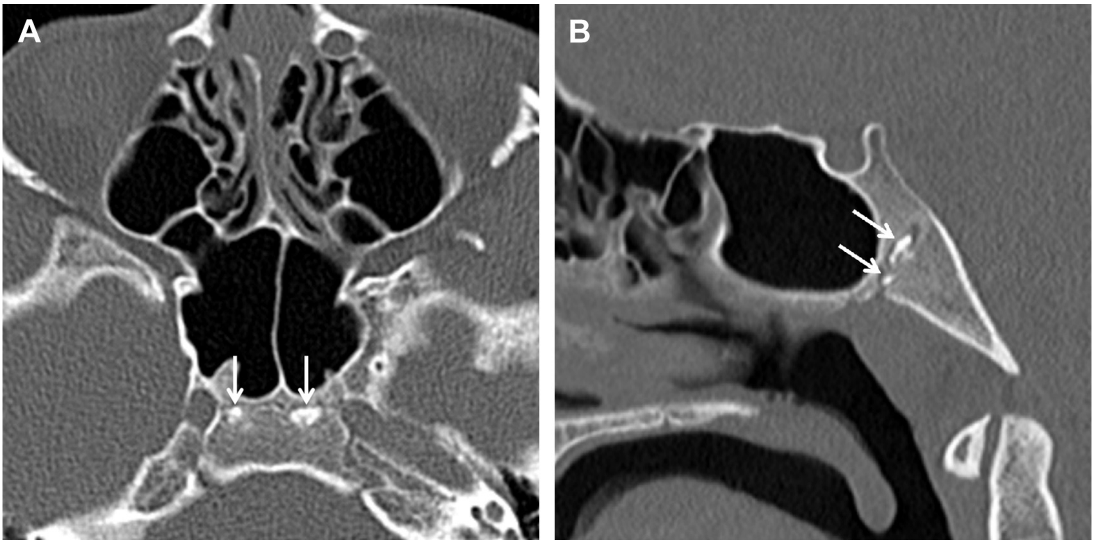

Fig. 2. Remnants of the spheno-occipital synchondrosis. (*A*) Axial and (*B*) sagittal bone window images in a 13 year old show normal variant small ossified bodies (*arrows*) within the closing spheno-occipital synchondrosis.

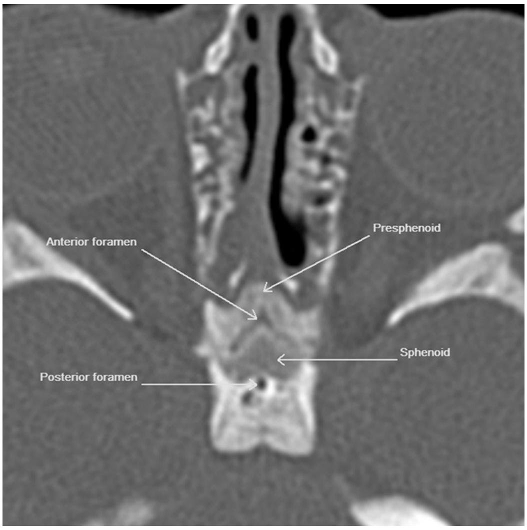

Fig. 3. Olivary eminence. Axial CT image in a 1 day old shows the typical triangular-shaped anterior foramen, called the olivary eminence, located posterior to the presphenoid and anterior to the paired main sphenoid ossification centers. This is only identifiable in infants, but may be present as a sclerotic remnant in children older than 9 months. Also easily identifiable is the posterior foramen, called the craniopharyngeal canal.

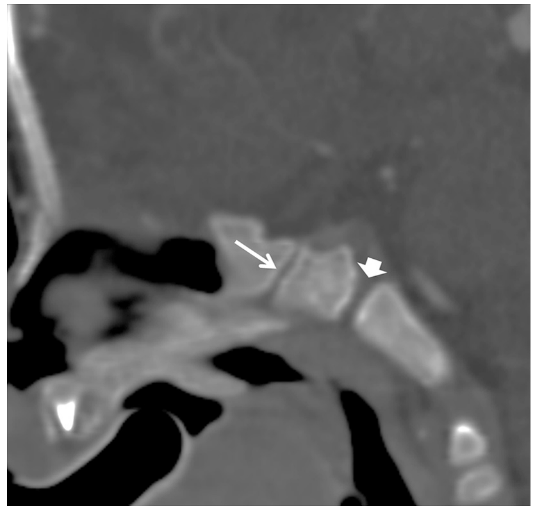

Fig. 4. Craniopharyngeal canal. Sagittal reformatted CT image in a 4-month-old child shows the normal craniopharyngeal canal (*arrow*), extending from the floor of the sella turcica to the roof of the nasopharynx, anterior to the patent spheno-occipital synchondrosis (*arrowhead*).

ossicle rather than fracture lines. Three-dimensional reconstructions are also helpful in proving that lucencies related to intrasutural bones, when they occur anywhere in the skull, are not fractures.

Most common normal variant sutures and lucencies in the skull base

Spheno-occipital synchondrosis: may see remnant clefts, fissures, or small ossified bodies

Olivary eminence: only identifiable in infants

Craniopharyngeal canal: floor of sella to roof of nasopharynx, completely fuses in most children, rarely contains cephalocele

Canalis basilaris medianus: usually incidental finding, rarely associated with nasopharyngeal cysts

Median raphe of the basiocciput

Coronal clefts of the basiocciput

Anterior intraocciptial synchondrosis: changes shape over time from somewhat cross-shaped to well-corticated round lucency

Unfused or partially fused Kerckring ossicle: may be confused with fracture

NORMAL ANTERIOR SKULL BASE OSSIFICATION

Imagers must also recognize several additional potential pitfalls related to the complex ossification pattern of the anterior skull base in order not to

Fig. 5. Craniopharyngeal canal cephalocele containing adenohypopyhsis. Sagittal T1-weighted MR image in a 10-day-old boy demonstrates a wide, primarily cerebrospinal fluid–containing cephalocele (*arrows*), extending through the floor of the sella, into the posterior nasopharynx. Notice posterior pituitary bright spot along the dorsal aspect of the cephalocele and patent spheno-occipital synchondrosis.

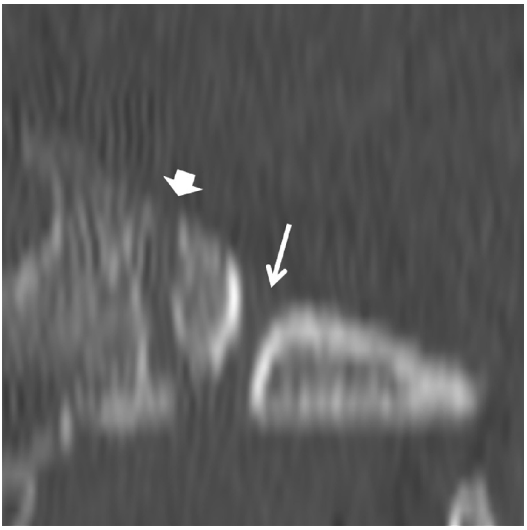

Fig. 6. Canalis basilaris medianus. Sagittal reformatted images from a temporal bone CT in a 4 year old show the patent canalis basilaris medianus (*arrow*), posterior to the patent spheno-occipital synchondrosis (*arrowhead*).

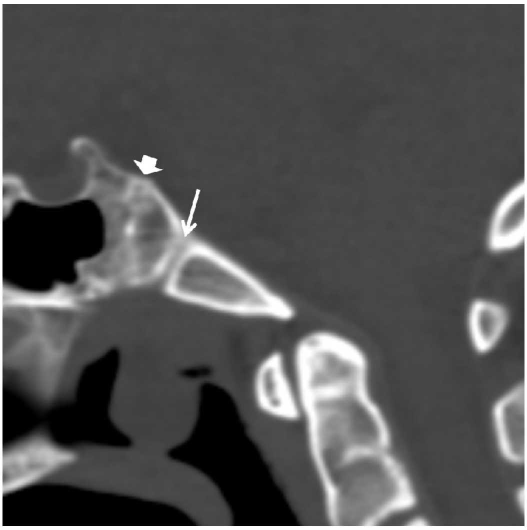

Fig. 7. Canalis basilaris medianus associated with nasopharyngeal cyst. Sagittal reformatted CT in the same child as Fig. 6, at 13 years of age, shows interval fusion of the spheno-occipital synchondrosis (*arrowhead*) and narrowing but persistent patency of the canalis basilaris medianus (*arrow*). Notice also the now visible nasopharyngeal mass just beneath the canalis basilaris medianus.

mistake such items as incomplete or multiple ossification centers as a defect from trauma, or as a cephalocele. Anterior skull base ossification occurs in a fairly predictable fashion, but with varying rates in young children. Most of the skull base at birth is composed of cartilage (Fig. 10). During the first few months of life, there is progressive ossification of the cribriform plate, roof of the nasal cavities, and crista calli. Ossification of the cribriform plate begins near the region where the superior and middle turbinates attach and extends medially to reach the crista galli by about 2 months of age. Ossification extends from the cribriform

Fig. 8. Anterior intraoccipital synchondrosis. (*A*) Axial CT image in a 2 year old demonstrates somewhat cross-shaped lucencies, which represent the incompletely fused intraoccipital synchondroses (*arrows*). (*B*) Axial CT image at 7 years of age in a different child demonstrates small, foramen-like remnants of the fused intraoccipital synchondrosis (*arrows*).

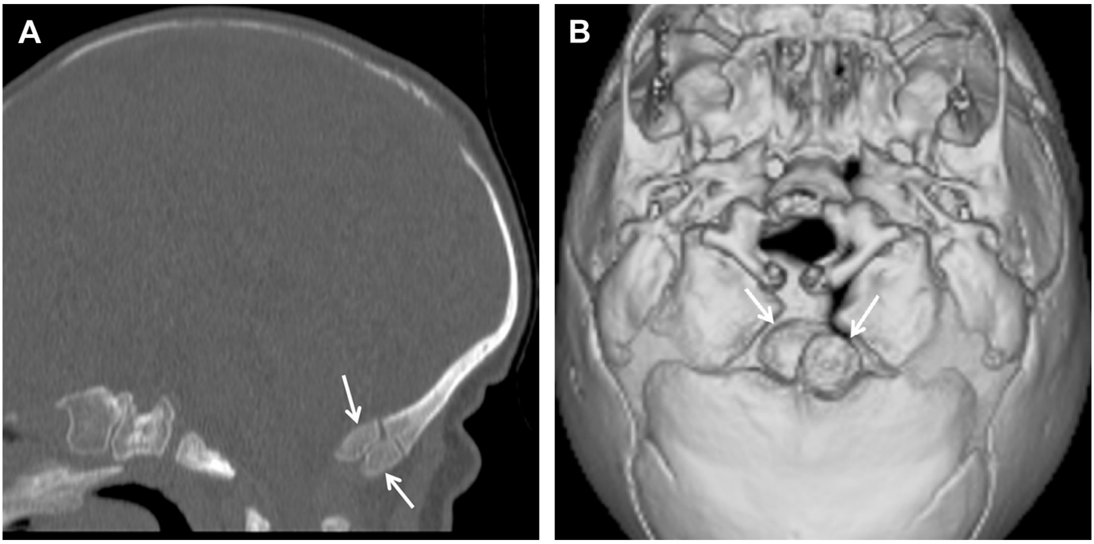

Fig. 9. Kerckring ossicle variants. (*A*) Sagittal CT in a newborn demonstrates unfused Kerckring ossicles (*arrows*). (*B*) Three-dimensional reformatted CT image in the same child demonstrates unfused duplicated Kerckring ossicles (*arrows*) at the posterior aspect of the foramen magnum.

plate, and proceeds posteriorly more quickly than anteriorly, therefore a nonossified gap is frequently present anterior to the crista galli in very young children. Only 4% of children in a study by Hughes and colleagues[7] had complete ossification of the anterior skull base by 2 years of age, whereas all patients had a fully ossified anterior skull base by the age of 3 years, 10 months (**Fig. 11**). After 4 years of age, the only unossified normal structure that remains in the midline anterior cranial fossa is the foramen cecum, just anterior to the crista galli (**Fig. 12**), which may transmit a small vein.

Anterior skull base ossification

Majority is unossified at birth

4% of children have completely ossified anterior skull base by 2 years of age

All children fully ossified anterior skull base by 4 years of age

After 4 years, only unossified portion of anterior skull basea is foramen cecum

PARANASAL SINUS DEVELOPMENT

Knowledge of normal paranasal sinus development is helpful to understand the impact and outcome of craniofacial injuries in children. For example, concern for frontal sinus fracture and its associated complications is not an issue in children who have not yet developed aeration of the frontal air cells. In addition, lack of sinus pneumatization is thought to provide increased stability and resultant decreased incidence of midface fractures in younger children. Paranasal sinus development follows a fairly predictable pattern; however, the ultimate degree of pneumatization of each sinus is variable between individuals. The maxillary sinus is formed, but rudimentary at birth. Lateral extension of the maxillary sinus to reach the maxillary bone and inferior extension to the level of the hard palate are usually achieved by 9 years of age, with progressive pneumatization sometimes occurring until early adulthood. The anterior ethmoid air cells are also present at birth and grow until late puberty. Ethmoid pneumatization progresses in the posterior, inferomedial, and inferolateral directions until early adulthood. The sphenoid bone initially contains red marrow at birth, and conversion to fatty marrow occurs during the first 2 years of life. Subsequently, the sphenoid sinus becomes progressively pneumatized until it reaches adult size by approximately 14 years of age. The frontal sinus is the last to develop, developing from the anterior ethmoid air cells. The earliest frontal sinus pneumatization occurs around 2 years of age, by 4 years of age the frontal sinus reaches half of the height of the orbit, and by 10 years of age the frontal sinuses extend into the vertical portion of the frontal bone.[8]

Orbital fracture types vary with age, in part secondary to normal variant development of the paranasal sinuses and nasal cavities. The height of the lateral nasal wall depends on the development of the ethmoid and maxillary sinuses, and the height of the lateral nasal wall determines the height of the orbit.[9] The infant typically has relative frontal

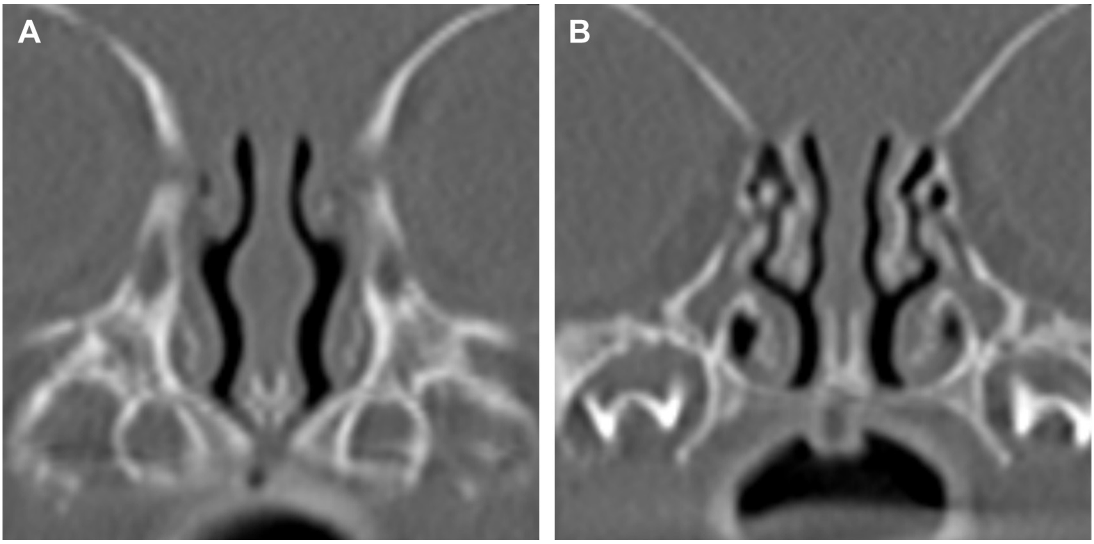

Fig. 10. Normal anterior skull base ossification at birth. (*A, B*) Coronal CT images in a newborn demonstrate the normal unossified appearance of the anterior skull.

bossing, which protects the orbital structures, but results in orbital roof fractures being more common than orbital floor fractures in the younger aged children. Furthermore, fractures of the supraorbital rim, with or without extension across the anterior cranial floor or orbital roof, are more common in younger children, and as the frontal sinuses develop, there is increased frequency of isolated frontal bone and frontal sinus fractures.[9]

Paranasal sinus development

Ethmoid sinuses present at birth, mature size by young adulthood

Maxillary sinuses present at birth, mature size by early teenage years

Sphenoid sinus absent at birth, begins development around 2 years of age, mature size by early teenage years

Frontal sinus last to develop, begins aeration at 2 years of age, mature size by early teenage years

DISTRIBUTION AND CAUSES OF PEDIATRIC FACIAL FRACTURES

Overall, facial fractures are less common in children than adults, with less than 15% of all facial fractures occurring in children. The lowest prevalence of pediatric facial fractures occurs in infants.[10] The prevalence of pediatric facial fractures, therefore, increases with age. There are two peaks of facial fracture, one at 6 to 7 years of age, correlating with the time when many children start attending school, and the other at 12 to 14 years of age, thought to be related to increasing physical activity and participation in sports.[10,11] In addition, there is a predominance of boys affected by facial fractures, with a ratio of up to 8.5:1.[10,12] The primary causes of pediatric facial fractures in descending order of frequency are motor vehicle accidents; sports-related injury; and accidental causes, such as falls, and violence.[12]

The frequency of different types of facial fractures in children varies in the literature, with most studies showing that mandible and nasal fractures are the most common, followed by maxillary/zygoma fractures. Although nasal fractures are common, septal hematomas remain rare, but of significant importance because when they occur, they require immediate surgical drainage to prevent septal cartilage necrosis, saddle nose deformity, and, in the young child, midface growth retardation.[10] The low incidence of mandible fractures in children younger than the age of 4 years is thought to be secondary to the relative increase in strength of the mandible at this age, which is at least in part secondary to the presence of unerrupted dentition. Incomplete dentition, with tooth buds still present within the maxilla and mandible, provides stability and resistance to fracture. In addition, children are thought to be relatively resistant to facial fractures because of more flexible suture lines, greater elasticity/flexibility of the osseous structures of the face, and a thicker layer of protective subcutaneous fat typically present in the pediatric face.[10,12]

When mandibular fractures occur in children, they are more likely to be unilateral fractures than in their adult counterparts. In children younger

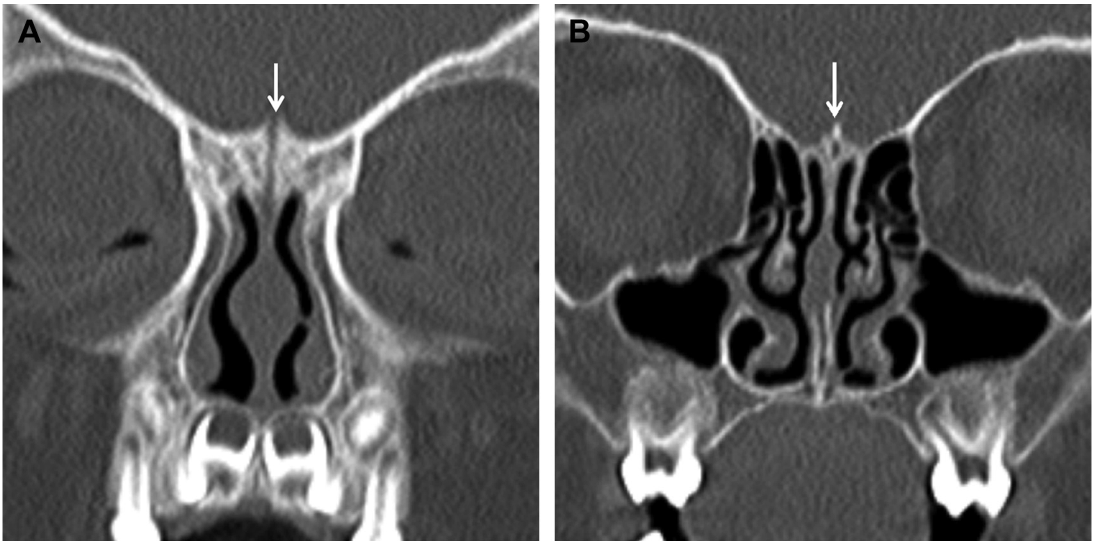

Fig. 11. Normal anterior skull base ossification at 2.5 years of age. (*A*) Coronal CT shows complete ossification of the anterior skull base with the exception of visible margins of the foramen cecum (*arrow*). (*B*) Coronal CT in the same child, 1 cm posterior to the foramen cecum, shows complete ossification of the floor of the anterior cranial fossa, on either side of the crista galli (*arrow*).

than 6 years of age, condylar fractures are typically intracapsular, whereas in older children they are more commonly extracapsular and involve the condylar neck. Subcondylar fractures with a greenstick fracture of the mandibular neck are common in children. CT imaging with multiplanar reconstruction and three-dimensional reformatted images are helpful in identifying mandible fractures in the young child, because many of the fracture lines are difficult, if not impossible, to see on conventional radiographs (**Fig. 13**).

Orbital floor fractures may be simple or comminuted and may occur as an isolated fracture or in association with other facial fractures. Orbital floor fractures are rare in children younger than the age of 5 years, increase in frequency as children get older, and do not exceed upper orbit fractures in frequency until after the age of 7.1 years.[13] In

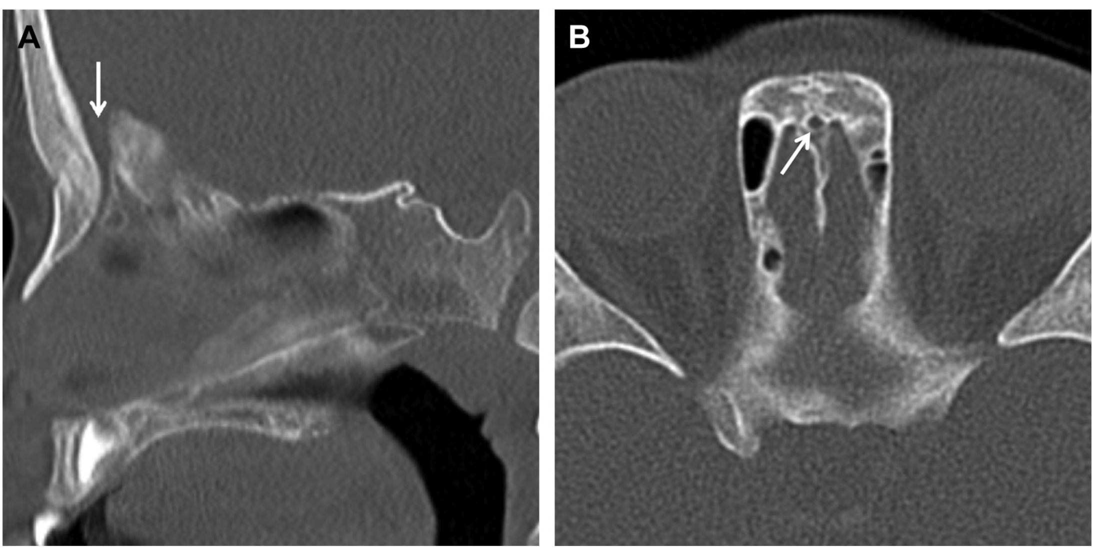

Fig. 12. Foramen cecum. (*A*) Sagittal CT reformatted image in a 2.5-year-old girl demonstrates the normal, small unossified foramen cecum (*arrow*), anterior to the ossified crista galli. (*B*) Axial CT images demonstrate the tiny, well-defined round remnant of unossified foramen cecum (*arrow*).

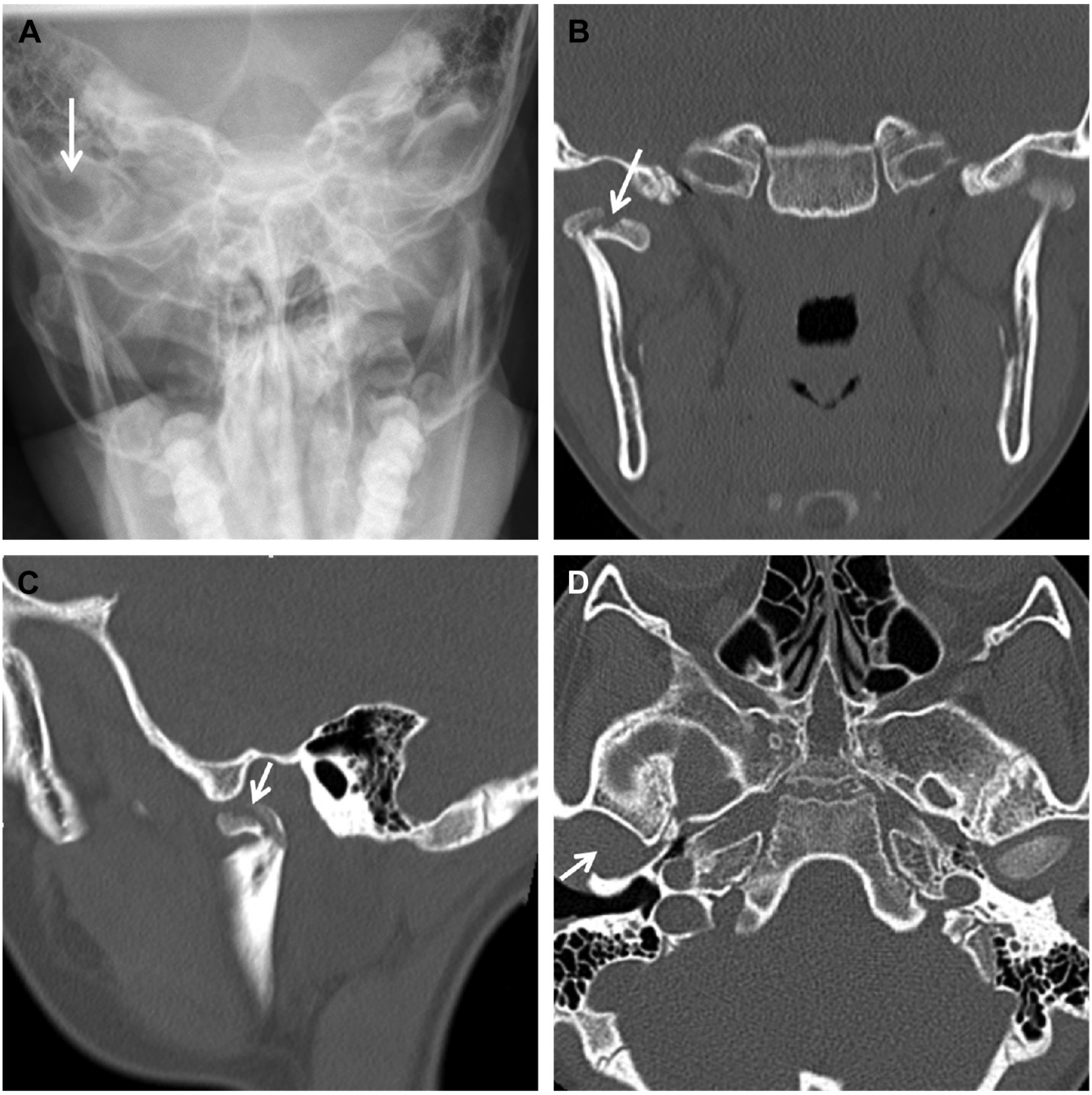

Fig. 13. Mandibular condyle fracture. A 13-year-old girl fell from her bike and has jaw pain with decreased range of motion. (*A*) Towne view from a mandibular radiograph series shows a more lucent right condylar fossa (*arrow*) when compared with the left, but no definite fracture (oblique mandible films were suboptimal in positioning and are not shown). (*B*) Coronal and (*C*) sagittal bone window reformatted CT images in the same child clearly demonstrate a comminuted right mandibular condyle fracture (*arrows*) with anterior and medial displacement of fragments out of the condylar fossa. (*D*) Axial CT bone window image at the level of the condylar fossa demonstrates the empty condylar fossa on the right (*arrow*). Care must be taken not to miss this finding, which may be the only indication of mandibular condyle fracture on axial head CT.

1957, Smith and Regan[14] first described a blowout fracture as one in which the orbital floor was fractured, but not the infraorbital rim. This is typically the result of a blow to the orbit by an object that is larger than the bony orbit, with the force absorbed by the orbital rim and transmitted to the orbital walls. The inferior and medial walls are most susceptible to fracture. With increased pressure on the intraorbital contents, there is a resultant "blowout" of the fractured inferior or medial orbital walls. A pure orbital floor blowout fracture spares the inferior orbital rim, whereas an impure blowout fracture involves the inferior orbital rim. In the original report, patients presented with diplopia, enophthalmos, paresthesia in the distribution of the infraorbital nerve, and soft tissue injury. In older children and adults, orbital floor fractures are most commonly secondary to interpersonal altercations or motor vehicle accidents, but younger children usually sustain orbital floor fractures related to accidents, such as falls, and sporting injuries.[15] A particular type of orbital floor fracture that occurs in children more frequently than adults is the trapdoor fracture. This is a linear, hinged, orbital floor fracture that occurs secondary to relatively deficient mineralization of the orbital floor. If the minimally displaced fracture fragment springs back into its normal position it may cause entrapment of intraorbital soft tissues and/or extraocular muscle (**Fig. 14**). This may result in ischemia and necrosis; may lead to fibrosis and scarring; and may be responsible for persistent diplopia, even after surgical correction. In addition, the trapdoor fracture may be associated with oculocardiac reflex, which may cause headache, nausea and vomiting, bradycardia, and potential syncope. When this occurs, urgent surgical correction is indicated.[16,17] Despite the presence of restricted extraocular muscle movement, external signs of swelling and ecchymosis may be minimal, and therefore this has also been termed the “white-eyed blowout fracture.”[18]

The overall goal of treatment of craniofacial fractures in children is the same as adults (ie, to re-establish anatomy and function back to the preinjury state). However, the specific timing and choice of treatment may vary depending on the age of the child with respect to how much future residual growth is predicted, and the overall phase of dentition at the time of injury.[10,19] Children in general have greater osteogenic potential and heal faster, therefore anatomic reduction may be accomplished earlier and necessary immobilization times may be shorter. However, fracture immobilization and fixation may be more difficult than in adults, depending on the stage of dentition. For various reasons, deciduous teeth may not be ideal for placement of fixation devices, and care must be taken not to injure intraosseous tooth buds and erupting teeth while trying to place fixation screws and plates.[10]

A few specific causes of craniofacial trauma in children deserve special attention, namely trauma secondary to toppled furniture, inflicted injury/child abuse, injuries related to ATV accidents, and impalement injuries.

Distribution of pediatric facial fractures

- Facial fractures much less common in children than adults
- Lowest prevalence of pediatric facial fractures occurs in infants
- Two peaks in prevalence of pediatric facial fractures: 6 to 7 years and 12 to 14 years of age
- Predominance of boys affected by pediatric facial fractures, up to 8.5:1 boys/girls
- Mandible and nasal bone fractures more common than maxillary/zygoma fractures
- Unilateral mandibular fractures more common in children than adults
- Trapdoor orbital floor fracture more common in children than adults

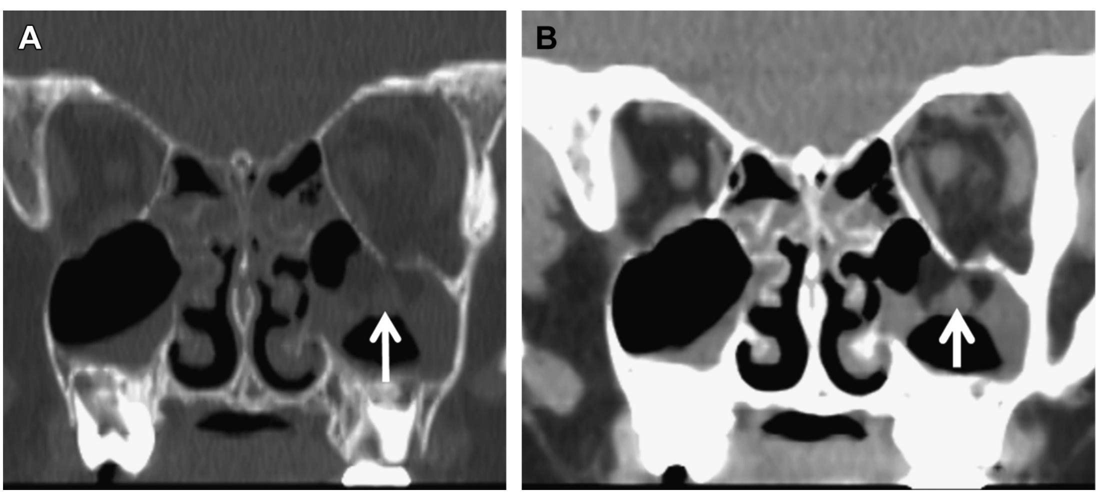

Fig. 14. Trapdoor orbital floor fracture. A 5 year old involved in an ATV accident, with complete inability to move the left eye upward. (*A*) Coronal reformatted bone window orbit CT image shows a minimally displaced left orbital floor fracture with mixed attenuation material extending into the superior left maxillary sinus (*arrow*). (*B*) Coronal reformatted soft tissue window image at the same level clearly defines the inferior rectus muscle (*arrow*) and fat herniated into the upper maxillary sinus.

TOPPLED FURNITURE

Much has been published in the literature on the topic of pediatric injuries related to toppled furniture.[20–29] These are almost universally preventable injuries and therefore there have been many attempts by the medical community and manufacturers to educate the consumer, with warning labels about securing furniture with appropriate straps and wall mounts, and recommendations about appropriate television stands. Despite these attempts, childhood injury from tipover or toppled furniture is a significant problem, and because of the weight of toppled furniture relative to small children, injuries can be severe and sometimes fatal. Televisions and clothes dressers/armoires are frequently the offending agents, and televisions placed on top of dressers, chests, or armoires account for nearly half of all injuries related to toppled televisions.[21,24,26]

Many years ago, cathode ray tube televisions were bulky and difficult to tip over. More recent changes in television construction have resulted in continued increase in pediatric injury related to toppled television. The flat-panel television sets

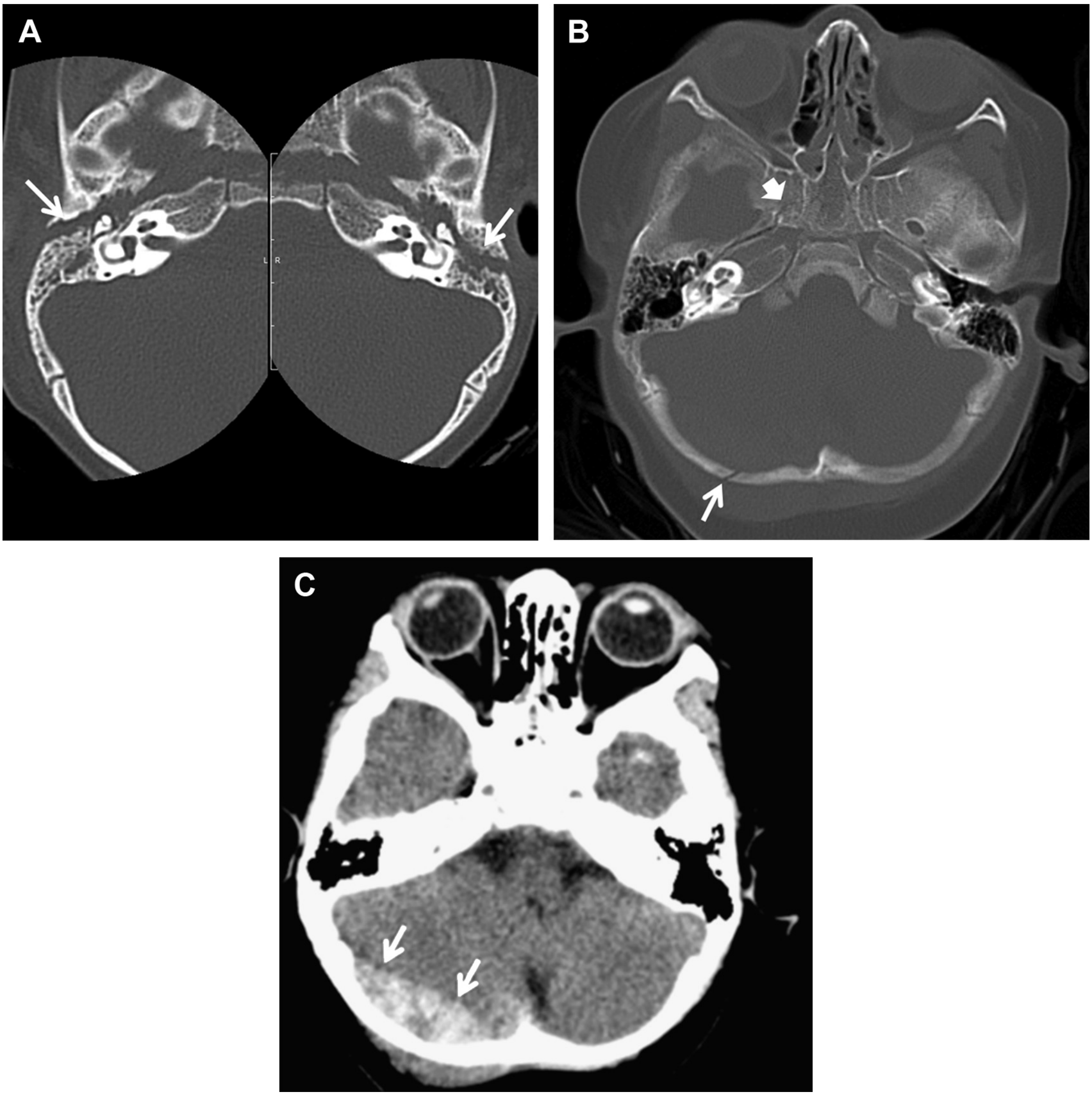

Fig. 15. Television tipover injury. (*A*) A 3 year old suffered fatal head injury secondary to 22-inch television falling on her head. Axial temporal bone CT demonstrates widely diastatic bilateral temporal bone fractures (*arrows*). (*B*) A 2-year-old boy was climbing on a dresser that supported a television when the television fell and landed on the child's head. Axial bone window CT images demonstrate nondisplaced fractures of the right occiput (*arrow*) and sphenoid base (*arrowhead*). (*C*) Axial soft tissue CT image in the same child as in *B* better identifies a lenticular-shaped high-attenuation venous epidural hematoma (*arrows*).

are larger and more slender in shape, frequently have a narrow base with a center of gravity that is more toward the front of the television, and are rarely secured to the object on which they rest. Televisions are reportedly present in 96.7% of households in the United States, and children reportedly watch more than 28 hours per week of television.[28] This incredibly frequent exposure to a television provides innumerable opportunities for children to suffer from television-related injuries, particularly because children understandably do not recognize the danger of climbing on unstable furniture. In a large-scale 22-year study by De Roo and colleagues, 17,313 children required emergency treatment of television-related injuries each year, and the rate of injury attributable to falling televisions increased by 95% over the 22-year period. The median age of patients was 3 years and 64.3% of patients were younger than 5 years. Lacerations and soft tissue injuries were most common but concussions and closed head injuries represented 13.3% of injuries among children younger than 5 years and 7.7% of injuries among patients aged 11 to 17 years.[30] Murray and coworkers[25] reported 42,122 injuries and found the injury rate to be highest for children 1 to 4 years of age; most injuries in that group of children involved the head and neck. Television-related crush injury frequently results in calvarial and skull base fractures, including involvement of the orbital roofs, sphenoid bone, and temporal bones (**Fig. 15**).[22,23,27] Mortality rates from television tipover injuries are reported between 1.9% and 20%, depending on the series,[20,24] with most deaths occurring in children younger than 3 years of age secondary to traumatic brain injury.[20]

NONACCIDENTAL, INFLICTED TRAUMA, AND CHILD ABUSE

Mandibular fractures are exceedingly uncommon in infants. When mandibular fractures do occur in infants, unilateral fractures are more common than bilateral fractures. Because they are so uncommon, recognition of such injury should at least raise suspicion for a direct blow related to child abuse, particularly if the mechanism of reported injury is incongruous with the resultant fracture.[19,31,32] In addition, because infants are unable to verbalize jaw pain, and signs of external trauma may be lacking, imagers should be sure to inspect the mandibular condyles and condylar fossa on head CTs performed for evaluation of suspected child abuse, because fractures of the mandibular condyle, with or without displacement of the condyle into the middle cranial fossa, may be clinically silent in young patients (**Fig. 16**).

ATV ACCIDENTS

With increasing popularity of ATV use comes an increase in ATV-related accidents, with an

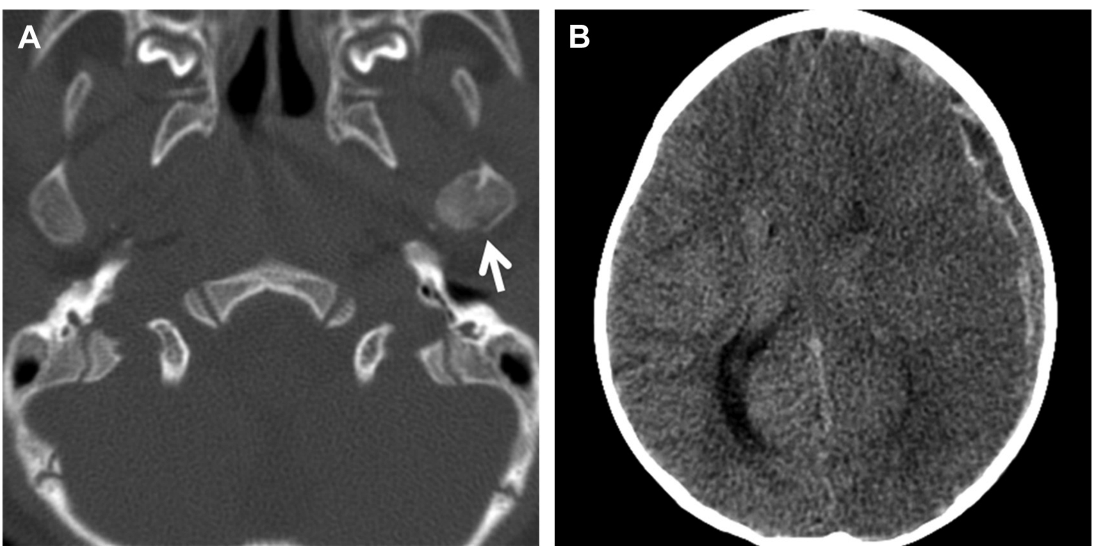

Fig. 16. Mandibular condyle fracture secondary to fatal nonaccidental trauma. (*A*) Axial bone window CT image obtained for evaluation of a 9 month old who was found down, with blown pupils, demonstrates a mildly displaced left mandible condyle fracture (*arrow*). (*B*) Axial soft tissue head CT image shows a large, mixed attenuation left-sided subdural hematoma with significant left-to-right shift of midline structures, and diffuse hypodensity and ill-definition of the left cerebral hemisphere gray-white differentiation consistent with hypoxic/ischemic injury. This unfortunate child also sustained parietal and occipital bone fractures and splenic and liver lacerations (not shown).

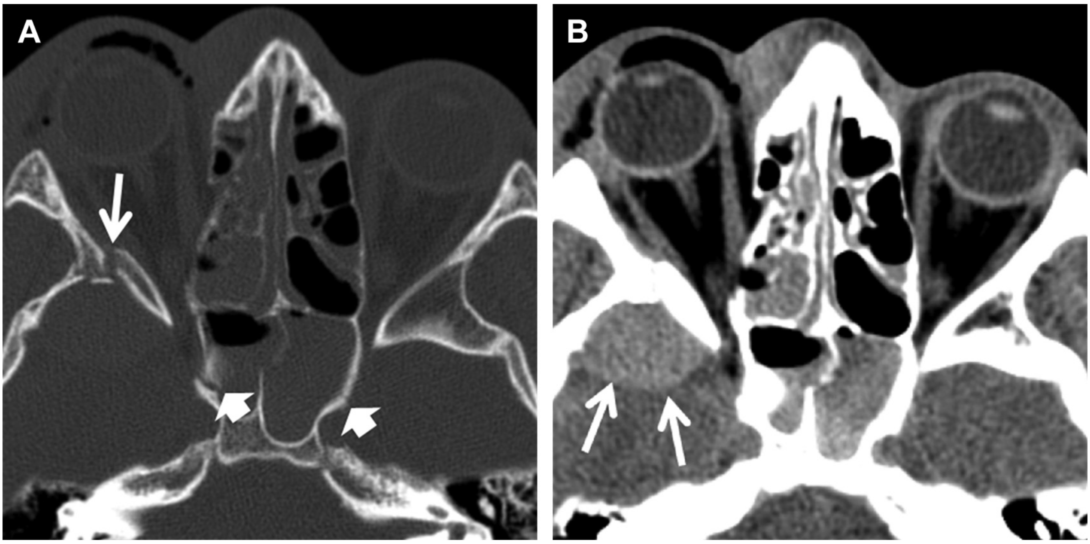

Fig. 17. ATV facial fractures. (*A*) Axial bone window CT image in a teenager who suffered craniofacial and intracranial trauma in an ATV accident shows fractures of the right greater sphenoid wing (*arrow*) and bilateral sphenoid sinus walls (*arrowheads*) with associated preseptal soft tissue gas and subtotal opacification of the ethmoid and sphenoid sinuses. (*B*) Axial soft tissue window CT image in the same patient shows to better advantage the associated right preseptal orbital soft tissue swelling and the right middle cranial fossa epidural hematoma (*arrows*). This child unfortunately sustained multiple additional sites of facial fracture and intracranial hemorrhage, in addition to bilateral carotid artery dissections and right cavernous-carotid fistula (images not included). (*Courtesy of* Carl Pergam, MD, Tucson, AZ.)

associated increase in pediatric craniofacial trauma (**Fig. 17**). A total of 40% of all ATV-related fatalities occur in pediatric patients, and many of these children die from head and neck injuries. Prigozen and coworkers[33] reviewed 26 children with a mean age of 13.1 years with craniofacial injuries secondary to ATV accidents. A total of 65% of them were drivers of the ATV. Injuries most frequently occurred secondary to loss of control/rollover accidents, falls from the vehicle, and collision with stationary objects. Fractures of the facial bones and skull occurred in 77%. Midface injuries

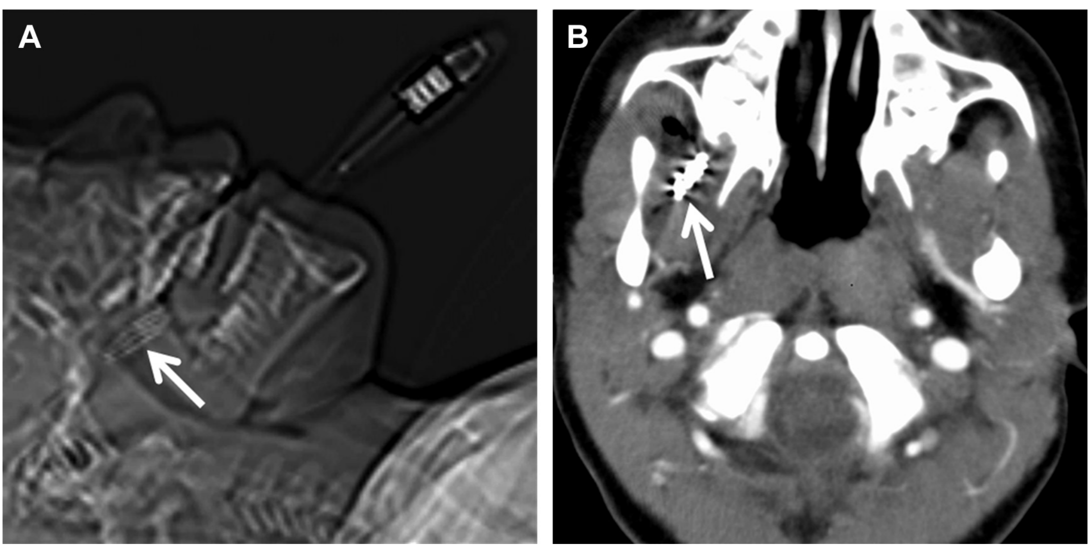

Fig. 18. Oral cavity impalement. Toddle fell while running with a toothbrush in her mouth. (*A*) Scout radiograph from face CT shows the toothbrush protruding from the child's mouth with the bristles of the brush overlying the oropharynx (*arrow*). (*B*) Axial soft tissue CT image clearly shows the bristle portion of the brush (*arrow*) embedded within the right masticator space with surrounding soft tissue gas. (*Courtesy of* Carl Pergam, MD, Tucson, AZ.)

were the most common and isolated craniofacial fractures of mandible, maxilla, nasal, or orbital bones were uncommon, occurring in only 20% of patients. Most patients suffered two or more concomitant facial fractures. A total of 35% of children had closed head injuries, and in these children, there was a significant association with mandible fractures. Patients sustaining mandibular fractures were nearly 13 times more likely to have associated closed head injury and tended to have a longer hospital stay. Only 8% of children were helmeted. Although not scientifically proved, it would seem intuitive that the use of helmets, particularly with face protection, would prevent many of these injuries. There have been multiple efforts by the medical community to educate consumers and dealers about the dangers of riding ATVs, and there have been increasing regulations with respect to ATV use. However, many parents and children fail to follow the recommended injury prevention measures, and therefore there has been a continued increase in the number of injuries and deaths in children involved in ATV-related accidents in recent years.[34–36]

IMPALEMENT INJURIES

Impalement injuries involving the oral cavity in children are common, especially in toddlers who fall while carrying objects in their mouth, most

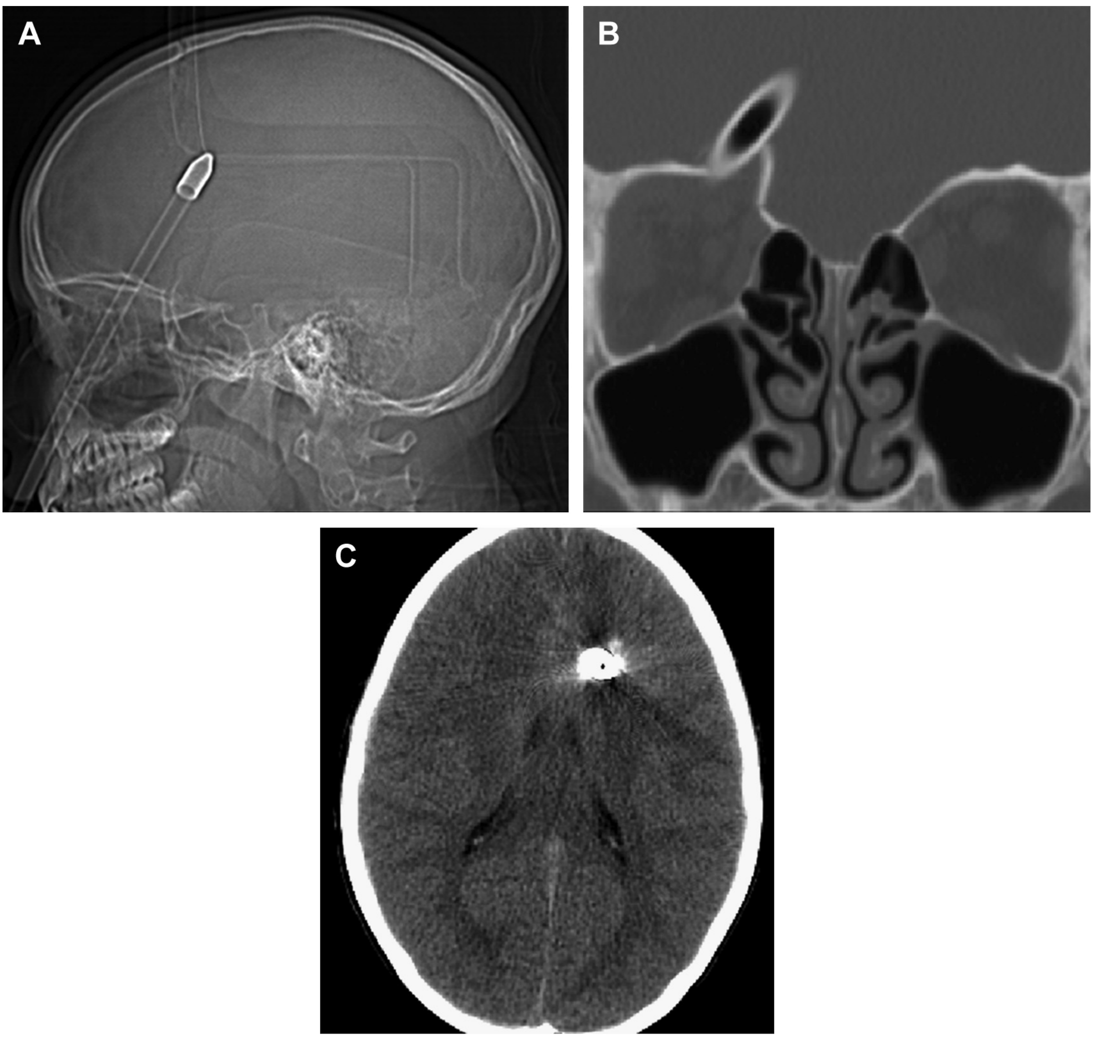

Fig. 19. Orbital impalement with intracranial extension. (*A*) Scout radiograph from head CT demonstrates a radiodense distal end of an arrow on which this 8 year old impaled his orbit. (*B*) Coronal bone window CT image shows to better advantage the trajectory of the hollow plastic shaft of the arrow traversing the fractured orbital roof. (*C*) Axial soft tissue window head CT image clearly shows the tip of the arrow within the contralateral left frontal lobe.

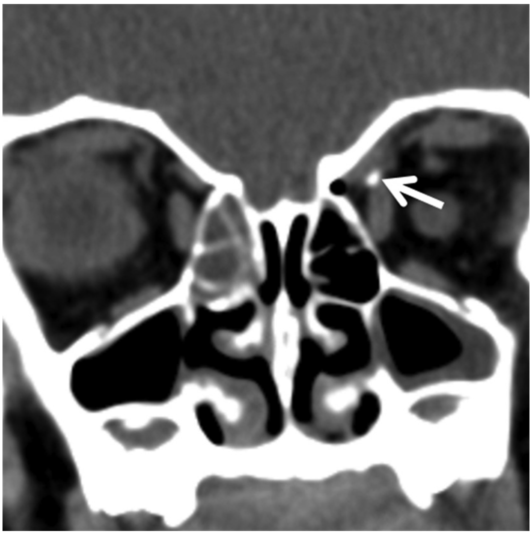

Fig. 20. Orbital impalement with retained intraorbital pencil graphite. A 5 year old fell on a pencil, had upper eyelid puncture wound, and suspicion of retained pencil fragments. Coronal soft tissue window CT image shows high-attenuation graphite fragment (*arrow*) along the superior margin of the left medial rectus muscle. In addition, there is extraconal soft tissue swelling, edema, and hemorrhage superior to the fragment, inseparable from the superior oblique muscle.

commonly sticks, writing instruments, toothbrushes (**Fig. 18**), cylindrical toys, and straws. Most children sustain injury to the palate, posterior oropharynx, tonsillar region, or cheek, with fewer injuries involving the tongue and floor of mouth. Most patients are treated conservatively, without complications, and only a minority of patients requires imaging.[37–39] Matsusue and coworkers[38] reviewed 144 children with oral cavity impalement injury. The impaled objects were toothbrushes in 20.8%, cylindrical toys in 18.8%, and chopsticks in 13.2%. The soft palate was involved in 44.4% and the hard palate was involved in 18.1%. CT examination was performed in 11.1%, admission was required in only 8.3%, most healed spontaneously or with minimal intervention, and there were no complications of deep infection or neurologic sequelae. However, there are occasional reports of complications, such as deep neck infection, life-threatening hemorrhage, internal carotid artery thrombosis, mediastinitis, and airway complications.[39–42]

Occasionally with oral cavity injuries, and frequently with other craniofacial impalement injuries (particularly those involving neck and orbit), the entrance wound is the only site of clinically evident trauma and imaging may be necessary to determine trajectory of the puncture injury and the total extent of injury (**Fig. 19**). Furthermore, imaging is helpful in children (and adults) who have sustained an impalement injury when there is suspicion of retained foreign body, for instance when the offending object is broken and the missing piece cannot be located (**Fig. 20**). CT is the primary imaging modality of choice in these patients, to identify the path of penetration, the presence or absence of retained foreign body, and any associated craniofacial fractures and/or intracranial

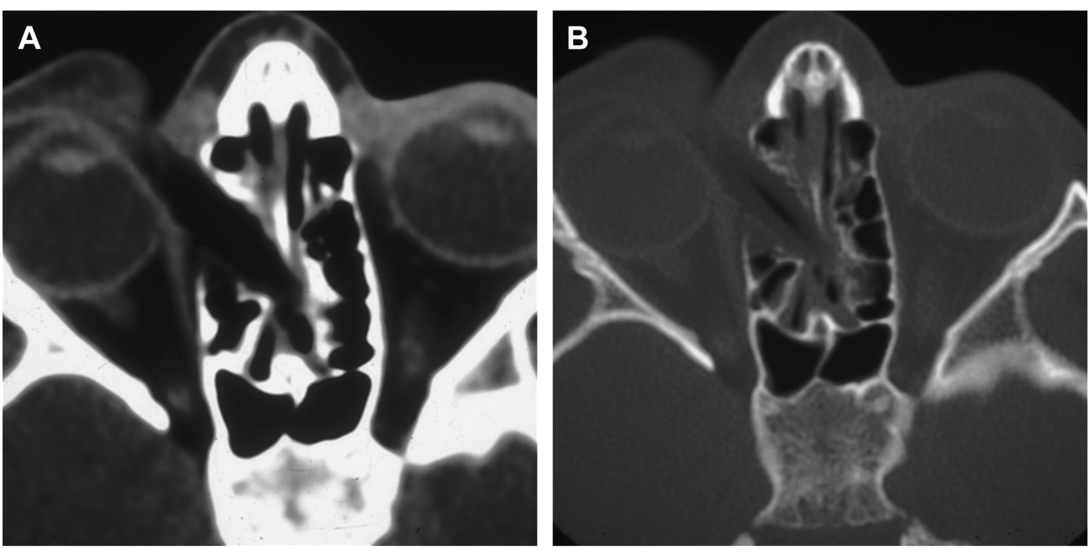

Fig. 21. Orbital impalement CT appearance of wood. (*A*) Axial soft tissue window CT image of an 11-year-old boy who fell on a twig, demonstrates a large linear orbital foreign body of air attenuation, deviating the globe laterally and traversing the left ethmoid air cells. (*B*) Axial bone window CT image at the same level demonstrates the periphery of the twig to be soft tissue attenuation and the central portion air attenuation.

injury.[43] Wood foreign bodies, such as remnants of twigs or pencils, may be particularly difficult to recognize, because early on they may have the attenuation of air (**Fig. 21**), becoming intermediate in attenuation and inseparable from surrounding soft tissues as they absorb moisture.[44–47] In addition, the CT appearance depends on the window/ level setting at which the image is viewed.

SUMMARY

When comparing craniofacial trauma in children with adults, there are several differences, primarily related to the mechanisms of injury and timing of the injury with respect to the normal growth and development of the pediatric face and skull base. Older children and adolescents are more similar to adults with respect to mechanism of injury and distribution of facial fractures, whereas younger children have a lower incidence of facial fractures and different distribution of fractures when they do occur. The lack of paranasal sinus pneumatization and presence of incomplete dentition impart a relative increased strength to the osseous structures of the pediatric face. The difference in size of the craniofacial ratio and relative frontal bossing in young children also contribute to the pattern of facial fractures in children. A few specific causes of craniofacial trauma in children are of particular interest, including injuries related to toppled furniture, nonaccidental trauma, ATVs, and those secondary to craniofacial or neck impalements.

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