Craniofacial Growth and Development
A Primer for the Facial Trauma Surgeon

Raquel Capote, DMD, MSD, MPH[a,b,*], Kathryn Preston, DDS, MS[c,d], Hitesh Kapadia, DDS, PhD[e,f,g]

KEYWORDS

• Craniofacial • Maxillofacial • Craniomaxillofacial • Facial • Pediatric • Growth and development • Trauma • Fractures

KEY POINTS

- The craniofacial complex follows a cephalocaudal gradient of development. The skeleton grows differentially with different regions reaching adult dimensions at different times.
- Trauma to growth centers and growth sites during childhood can disrupt normal development. Intraoperative soft tissue damage, periosteal stripping, scar formation, and rigid fixation may also affect facial growth.
- The cranial vault after age 5 years and the orbit after age 7 years are generally of adult size and definitive reconstruction may be achieved. The maxilla, mandible, and nose do not achieve their adult dimensions until the adolescent pubertal growth spurt.
- Surgical treatment before skeletal maturity is less predictable and may require reoperation following skeletal maturation.
- It is critical to educate the patient/family on the potential for growth disturbances with long-term follow-up recommended until growth is completed.

INTRODUCTION

The craniofacial complex comprises the neurocranium, face, and oral apparatus, representing a morphologic and multifunctional region fundamental to being human. Structures develop through a dynamic process that begins in utero and reply upon complex temporally and spatially coordinated tissue interactions.[1] Craniofacial growth is characterized by cephalocaudal and allometric patterns.[2] Growth of tissues such as bone, cartilage, and muscle as well as highly specialized organs such as the brain and teeth contribute to the development of structures and creation of spaces supporting multiple functions.

The process of craniofacial growth is controlled by genetics, influenced by environmental factors.[3] Trauma can lead to dysmorphology as a result of the disruptive injury, secondary to the treatment, and/or subsequent growth and development.[4] The pediatric craniofacial complex exhibits distinct anatomic and physiologic differences compared to adults. It is important to identify not only the most appropriate intervention but the

[a] Department of Oral and Maxillofacial Surgery, Vanderbilt University Medical Center, Nashville, TN, USA; [b] Cleft and Craniofacial Program, Monroe Carell Jr. Children's Hospital at Vanderbilt, Nashville, TN, USA; [c] Center for Cleft and Craniofacial Care, Phoenix Children's Hospital, Phoenix, AZ, USA; [d] Department of Orthodontics, Arizona School of Dentistry & Oral Health, A.T. Still University, Mesa, AZ, USA; [e] Craniofacial Center, Seattle Children's Hospital, Seattle, WA, USA; [f] Division of Plastic Surgery, Department of Surgery, University of Washington, Seattle, WA, USA; [g] Department of Orthodontics, School of Dentistry, University of Washington, Seattle, WA, USA

* Corresponding author. Department of Oral and Maxillofacial Surgery, Vanderbilt University Medical Center, T-4323A Medical Center North, 1161 21st Avenue South Nashville, TN 37232-2596.
E-mail address: raquel.capote@vumc.org

Oral Maxillofacial Surg Clin N Am 35 (2023) 501–513
https://doi.org/10.1016/j.coms.2023.04.007

oralmaxsurgery.theclinics.com

optimal time to surgically intervene for best outcome while minimizing detrimental impacts on subsequent growth. An understanding of craniofacial growth and development is critical to the management of pediatric facial trauma.

CORE CONCEPTS OF NORMAL GROWTH AND DEVELOPMENT

Concept 1: Growth Pattern

Craniofacial growth is expressed as a patterned series of changes in size, shape, and location.[5] At birth, the head is large in relation to the rest of the body and the cranium is disproportionately large relative to the face, following a cephalocaudal growth gradient and reflecting the dominance of brain development.[1] The newborn skull is given over to the neurocranium. The eyes, outgrowths of the forebrain, are relatively large. The midface and lower third of the face are diminutive and take a more retruded position (**Fig. 1**). With growth and development, the face "catches up" to cranial development, increasing susceptibility to midface and mandibular trauma.

Concept 2: Differential Development and Maturation

The complexity and diversity of the skull arises because the constituent bones enlarge differentially.[1] Facial growth occurs in 3 planes with the transverse dimension completed first, followed by completion of horizontal growth, and finally by vertical facial growth that continues into adulthood.[6–9] The facial region demonstrates variation in growth rates during development (**Table 1**[10–18] and **Fig. 2**). In particular, the mandible exhibits rapid growth around puberty. Surgical treatment performed before skeletal maturity is less predictable and may require reoperation following skeletal maturation.[14]

Concept 3: Mechanisms of Bone Growth: Drift and Displacement

There are 2 main mechanisms by which bone growth occurs: (1) drift (surface remodeling) through bone formation on one side and resorption on the other that produce a change in the size and shape of the bone and result in a change in location in the direction of external bony deposition; (2) displacement (translation) is the movement of the whole bone as a unit to a new location caused by the growth of adjacent structures. The overall direction of growth is the cumulative effect of displacement and drift[19] (**Fig. 3**).

Concept 4: Growth Centers versus Growth Sites

Fundamental to the understanding of craniofacial growth is an appreciation of growth centers and growth sites.[20,21] Areas of the growing skeleton that are primarily under the control of heredity are referred to as growth centers. Growth centers exhibit intrinsic, independent growth potential and active tissue separating force. Examples are synchondroses uniting endochondral bones in the cranial base and the nasal septal cartilage.

In contrast, a growth site is an area of skeletal growth that occurs secondarily and grows in compensatory fashion due to growth in a proximate location. Growth sites exhibit passive filling-in and/or remodeling in response to extrinsic forces or functional demands imposed by adjacent structures. This concept falls closely in line with the functional matrix theory. The brain, eye, and tooth may be considered the functional matrix of the cranial vault, orbits, and alveolus, respectively. The sutures connecting intramembranous bones found in the cranial vault and face and the periosteum are growth sites. It has been previously believed that the mandibular condyle is a primary

Fig. 1. Comparison between neonate and adult skull.

Table 1
Average percentage growth completion of various craniofacial structures

	Average % of Growth Completed by Age 1	Average % of Growth Completed by Age 5	Average Age at Maturity in Years
Cranial vault	84–88	93–96	Males: 14–15 Female: 13–14
Orbits	77–86	87–93	Males/females: 7 variable
Zygoma	72–74	83–86	Males: 15 Female: 13
Maxilla	75–80	85	Males: 16 Female: 14
Mandible	60–70	74–85	Males: 18–20 Female: 14–16

Data from Refs[10–18].

growth center although now the condyle is not credited as the sole determinate of mandibular growth potential. It is considered a growth site that is necessary for mandibular development and is influenced by intrinsic and extrinsic factors.[22]

NEUROCRANIUM

The neurocranium is the part of the skull that encloses the brain and includes the cranial base and cranial vault.

Cranial Base

The cranial base forms the inferior aspect of the cranium and provides a platform from which the face grows.[1] At birth, the cranial base is short and the spheno-ethmoidal synchondrosis (SES) and the spheno-occipital synchondrosis (SOS) are patent (**Fig. 4**). Bidirectional growth occurs interstitially at the cartilaginous joints located between 2 bones of endochondral origin[23] (**Fig. 5**). The SES and SOS are growth centers and are major contributors to anteroposterior growth of the cranial base and displacement of the face. The maxilla articulates with the anterior cranial base and the mandible articulates with the temporal bone. Growth at the SES displaces the facial skeleton forward relative to the braincase. As the SES grows and elongates the anterior cranial base, the cranial vault expands and the ethmoid, zygomatic and palatine bones elongate, thereby increasing the size of the orbits and midface. As the SOS grows and elongates the middle cranial fossa, it displaces the glenoid fossa in the temporal bone posteriorly and inferiorly[24,25] while moving the face forward away from the vertebral column. This deepens the nasopharynx, creating more space for airway, muscles of mastication and room for growth of the ascending ramus of the mandible.

The greatest rate of increase in cranial base length and decrease in cranial base angulation occurs during the first 2 to 3 postnatal years.[26] The anterior cranial base is closer to its adult size than the posterior cranial base throughout postnatal growth. The SES fuses at about 7 years of age whereas the SOS continues to grow through adolescence. The SOS is the last synchondroses

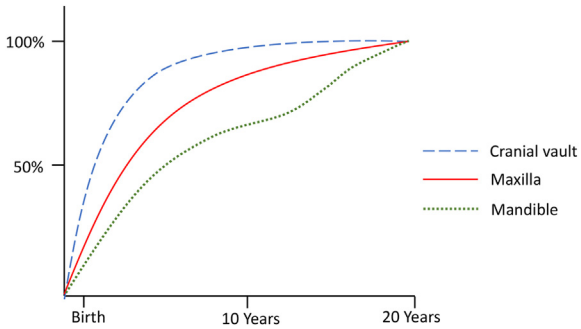

Fig. 2. Growth of the cranial vault, maxilla, and mandible from infancy to skeletal maturity. The cranial vault reaches maturity well before the midface and is followed by the mandible.

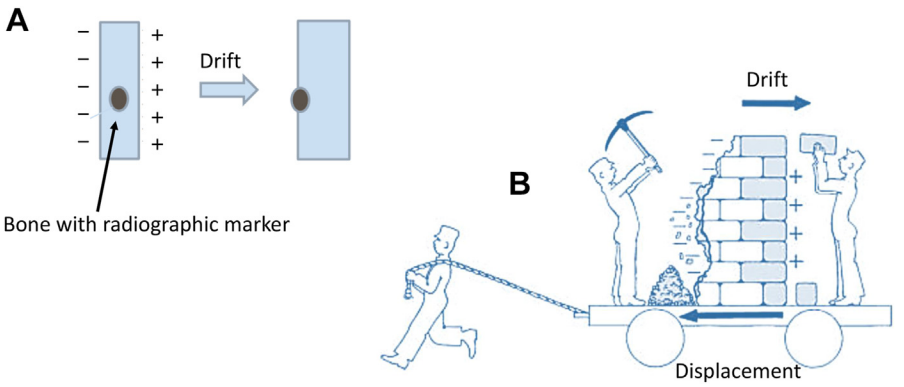

Fig. 3. Mechanisms of bone growth: drift and displacement. (*A*) Drift (surface remodeling). (*B*) Drift and displacement can occur in the same direction or in opposite directions as illustrated. (*From* Enlow D, Hans M. *Essentials of facial growth*. W. B. Saunders; 1996. (Figure 3B).)

of the cranial base to fuse with closure time occurring in girls at approximately 13 to 15 years of age and in boys around 15 to 17 years of age.[27] Once growth in the synchondroses ceases, the cartilage is replaced by bone to form synostoses.

Cranial Vault

The calvaria or cranial vault, together with the cranial base, encase and protect the brain. Like the cranial base, the most rapid postnatal expansion of the calvaria is during the first 2 years after birth.[23] However, unlike the cranial base, cranial sutures connecting the intramembranous bones of the calvaria are growth sites.[20]

The cranial vault is composed of paired frontal and parietal bones, the squamous parts of the temporal bone, and interparietal part of occipital bone that are separated by unossified sutures of fibrous connective tissues (**Fig. 6**). The cranial vault enlarges primarily as a result of compensatory growth at the sutural bone fronts stimulated by expansile growth of the neural elements.

Growth proceeds rapidly during the first 24 months after birth, secondary to the brain doubling in volume in the first 6 months and again by the second birthday.[28] The sutures normally remain patent and actively growing to keep pace as the brain expands. By age 5 years, the cranium is 90% of adult size.[15] After age 7 years, bony apposition on the outer surface of the frontal bone and development of the frontal sinus drifts the frontal bone and root of nose anteriorly.[23] The frontal sinuses that are absent at birth begin to develop at age 2 years, are radiographically detectable around age 6 to 7 years, and reach their full size after puberty.[29]

At birth, bones of the vault are thin, malleable, and unilaminar. As displacement of the individual flat bones of the cranial vault takes place, compensatory bone growth occurs at the sutures and by surface remodeling of the outer and inner cortex of the skull. An intervening diploë layer of spongy cancellous bone appears around age 4 years. By adulthood, the calvarial bones are thicker, rigid, and trilaminar. The calvaria bones remain separated by thin, periosteum-lined sutures for many years, eventually fusing in adult life.[30]

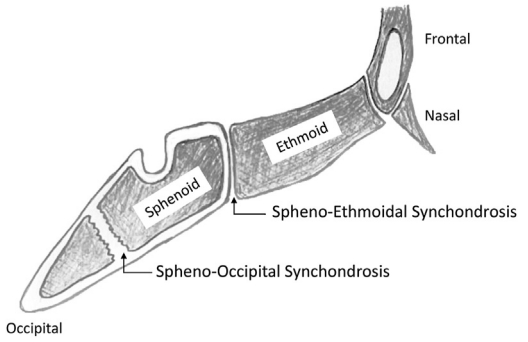

Fig. 4. Synchondroses.

FACE

The face incorporates different anatomic and functional spaces and is composed of numerous individual bones, several of which are paired and most developing intramembranously. The anatomy of the face is divided into 3 main regions: upper, middle (midface), and lower (mandible).

Upper Face

The upper face contains the forehead, eyes, and temporal region. The orbit is composed of bones from the cranium (frontal, sphenoid, ethmoid, lacrimal) and nasomaxillary complex (maxillary, zygomatic, and palatine bones). The sutures

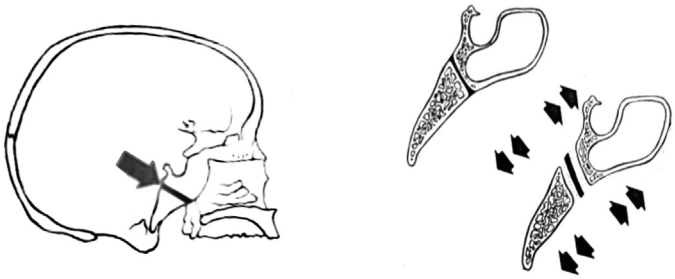

Fig. 5. Schematic drawing of a midsagittal view of the cranial base showing the spheno-occipital synchondrosis. Diagrammatic representation of the spheno-occipital synchondrosis. The spheno-ethmoidal synchondrosis, not shown, and the spheno-occipital synchondrosis exhibit a "bidirectional" pattern of growth. In other words, both bones at the joint increase in size as growth proceeds. (*From* Enlow D, Hans M. *Essentials of facial growth*. W. B. Saunders; 1996.)

between the bones of the eye are growth sites and, in a similar fashion to the cranial sutures and other facial sutures, are important sites of compensatory growth.

The orbits expand primarily in response to the rapidly developing eyeballs. This is greatest between birth and 2 years of age and contributes to anterior and lateral displacement of the midface. The orbits complete approximately half of postnatal growth by age 2 years.[18,31] Adult dimensions are nearly attained by 7 years of age,[18] after which the rate slows considerably until maturity.[32] There is inferior and lateral expansion in this region secondary to changes in the anterior cranial fossa and maxilla associated with midface displacement. The intercanthal width reaches full maturation at age 8 years in females and 11 years in males and the biocular width at 13 years in females and 15 years in males.[11] Bony apposition on the orbital floor offsets the anteroinferior displacement of the whole maxilla and contributes to midface height.[23]

Midface/Nasomaxillary Complex

The midface is connected to the neurocranium by a circummaxillary suture system and the midline

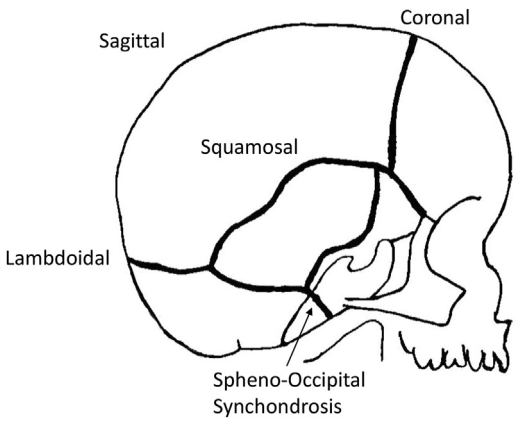

Fig. 6. Diagram of the calvarial sutural complex. Notice that peripheral sutures coalesce as they course inferiorly and medially, ending in the spheno-occipital synchondrosis.

by the nasal septum.[23] The nasal septum consists of the perpendicular plate of the ethmoid bone, septal cartilage, and vomer.[33] The nasal septum cartilage is continuous with the perpendicular plate of the ethmoid bone in the anterior cranial fossa at its caudal end and is firmly attached to the anterior nasal spine of the premaxilla through the septospinal ligament. The nasal septum cartilage is a growth center affecting vertical and sagittal growth of the nose and maxilla.[33–35] The majority of nasal growth occurs in 2 growth spurts, between 2 to 5 years of age and again at puberty.[35,36] Growth is usually completed by age 16 to 18 years in girls and 18 to 20 years in boys, although additional growth of the nasal septum may continue thereafter.

The maxilla moves downward and forward relative to the cranial base, accompanied by the orbits and nasal cavity, with each of these structures increasing in volume as they grow.[1] The zygomatic arches also grow laterally and are relocated in a posterior direction within the face. The zygomatic-arch length is 83% of adult length at 5 years of age. By 5 years of age, the bizygomatic width is 86% and the midfacial width is 89% of adult width.[15] The zygomatic bones provide midface width, cheek definition, and shape/definition to the lateral and inferior orbital borders.

The maxilla grows by (1) bony apposition at the circummaxillary and intermaxillary sutures compensatory to midfacial displacement and (2) surface remodeling (drift). Growth at the cranial base and nasal septum results in downward and forward displacement of the nasomaxillary complex followed by bony apposition at the circummaxillary and intermaxillary sutures[13,23] (**Fig. 7**). The facial aspect of the premaxillary–maxillary suture is partially ossified at birth, whereas the palatal region tends to close by age 6 years, although variability of complete suture obliteration with age has been reported.[37–39] An increase in maxillary width is achieved predominantly through growth of the midpalatal suture, with a smaller contribution from external remodeling. The midpalatal maxillary suture has been reported to close

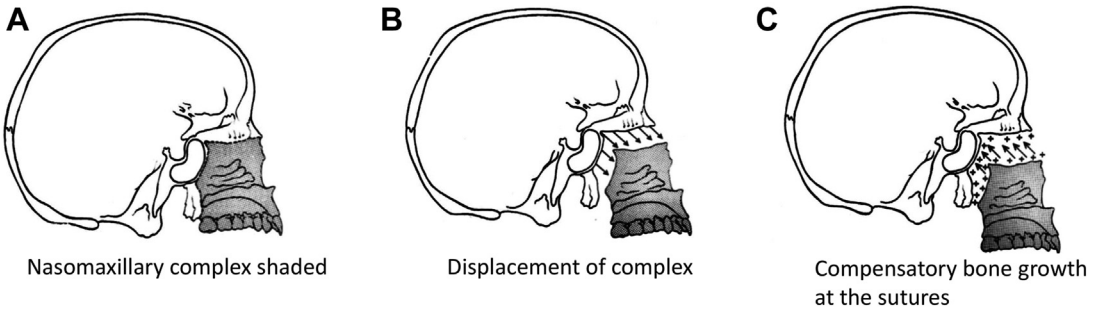

Fig. 7. Maxillary growth. The whole maxillary region is displaced downward and forward away from the cranium. This then triggers new bone growth at the various sutural contact surfaces between the nasomaxillary complex and the cranial floor. (*From* Enlow D, Hans M. *Essentials of facial growth*. W. B. Saunders; 1996.)

between 15 and 19 years of age.[40] Sagittal growth of the maxilla continues until about 14 years of age in females and 16 years of age in males.[19]

A complex pattern of bone resorption and deposition occurs over the surface of the maxilla as it is displaced downward and forward within the face. Bone is deposited in the maxillary tuberosity region, contributing to an increase in length of the entire maxilla and creation of additional space for the developing dentition. Concomitantly, almost the entire anterior surface of the maxilla is an area of resorption. Growth and development of the maxilla parallels growth and pneumatization of the maxillary sinus.[41] Midfacial height increases due to the combined effects of inferior cortical drift and inferior displacement. The inferior translation of the maxilla is associated with bone resorption at the nasal floor (increasing the nasal cavity) and bony deposition along the hard palate.[6] The height of the midface is further increased by continued development of the dentition and alveolar bone. Vertical facial growth is the last dimension to be completed and continues into adulthood.

Mandible

The lower third of the face is composed of a single bone in the adult, the mandible. The mandible functions as a lever and a link for muscles involved in mastication, speech, and other oral functions. At birth, the right and left hemimandibles have not yet fused, the chin is rudimentary and retrusive, the gonial angle is obtuse, the ramus is short, both in absolute terms and in proportion to the corpus, and there is no appreciable alveolar bone. The developing primary teeth are discernible in their crypts on radiographs.

The mandible articulates at each glenoid fossa of the temporal bone in the middle cranial fossa.[13] As the whole mandible is displaced downward and forward relative to the cranial base, the condyle and ramus grow upward and backward (**Fig. 8**). Although all aspects of the mandible increase substantially in size, the paramount posterior-superior growth vector of the mandible is achieved through the combined processes of endochondral ossification at the condyle and surface remodeling at the ascending ramus.[13]

The cartilage of the mandibular condyle provides movable articulation, endochondral bone growth, and regional adaptive growth. Its responsiveness to mechanical, functional, and hormonal stimuli set it apart from primary cartilaginous growth centers.[21,23] Therefore, the secondary cartilage of the condyle is more consistent with the concept of an adaptive, compensatory growth site.[42]

The mandible increases in size by a combination of 3 growth processes: endochondral bone growth at the condyle; surface remodeling throughout, particularly on the posterior ramus; and dental

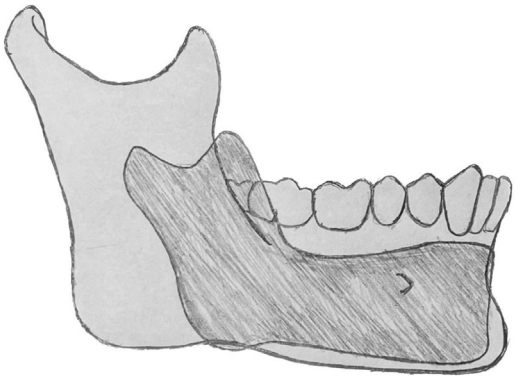

Fig. 8. Mandibular growth: biologically correct superimposition of the mandible registered on the inner table on the mandibular symphysis. The condyle and ramus elongate in a posterior and superior direction while the body of the mandible lengthens. There is little growth of the chin in the development of the mandible. (Adapted from Enlow D, Hans M. Essentials of facial growth. W. B. Saunders; 1996.)

eruption with development of alveolar bone.[13,43] Significant postnatal development is attributed to differential formation and modeling of bone along nearly the entire surface of the mandible, particularly along its superior and posterior aspects. The increase in ramus height and anteroposterior depth is achieved through resorption on the anterior surface of the ramus and greater deposition along the posterior surface of the ramus. At the same time, the corpus increases in length, providing the necessary space for development and eruption of the mandibular dentition. Associated with these changes in the absolute and relative sizes of the mandible are decreases in the gonial angle between the ramus and corpus.[23] The mandibular width increases by bony apposition along the buccal outer surface of the corpus and ramus and, to a lesser extent, resorption of bone occurs along the lingual, inner surfaces. Expansion of the mandible in the posterior direction via bone deposition along the posterior border of the ramus results in a longer and wider mandible.

At 7 years of age, bigonial width in males is 85% of adult width and in females 88% of adult width.[44] Growth in length and height of the mandible continues through the period of puberty. Height of the mandibular corpus depends in large part on growth of the alveolar bone. The mandible typically reaches adult size between 14 and 16 years in females and between 18 and 20 years in males.[16,17,19]

ORAL APPARATUS

The oral apparatus is composed of the dentition and the supporting structures within the maxilla and mandible. It is greatly influenced by the soft tissues such as the tongue and muscles of mastication.[23]

Teeth

The deciduous and permanent teeth are specialized organs of epithelial-mesenchymal origin. Normally, a complete set of primary teeth (20) have erupted by 2.5 to 3 years of age. The mixed dentition stage is heralded by eruption of the first permanent molars around 6 to 7 years of age. During the mixed dentition, both primary and permanent teeth are present. The mixed dentition stage is complete following the exfoliation of the last primary tooth. All permanent teeth except third molars (28 total in number) erupt by 12 to 13 years of age.

Alveolar Bone

Alveolar bone anchors the teeth and absorbs the stresses of mastication. Developing and maintaining alveolar bone depends on the presence of teeth. Prior to the eruption of the deciduous teeth, there is no appreciable alveolar bone. As the teeth erupt into functional occlusion, the alveolus proliferates in response to migration of the periodontal ligament. When a tooth is extracted, the alveolus at that site resorbs. If a tooth is surgically transposed or moved orthodontically into that site in the arch, alveolar bone will proliferate. In sum, the tooth is the functional matrix of the alveolus. Ankylosis arrests both dental eruption and alveolar bone formation in the affected area. Likewise, when a tooth is congenitally absent, the alveolar bone in that segment of the dental arch does not form (unless an adjacent tooth migrates into that space).

Appositional bone growth of the alveolar process occurs rapidly during the first 2 to 3 years to accommodate the deciduous teeth.[45] Dental arch width and perimeter change dramatically, especially during the transitions to the early mixed and permanent dentitions.[46] The teeth continue to migrate and erupt throughout childhood and adolescence, even after they have attained functional occlusion.[23] Teeth normally continue to erupt and form alveolar bone in synchrony with vertical growth. The posteruptive movements of teeth are directly related to the spaces created by growth displacements and movements of other teeth. The dentoalveolar compensation mechanism attempts to maintain a normal interarch occlusal relationship in the presence of variation in skeletal pattern.[47] Unlike teeth, dental implants are not capable of compensatory eruption or other physiologic movements.[48]

CLINICAL IMPLICATIONS

Anatomic and Physiologic Differences in the Pediatric Skeleton

Many age-related trends in pediatric facial trauma are explained by growth and development of the craniofacial skeleton. Soft-tissue injuries such as soft tissue avulsion, lacerations, and contusions[49] are more common than fractures in children, especially in younger children where the bones have a greater tendency to bend rather than break. Additionally, the facial nerve is in a more superficial position in the infant and young child, leading to greater chance of nerve damage in lateral facial soft-tissue injuries.[50]

A significant force of impact must be endured for the elastic pediatric craniofacial bones to fracture.[51] The pediatric facial skeleton has increased cancellous bone stock, larger buccal fat pads, decreased pneumatization of sinuses, buttressing unerupted teeth, and compliant sutures. These anatomic

features allow the facial skeleton to absorb energy without fracturing and when fracture does occur, it more likely results in a greenstick or nondisplaced fracture[49,51,52] (Table 2).

The changing anatomy and physiology of a child affects facial fractures considerably. The ratio of cranium-to-facial skeleton, development of the paranasal sinuses, and stage of dentition all influence the incidence and fracture patterns observed. In addition, compared to adults, children exhibit greater osteogenic potential, faster healing rate, and capacity for significant dental compensation.[53]

Fracture Patterns and Locations

There is a higher incidence of cranial injuries in young children (less than 5 years of age) due to the large cranium-to-face ratio. In young children, a prominent forehead "protects" the later maturing lower face from trauma. The forehead during this period, therefore, is more exposed and prone to injury. Because of the lack of pneumatization of the frontal sinus before age 7 years, orbital roof fractures are more likely to occur.[54] In childhood, orbital roof injuries are considered fractures of the skull base. As such, intracranial injuries are frequently coincident.[51,54,55] Meanwhile, fractures of the orbital floor are relatively rare in children younger than 5 years. After age 7 years, there is an increased incidence in orbital floor fractures that coincides with growth of the maxilla and maxillary sinus pneumatization.[56–58] Children with orbital floor fractures are prone to entrapment[59] due to the elasticity of the pediatric orbital bone and potential for greenstick fracture.

Whereas cranio-orbital injuries are seen more in the very young, midface and lower face injuries occur more frequently in the older and adolescent child. As the child grows, the forward and downward projection of the face increases the incidence of midface and mandibular fractures. Classical Le Fort midface fractures are rare in young children due to the presence of prominent buccal fat pads, immature sinus development, and buffering unerupted tooth buds.[52,60] Maxillary sinus expansion coincides with dental eruption during the mixed dentition, ages 6 to 12 years, and achieves full dimensions by puberty.[53] After age 6 years, maxillary fractures occur more frequently but the elasticity of the bone and mixed dentition may limit displacement. At age 12 years, which coincides with the permanent dentition and further expansion of the maxillary sinus, Le Fort midface type fractures become more common.[61] Similarly, zygomaticomaxillary complex fractures parallel the pneumatization of the maxillary sinus.

Midface fractures typically result from high energy impacts such as motor vehicle collisions and when present in young children, they are rarely isolated. There is a high incidence of associated neurocranial injuries because the force required to cause the maxillary fracture is sufficient to be transmitted to the cranial cavity.[62]

Likewise, nasal orbital ethmoid fractures (NOE) typically require high impact forces to the central nasal region. NOE fractures are rare compared to isolated nasal fractures,[63,64] which require less force to produce.[65] Nasal fractures are one of the most common pediatric facial fractures. As the nasal framework is more cartilaginous than bony, fractures of the cartilaginous septum are often found in children, whereas fractures of the bony nasal pyramid do not occur as frequently. Septal cartilage in children tends to buckle during trauma, making septal hematoma formation a more common finding than in adults.[36,66] Expansion of the hematoma separates the cartilage from the mucoperichondrium, obstructing blood flow to the nasal cartilage and causing pressure induced avascular necrosis of the nasal cartilage if left untreated.

The mandibular condyle is also a common site of fracture. Children younger than 5 years of age

Table 2
Summary overview of differences within the pediatric population

Infant/Child	Young Adolescent	Older Adolescent
Greenstick fracture	Fracture pattern variable	Fracture pattern may be adult-like
Small or absent sinuses	Developing sinuses	Mature sinuses
Developing tooth buds and challenging interdental fixation	Late mixed dentition/ permanent dentition	Permanent dentition
Bony healing occurs quickly		High energy trauma more frequent

are more likely to sustain intracapsular fractures and condylar neck fractures.[67] With increasing age, there is a shift toward subcondylar fractures and in adolescence angle and body fractures are more common.[36,67,68]

Growth Disturbance and Other Sequela

Trauma to growth centers (synchondroses, nasal septal cartilage), interruption of growth sites (sutures, periosteal absorption-resorption processes), damage to the mandibular condyle, and scar formation can disrupt normal facial growth and development. In addition, intraoperative soft tissue damage, periosteal stripping, and rigid fixation may affect the growing skeleton and developing dentition.[16,69] Interdental fixation is challenging in the mixed dentition.[53] Metal fixation systems carry real risk of injury to unerupted permanent tooth follicles and theoretical complications such as growth restriction (i.e., plate across suture lines). Bioresorbable plates and screws have been proposed as an alternative.[60] When internal bony fixation is necessary in younger patients, monocortical screws should be used and the hardware placed to avoid developing tooth buds.[52]

Generally, 3 main areas of growth may be affected: the orbital region, the nasomaxillary complex, and the condyle.[70] Dysmorphology may result from the initial trauma, the intervention, or a combination of both, during the growth years. Whether a growing child is initially managed nonoperatively or operatively, the patient and family should be made aware of the potential for aberrant growth resulting in asymmetries and occlusal discrepancies which may not become evident until adolescence.[53] Long-term follow-up is essential.

Orbital Fractures

Orbital roof fractures can be associated with dural disruption.[67] A very rare, delayed complication is a growing skull fracture that occurs secondary to a tear in the dura mater that coexists with the fracture, allowing intracranial content to herniate through it and widening of the skull defect. Following surgical correction, some children require a second operation to restore the axis of the orbit or correct orbital asymmetry.[67,71]

Fractures of the orbital region in very young children may also result in periorbital tissue atrophy and subsequent orbital volume loss. Because the development of the bony orbit is driven by the volumetric expansion of its intraorbital contents, namely the eyeball, in children with traumatic or disease-related loss of an eye, an implanted expander device may be used to sustain growth of the orbit until skeletal maturity.[72,73] The cranium and orbits are about 90% of adult size by age 5 to 7 years. To avoid or minimize growth impedance, when possible, reconstructions are performed when growth is nearly complete.

Nasomaxillary Complex Fractures

Nasoseptal and midface trauma in children can result in progressive deformity of the nose and midface with both functional and esthetic consequences.[36,74] It has been suggested that the younger the child at the time of nasal septum destruction, the greater the long-term effects on midfacial growth.[74] Thus, "in the pediatric population, aggressive open septorhinoplasties are typically avoided until skeletal maturity, and early closed reductions are recommended."[67] Nonetheless, long-term follow-up studies of pediatric nasal fractures treated with closed reduction have reported that over half the patients have deformity of the external nose including bony and cartilage deviations, saddling, dorsum widening, and hump.[65,75,76] Grymer and colleagues[75] pointed out that 70% of the patients with nasal deformities at the time of their study had reported satisfactory results following the initial treatment by closed reduction indicating that improvements diminished with future nasal growth. Secondary corrections may still be needed at skeletal maturity.[67]

Severe midface fractures, both treated and untreated, have been shown to produce very high incidence of subsequent deformities.[67,77–79] These include maxillary hypoplasia, facial asymmetry, nasal airway obstruction, and malocclusion.[77–82] Complications are most pronounced in patients who sustained multiple facial fractures likely because of both injury burden and extensive surgery required for reconstruction.

Mandibular Condylar Fractures

Condylar fractures can result in retrognathia, facial asymmetry, mandibular midline deviation to the affected side, malocclusion, tooth loss or injury, temporomandibular joint dysfunction and/or degeneration, limited mouth opening, and bony ankylosis.[4,67,83–88] An opposing maxillary occlusal cant may also be observed.[89] Several studies have reported less growth disturbances in young children less than 5 years of age due to high vascularity and regenerative capacity.[90–92] Demianczuk and colleagues[93] showed the greatest risk for significant growth disturbances was in the 4 to 7 year old and 8 to 11 year old groups, observing respectively that 24% and 16% of these children required corrective orthognathic surgery.

Patients with condylar fractures, whether unilateral or bilateral, may exhibit loss of posterior ramus height secondary to telescoping of the condylar fragment(s). Because children demonstrate remarkable bony remodeling, condylar fractures are frequently treated with closed reduction with a short period of maxillomandibular fixation as needed.[83,87,91] Zhu and colleagues[94] reported that in children aged 6 years and older, the condylar process remodeled incompletely. However, the remodeling of the glenoid fossa and increase in ramus height compensated for the hypotrophy of the condylar process on the fractured side. As children age, the ability to remodel and spontaneously heal is reduced. Mild malocclusions from minimally displaced fractures have the potential to resolve spontaneously with eruption of permanent dentition and through bony remodeling that occurs with growth and function.[67,69] Patients who sustain multiple fractures of the mandible are significantly more likely to have an adverse outcome that those with isolated mandibular fractures.[86] The mandible is one of the last bones to reach skeletal maturity and as such is vulnerable to growth and functional perturbations after injury to the condyles.

CONFLICTS OF INTEREST

The authors do not have any conflict of interests, financial or otherwise.

REFERENCES

1. Cobourne MT, DiBiase AT. Chap 3: Handbook of Orthodontics. Postnatal growth of the craniofacial region. 2nd edition. Edinburgh: Elsevier Health Sciences; 2016. p. 67–105.
2. Ranly DM. Craniofacial growth. Dent Clin North Am 2000;44(3):457–70.
3. Carlson DS. Evolving concepts of heredity and genetics in orthodontics. Am J Orthod Dentofacial Orthop 2015;148(6):922–38.
4. Rottgers SA, Decesare G, Chao M, et al. Outcomes in pediatric facial fractures: early follow-up in 177 children and classification scheme. J Craniofac Surg 2011;22(4):1260–5.
5. Moss ML, Skalak R, Dasgupta G, et al. Space, time, and space-time in craniofacial growth. Am J Orthod 1980;77(6):591–612.
6. Björk A, Skieller V. Growth of the maxilla in three dimensions as revealed radiographically by the implant method. Br J Orthod 1977;4(2):53–64.
7. Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. Am J Orthod 1972;62(4):339–83.
8. Bjork A. Facial growth in man, studied with the aid of metallic implants. Acta Odontol Scand 1955;13(1):9–34.
9. Fields HW. Craniofacial growth from infancy through adulthood. Background and clinical implications. Pediatr Clin North Am. Oct 1991;38(5):1053–88.
10. Farkas LG, Posnick JC, Hreczko TM. Anthropometric growth study of the head. Cleft Palate Craniofac J 1992;29(4):303–8.
11. Farkas LG, Posnick JC, Hreczko TM, et al. Growth patterns in the orbital region: a morphometric study. Cleft Palate Craniofac J 1992;29(4):315–8.
12. Farkas LG, Posnick JC, Hreczko TM. Growth patterns of the face: a morphometric study. Cleft Palate Craniofac J 1992;29(4):308–15.
13. Enlow D, Hans M. Essentials of facial growth. Philadelphia, PA: W. B. Saunders; 1996.
14. Costello BJ, Rivera RD, Shand J, et al. Growth and development considerations for craniomaxillofacial surgery. Oral Maxillofac Surg Clin North Am 2012;24(3):377–96.
15. Waitzman AA, Posnick JC, Armstrong DC, et al. Craniofacial skeletal measurements based on computed tomography: Part II. Normal values and growth trends. Cleft Palate Craniofac J 1992;29(2):118–28.
16. Wheeler J, Phillips J. Pediatric facial fractures and potential long-term growth disturbances. Craniomaxillofac Trauma Reconstr 2011;4(1):43–52.
17. Bhatia SN, Leighton BC. A manual of facial growth: a computer analysis of longitudinal cephalometric growth data. Oxford: Oxford University Press; 1993.
18. Berger AJ, Kahn D. Growth and development of the orbit. Oral Maxillofac Surg Clin North Am 2012;24(4):545–55.
19. Proffit W.R., Sarver D.M. and Jr H.W.F., Chap 2: Concepts of growth and development. Contemporary orthodontics, 4 edition, 2006, Elsevier, Philadelphia, PA.
20. Carlson DS. Theories of Craniofacial Growth in the Postgenomic Era. Semin Orthod 2005;11(4):172–83.
21. Baume LJ. Principles of cephalofacial development revealed by experimental biology. Am J Orthod 1961;47(12):881–901.
22. Meikle MC. The role of the condyle in the postnatal growth of the mandible. Am J Orthod 1973;64(1):50–62.
23. Carlson D. and Buschang P., Craniofacial Growth and Development: Developing a Perspective, In: LW G., RL V., KW V., et al., *Orthodontics current principles and technique*, 6 edition, 2017, Elsevier, chap 1. St. Louis
24. Buschang PH, Santos-Pinto A. Condylar growth and glenoid fossa displacement during childhood and adolescence. Am J Orthod Dentofacial Orthop 1998;113(4):437–42.
25. Agronin KJ, Kokich VG. Displacement of the glenoid fossa: a cephalometric evaluation of growth during

treatment. Am J Orthod Dentofacial Orthop 1987; 91(1):42–8.
26. Ohtsuki F, Mukherjee D, Lewis AB, et al. A factor analysis of cranial base and vault dimensions in children. Am J Phys Anthropol 1982;58(3):271–9.
27. Melsen B. Time and mode of closure of the sphenooccipital synchrondrosis determined on human autopsy material. Acta Anat 1972;83(1):112–8.
28. Pattisapu JV, Gegg CA, Olavarria G, et al. Craniosynostosis: diagnosis and surgical management. Atlas Oral Maxillofac Surg Clin North Am 2010;18(2): 77–91.
29. Morris C, Kushner GM, Tiwana PS. Facial skeletal trauma in the growing patient. Oral Maxillofac Surg Clin North Am 2012;24(3):351–64.
30. Ruengdit S, Troy Case D, Mahakkanukrauh P. Cranial suture closure as an age indicator: A review. Forensic Sci Int 2020;307:110111.
31. Escaravage GK Jr, Dutton JJ. Age-related changes in the pediatric human orbit on CT. Ophthalmic Plast Reconstr Surg 2013;29(3):150–6.
32. Smith EA, Halbach CS, Robertson AZ, et al. Orbital volume changes during growth and development in human children assessed using cone beam computed tomography. Head Face Med 2022; 18(1):8.
33. Kim JH, Jung DJ, Kim HS, et al. Analysis of the development of the nasal septum and measurement of the harvestable septal cartilage in koreans using three-dimensional facial bone computed tomography scanning. Arch Plast Surg 2014;41(2): 163–70.
34. Verwoerd CD, Verwoerd-Verhoef HL. Rhinosurgery in children: developmental and surgical aspects of the growing nose. Laryngo-Rhino-Otol 2010; 89(Suppl 1):S46–71. Rhinochirurgie bei Kindern: Entwicklungsphysiologische und chirurgische Aspekte der wachsenden Nase.
35. Kim SH, Han DG, Shim JS, et al. Clinical characteristics of adolescent nasal bone fractures. Arch Craniofac Surg 2022;23(1):29–33.
36. Wright RJ, Murakami CS, Ambro BT. Pediatric nasal injuries and management. Facial Plast Surg 2011; 27(5):483–90.
37. Nicol P, Elmaleh-Bergès M, Sadoine J, et al. Incidence and morphology of the incisive suture in CT scanning of young children and human fetuses. Surg Radiol Anat 2020;42(9):1057–62.
38. Behrents RG, Harris EF. The premaxillary-maxillary suture and orthodontic mechanotherapy. Am J Orthod Dentofacial Orthop 1991;99(1):1–6.
39. Trevizan M, Nelson Filho P, Franzolin SOB, et al. Premaxilla: up to which age it remains separated from the maxilla by a suture, how often it occurs in children and adults, and possible clinical and therapeutic implications: Study of 1,138 human skulls. Dental Press J Orthod 2018;23(6):16–29.
40. Persson M, Thilander B. Palatal suture closure in man from 15 to 35 years of age. Am J Orthod 1977;72(1):42–52.
41. Lorkiewicz-Muszynska D, Kociemba W, Rewekant A, et al. Development of the maxillary sinus from birth to age 18. Postnatal growth pattern. Int J Pediatr Otorhinolaryngol 2015;79(9):1393–400.
42. Voudouris JC, Woodside DG, Altuna G, et al. Condyle-fossa modifications and muscle interactions during Herbst treatment, Part 2. Results and conclusions. Am J Orthod Dentofacial Orthop 2003;124(1):13–29.
43. Björk A, Skieller V. Growth in width of the maxilla studied by the implant method. Scand J Plast Reconstr Surg 1974;8(1–2):26–33.
44. Lux CJ, Conradt C, Burden D, et al. Transverse development of the craniofacial skeleton and dentition between 7 and 15 years of age–a longitudinal postero-anterior cephalometric study. Eur J Orthod 2004;26(1):31–42.
45. Laowansiri U, Behrents RG, Araujo E, et al. Maxillary growth and maturation during infancy and early childhood. Angle Orthod. Jul 2013;83(4): 563–71.
46. Moorrees CF, Gron AM, Lebret LM, et al. Growth studies of the dentition: a review. Am J Orthod 1969;55(6):600–16.
47. Solow B. The dentoalveolar compensatory mechanism: background and clinical implications. Br J Orthod 1980;7(3):145–61.
48. Oesterle LJ, Cronin RJ Jr, Ranly DM. Maxillary implants and the growing patient. Int J Oral Maxillofac Implants 1993;8(4):377–87.
49. Ferreira P, Soares C, Amarante J. Facial Trauma. Pediatr Surg 2021;501–33. chap Chapter 133.
50. Kellman RM, Tatum SA. Pediatric craniomaxillofacial trauma. Facial Plast Surg Clin North Am 2014;22(4): 559–72.
51. Oppenheimer AJ, Monson LA, Buchman SR. Pediatric orbital fractures. Craniomaxillofac Trauma Reconstr 2013;6(1):9–20.
52. Andrew TW, Morbia R, Lorenz HP. Pediatric Facial Trauma. Clin Plast Surg 2019;46(2):239–47.
53. Maqusi S, Morris DE, Patel PK, et al. Complications of pediatric facial fractures. J Craniofac Surg 2012; 23(4):1023–7.
54. O-Lee TJ, Koltai PJ. Pediatric orbital roof fractures. Operat Tech Otolaryngol Head Neck Surg 2008; 19(2):98–107.
55. Gerbino G, Roccia F, Benech A, et al. Analysis of 158 frontal sinus fractures: current surgical management and complications. J Craniomaxillofac Surg 2000;28(3):133–9.
56. Broyles JM, Jones D, Bellamy J, et al. Pediatric Orbital Floor Fractures: Outcome Analysis of 72 Children with Orbital Floor Fractures. Plast Reconstr Surg 2015;136(4):822–8.

57. Guyot L, Lari N, Benso-Layoun C, et al. Orbital fractures in children. J Fr Ophtalmol 2011;34(4):265–74. Fractures de l'orbite de l'enfant.
58. Koltai PJ, Amjad I, Meyer D, et al. Orbital fractures in children. Arch Otolaryngol Head Neck Surg 1995; 121(12):1375–9.
59. Grant JH 3rd, Patrinely JR, Weiss AH, et al. Trapdoor fracture of the orbit in a pediatric population. Plast Reconstr Surg 2002;109(2):482–9 [discussion: 490-5].
60. Meier JD, Tollefson TT. Pediatric facial trauma. Curr Opin Otolaryngol Head Neck Surg 2008;16(6): 555–61.
61. Chandra SR, Zemplenyi KS. Issues in Pediatric Craniofacial Trauma. Facial Plast Surg Clin North Am 2017;25(4):581–91.
62. Koltai PJ, Rabkin D. Management of facial trauma in children. Pediatr Clin North Am 1996;43(6):1253–75.
63. Park SW, Choi J, Park HO, et al. Are gender differences in external noses caused by differences in nasal septal growth? J Cranio-Maxillo-Fac Surg 2014;42(7):1140–7.
64. Lopez J, Luck JD, Faateh M, et al. Pediatric Nasoorbitoethmoid Fractures: Cause, Classification, and Management. Plast Reconstr Surg 2019;143(1): 211–22.
65. Kopacheva-Barsova G, Arsova S. The Impact of the Nasal Trauma in Childhood on the Development of the Nose in Future. Open Access Maced J Med Sci 2016;4(3):413–9.
66. Johnson MD. Management of Pediatric Nasal Surgery (Rhinoplasty). Facial Plast Surg Clin North Am 2017;25(2):211–21.
67. Chao MT, Losee JE. Complications in pediatric facial fractures. Craniomaxillofac Trauma Reconstr 2009; 2(2):103–12.
68. Cleveland CN, Kelly A, DeGiovanni J, et al. Maxillofacial trauma in children: Association between age and mandibular fracture site. Am J Otolaryngo 2021;42(2):102874.
69. Lim RB, Hopper RA. Pediatric Facial Fractures. Semi Plast Surg 2021;35(4):284–91.
70. Miloro M, Ghali GE, Larsen PE, et al. *Peterson's principles of oral and maxillofacial surgery*. 4th edition. Switzerland: Springer International Publishing; 2022.
71. Amirjamshidi A, Abbassioun K, Sadeghi Tary A. Growing traumatic leptomeningeal cyst of the roof of the orbit presenting with unilateral exophthalmos. Surg Neurol. Aug 2000;54(2):178–81 [discussion: 181-2].
72. Tse DT, Abdulhafez M, Orozco MA, et al. Evaluation of an integrated orbital tissue expander in congenital anophthalmos: report of preliminary clinical experience. Am J Ophthalmol 2011;151(3): 470–82.e1.
73. Chojniak MM, Chojniak R, Testa ML, et al. Abnormal orbital growth in children submitted to enucleation for retinoblastoma treatment. J Pediatr Hematol Oncol 2012;34(3):e102–5.
74. Verwoerd CD, Verwoerd-Verhoef HL. Rhinosurgery in children: basic concepts. Facial Plast Surg 2007;23(4):219–30.
75. Grymer LF, Gutierrez C, Stoksted P. Nasal fractures in children: influence on the development of the nose. J Laryngol Otol 1985;99(8):735–9.
76. Dommerby H, Tos M. Nasal fractures in children–long-term results. ORL J Otorhinolaryngol Relat Spec 1985;47(5):272–7.
77. Davidson EH, Schuster L, Rottgers SA, et al. Severe Pediatric Midface Trauma: A Prospective Study of Growth and Development. J Craniofac Surg 2015; 26(5):1523–8.
78. Kao R, Campiti VJ, Rabbani CC, et al. Pediatric Midface Fractures: Outcomes and Complications of 218 Patients. Laryngoscope Investig Otolaryngol 2019; 4(6):597–601.
79. Precious DS, Delaire J, Hoffman CD. The effects of nasomaxillary injury on future facial growth. Oral Surg Oral Med Oral Pathol 1988;66(5): 525–30.
80. Ousterhout DK, Vargervik K. Maxillary hypoplasia secondary to midfacial trauma in childhood. Plast Reconstr Surg 1987;80(4):491–9.
81. Macmillan A, Lopez J, Luck JD, et al. How Do Le Fort-Type Fractures Present in a Pediatric Cohort? J Oral Maxillofac Surg 2018;76(5): 1044–54.
82. Aizenbud D, Morrill LR, Schendel SA. Midfacial trauma and facial growth: a longitudinal case study of monozygotic twins. Am J Orthod Dentofacial Orthop 2010;138(5):641–8.
83. Ghasemzadeh A, Mundinger GS, Swanson EW, et al. Treatment of Pediatric Condylar Fractures: A 20-Year Experience. Plast Reconstr Surg 2015; 136(6):1279–88.
84. Thorén H, Hallikainen D, Iizuka T, et al. Condylar process fractures in children: a follow-up study of fractures with total dislocation of the condyle from the glenoid fossa. J Oral Maxillofac Surg 2001;59(7): 768–73 [discussion: 773-4].
85. Lund K. Mandibular growth and remodelling processes after condylar fracture. A longitudinal roentgencephalometric study. Acta Odontol Scand Suppl 1974;32(64):3–117.
86. Smith DM, Bykowski MR, Cray JJ, et al. 215 mandible fractures in 120 children: demographics, treatment, outcomes, and early growth data. Plast Reconstr Surg 2013;131(6):1348–58.
87. Cooney M, O'Connell JE, Vesey JA, et al. Non-surgical management of paediatric and adolescent mandibular condyles: A retrospective review of 49 consecutive cases treated at a tertiary referral centre. J Craniomaxillofac Surg 2020;48(7): 666–71.

88. McGuirt WF, Salisbury PL 3rd. Mandibular fractures. Their effect on growth and dentition. Arch Otolaryngol Head Neck Surg 1987;113(3):257–61.
89. Vesnaver A. Dislocated pediatric condyle fractures - should conservative treatment always be the rule? J Cranio-Maxillo-Fac Surg 2020;48(10):933–41.
90. Chang S, Yang Y, Liu Y, et al. How Does the Remodeling Capacity of Children Affect the Morphologic Changes of Fractured Mandibular Condylar Processes After Conservative Treatment? J Oral Maxillofac Surg 2018;76(6):1279 e1–e1279 e7.
91. Du C, Xu B, Zhu Y, et al. Radiographic evaluation in three dimensions of condylar fractures with closed treatment in children and adolescents. J Craniomaxillofac Surg 2021;49(9):830–6.
92. Sahm G, Witt E. Long-term results after childhood condylar fractures. A computer-tomographic study. Eur J Orthod 1989;11(2):154–60.
93. Demianczuk AN, Verchere C, Phillips JH. The effect on facial growth of pediatric mandibular fractures. J Craniofac Surg 1999;10(4):323–8.
94. Zhu YF, Zou Y, Wang SZ, et al. Three-dimensional evaluation of condylar morphology after closed treatment of unilateral intracapsular condylar fracture in children and adolescents. J Cranio-Maxillo-Fac Surg 2020;48(3):286–92.