Chapter 143

c00143 # Oropharyngeal Growth and Skeletal Malformations

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Chapter Highlights

p0010
- Sleep-disordered breathing (SDB) is marked by varying degrees of collapsibility of the pharyngeal airway. The hard tissue boundaries of the airway dictate the size and therefore the responsiveness of the muscles that form this part of the upper airway. Thus, the airway is shaped not only by the performance of the pharyngeal muscles to stimulation but also by the surrounding skeletal framework.

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- The upper and lower jaws are key components of the craniofacial skeleton and the determinants of the anterior wall of the upper airway. The morphology of the jaws can be negatively altered by dysfunction of the upper airway during growth and development. In turn, the altered morphology of the jaws can be positively influenced by orthodontic treatment.

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- The association between altered dentofacial morphology and SDB has been well documented in children, adults, and patients with craniofacial syndromes. Whether this disease of childhood has the same origins as adult obstructive sleep apnea but more subtle manifestations has not been determined. The length and volume of the airway increase until the age of 20 years, at which time there is a variable period of stability, followed by a slow decrease in airway size after the fifth decade of life. The possibility of addressing the early forms of this disease with the notions of intervention and prevention can change the landscape of care.

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- Correction of specific skeletal anatomic deficiencies can improve or eliminate SDB symptoms in both children and adults. It is possible that the clinician may adapt or modify the growth expression, although the extent of this impact is uncertain. These strategies seek to alter an abnormal facial growth pattern wherein SDB worsens over time. Future research should focus on determining in which individuals dentofacial morphology makes a significant contribution to the pathogenesis of SDB. This may bring clinicians one step closer to targeting specific treatments that more effectively treat the disorder.

p0040 This chapter is directed to provide an integrated vision to both the scientist and clinician dealing with growth and development of the oropharyngeal structures. It is divided into three sections. The first reviews the fundamentals of growth of the craniofacial complex and the development of the upper airway. The second section outlines those characteristics based on current definitions of the sleep-disordered breathing (SDB) syndrome, that is, there is a broad range of anthropomorphic characteristics and OSA severity that needs to be better delineated. The last section reviews management and treatment strategies based on what is known about craniofacial development and anatomy. This chapter is a primer to other chapters in this section and to the ones in Section 14.

s0010 CRANIOFACIAL GROWTH AND DEVELOPMENT

p0045 Early theories of craniofacial development were based on the belief that growth of the face and jaws was essentially immutable because of intrinsic regulation by inherited genetic traits. Research centered on finding the location or sites where these traits were expressed that drove normal form and function of the other surrounding structures. During the early twentieth century, it was believed that differential deposition and resorption on the surface of bones were largely responsible for growth of the craniofacial skeleton. This remodeling theory then gave way to one heralding the role of the sutures, which held that similar to the epiphyses of the long bones, it was the connective tissue and cartilaginous joints of the craniofacial skeleton that produced expansive proliferative growth forcing the bones and soft tissues away from each other.[1] Exemplifying this theory was the concept that the mandible was much like a horseshoe-shaped long bone, with the condylar cartilage acting like open-ended epiphyseal plates, pushing the mandible down and away from the rest of the head.

p0050 Several inconsistencies in the hypothesis that sutures alone could be the determinants of craniofacial growth surfaced. Sutural growth was more similar to periosteal apposition of bone than previously understood, and sutures acted as reactive sites of bone growth rather than growth centers. Scott, in 1953, proposed the nasal septum theory of growth, which held as its main tenet that the anterior and inferior growth of the nasal septal cartilage was the determining, driving force of facial growth.[2] Whereas Scott's theory contributed to the understanding that growth at the sutures and surface

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remodeling were essentially reactive sites of bone growth, it was still founded in the paradigm that craniofacial development and morphogenesis were genetically predetermined and unalterable.[1] In modern perspective, the nasal septal cartilage is considered an important growth center; however, the mechanism behind this growth has been appropriately updated as described here.

p0055 A fundamental shift in the field of craniofacial biology emerged with Moss' introduction of the functional matrix hypothesis in 1960.[3] Whereas all previous theories deemed craniofacial growth to be predetermined by genetic traits, the functional paradigm introduced the idea of plasticity of development and growth of the craniofacial skeleton.[1] According to this theory, the role of our genes was to initiate the process by setting the initial context under which development could occur; beyond that, it was the extrinsic environmental and functional demands of the various craniofacial components that determined all future aspects of growth. This theory revolutionized the field by introducing two important concepts. First, it brought the possibility of growth modification as a treatment option for malocclusions or facial malformations by changing the direction of facial development to a more desired outcome. Second and perhaps more important, the plasticity of development opened a new area of research, focusing on the critical time and specific factors that lead to the maladaptive plasticity of the form and function of the craniofacial complex.[4]

p0060 With the emergence of developmental molecular biology, the genetic and external or epigenetic regulation of craniofacial growth is now better understood, and the modern synthesis is that both genomic and epigenetic factors are necessary contributors to craniofacial development.[5,6] Several genes and gene products regulate the morphogenesis and intrauterine development of the craniofacial complex. There is an extremely complex interaction between these genetic factors and epigenetic influences. The plasticity of early development depends not only on the effects of certain environmental conditions on the underlying genetic code but also on previous environmental conditions that may have directly upregulated or downregulated specific genetic regulatory factors and indirectly further influenced the response.

s0015 Prenatal Craniofacial Growth

p0065 The earliest form of the face appears in the fourth week of life with the enlargement and movement of the frontonasal prominence as well as the paired maxillary and mandibular prominences stemming from the first branchial arch. These five prominences emerge to encircle the stomodeum, or primitive mouth. A critical aspect of this process is the migration of cranial neural crest cells into the developing facial prominences. Unlike in the rest of the body, these neural crest cells develop into the majority of the craniofacial hard tissues, including the bone, cartilage, and teeth of the craniofacial complex.[7] The specific end tissue into which these cranial neural crest cells develop is determined largely by the family of Homeobox or HOX genes. The variable expression of HOX transcription factors causes the groups of cranial neural crest cells in the distinct prominences to respond differently to the same growth factors.[8]

p0070 After rapid growth of the two mandibular prominences, there is midline fusion in the fifth week of development.[9] Initial mandibular development is dependent on tightly controlled molecular signaling between the oral ectoderm and the underlying core of cranial neural crest.[7] Further development is contingent on the formation and growth of Meckel cartilage, the first skeletal element of the mandible. The subsequent elongation of this rod of cartilage leads to promotion of outgrowth of the mandible, and the primary ossification of the mandible occurs as intramembranous bone formation along this cartilaginous core.[10] As the bony mandible further develops, Meckel cartilage largely disappears, eventually to persist only as small portions of the incus and malleus of the middle ear. In addition to the mandible, the mandibular processes also form the lower lip and the lower areas of the cheeks.

p0075 The lateral and medial nasal processes originate as ectodermal thickenings on the surface of the frontonasal process early in the fifth week of embryonic life. Subsequent broadening of the head and medial growth of the maxillary processes result in medial displacement of the early nasal processes. As the two medial nasal processes merge at the midline, the philtrum and columella of the nose are formed. Deeper aspects of the medial nasal processes will differentiate to form the nasal septum, which is a key growth center of the midface postnatally. Fusion of the medial nasal process with the maxillary process leads to formation of the majority of the upper lip, the zygomas, and the maxilla bilaterally. The lateral nasal processes go on to form the sides and alae of the nose as the subsequent 2 weeks sees the formation of the future nostrils and nasal cavity with the development of the primary and secondary palates.[11] Secondary palate formation relies on the coordinated growth and movement of the primordia of the tongue and both lower and upper jaws. During the sixth week of development, paired lateral palatal shelves arise as medial projections from each maxillary process. Critically, at this time, there is rapid anterior growth of the early mandible by proliferation of Meckel cartilage, which displaces the tongue forward, lowering it relative to the palatal shelves.[12] Once the tongue has descended, during the seventh week, the palatal shelves rotate from a vertical to a horizontal position directed toward the midline. Further growth of the shelves sees them fuse at the midline as well as with the primary palate anteriorly and the nasal septum superiorly.[13]

p0080 By the ninth week of fetal development, the initial cartilaginous facial skeleton is well established, composed of the chondrocranium forming the skull base, the nasal capsule in the upper face, and the Meckel cartilage in the lower face. Within the twelfth week of fetal growth, areas of ossification begin to appear and bone begins to rapidly replace this cartilaginous template to form the early cranial base. At this same time, the bones of the cranial vault as well as the mandible and maxilla develop through intramembranous ossification.

Postnatal Craniofacial Growth s0020

p0085 The general pattern of postnatal development is the cephalocaudal gradient of growth, in which there is an axis of increased growth that extends away from the head. Structures away from the brain tend to grow more and later than those structures closer to the brain. Mandibular growth begins later and continues longer than does the growth of the midface.[14] This pattern of growth continues until maturity and is exemplified in the proportionality of head size to total body length. At birth, the head makes up almost a quarter of the total body length, which decreases to around

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12% in the adult. Facial growth can be summarized as being driven forward initially by growth of the cranial base, then the maxilla and mandible both grow back and up to fill in the space created as they are being pulled down and forward away from the cranial base by the soft tissues in which they are embedded.

p0090 During infancy and early childhood, the cranial base increases in length through endochondral ossification that occurs at important growth sites called synchondroses. The synchondroses push the growth of the face forward until around the age of 7 years, when they begin to become less active and later ossify and fuse. Much of the forward movement of the maxilla is due to the growth of the cranial base pushing it downward and forward from behind. Further forward displacement of the maxilla results from bone apposition at the sutures located posteriorly and superiorly that connect it to the cranial base. Unlike the forward displacement generated by the synchondroses, the bone formation at these sutures is instead responsive in nature to the downward and forward pull of the maxilla from the growth of the associated soft tissues and nasal septum. Much of the increase in size of both the nasal and oral cavities occurs from surface remodeling of the maxilla and not from sutural growth. As the maxilla is translated downward and forward, the periosteum acts to remove bone at the floor of the nose, while at the same time bone is formed on the roof of the mouth. Over time, this results in a hollowing out and widening of the nasal cavity.[15] In addition, the palatal vault deepens with age despite the bone apposition on its surface because of the increased growth of the alveolar process that accompanies tooth eruption. Transverse growth in maxillary width results from growth at the midpalatal suture and appositional remodeling along the lateral aspects of the posterior region of the maxilla and the maxillary tuberosity.[16] This bone deposition at the tuberosity allows sagittal lengthening of the maxilla. The midpalatal suture begins to fuse in early adolescence but stays amenable to orthopedic force required for maxillary expansion treatment until around the age of 14 years in most individuals.

p0095 Lacking open sutures necessary for suture apposition of bone, the mandible instead grows by endochondral ossification at the condyle as well as by a combination of extensive surface bone remodeling. Unlike an epiphyseal plate or synchondroses, the growth of the condyle is responsive to translation of the mandible rather than driving it.[17] Transverse increases in the body of the mandible occur through surface apposition and remodeling of bone. The dentoalveolar structures develop with the eruption of the teeth, which continue to erupt throughout life to maintain occlusal contact, matching the vertical growth of the ramus.

p0100 First described nearly a century ago,[18] the hard and soft tissues undergo different rates of growth throughout childhood development. This is evident in the upper airway of children and has important implications for obstructive sleep disorders. Because of the significance of suckling to the newborn infant, the epiglottis is located close to the soft palate, which facilitates separation of the pathways for respiration and deglutition.[19] Neonates are born as obligate or preferential nasal breathers, but this changes as the upper airway matures. Between the ages of 1 and 2 years, vertical growth allows the larynx to descend to the level of the fifth cervical vertebra, and the epiglottis descends, which accommodates the newly acquired function of speech for the child.[20] The hyoid bone descends to a lower position in the neck, and the posterior third of the tongue descends to form the anterior wall of the oropharynx. Due mainly to hypertrophy of the adenoids and tonsils that frequently can exceed the growth of surrounding skeletal structures, adenoid and tonsillar tissue is found to be largest relative to the surrounding anatomy between the ages of 4 and 6 years.[21-23] This not coincidentally is the same age range at which OSA is most frequently seen in children. The upper airway volume then increases in adolescence because of both the concurrent increase in vertical skeletal growth and involution of the lymphoid tissue, which decreases in size after 12 years of age.[22,24] During the adolescent years, the upper airway also becomes larger in the transverse dimension and more elliptical.[24] The length and volume of the airway increase until the age of 20 years, at which time there is a variable period of stability, followed by a slow decrease in airway size after the fifth decade of life.[25]

DENTOFACIAL MORPHOLOGY ASSOCIATED WITH SLEEP DISORDERED BREATHING s0025

p0105 Dentofacial morphology in children and in adults has been assessed by lateral and anterior-posterior cephalography, dental casts of the upper and lower teeth, digital photography, and three-dimensional magnetic resonance imaging (Table 143-1).[26] Cephalography is limited by landmark identification, measurement variability, and two-dimensional assessment of a three-dimensional anatomy. In children, only a few studies assess three-dimensional dentofacial measures.[27,28] However, all these imaging methods are performed while the patient is either awake or sedated, which does not reflect upper airway volume and soft tissue sleep-related changes. Whereas dentofacial morphology is an important component to the multidisciplinary assessment and management of SDB, there is no single cephalometric measurement that can effectively predict OSA severity,[29] as outlined in Table 143-1.

Children s0030

p0145 In children, although adenotonsillar hypertrophy and obesity often contribute to SDB, dentofacial morphology can further contribute to the narrowing of the upper airway. Behavioral or functional oral breathing in SDB is associated with altered craniofacial growth,[30] altered muscle recruitment in the nasal and oral cavities,[31] and change in posture.[32] These ideas will be further explored in this chapter, in the section on treatment strategies. For children between 6 and 8 years, dentofacial morphology is a stronger risk factor for SDB than obesity is.[33] Cephalometric studies suggest that a long and narrow face, transverse deficiency, and retrognathia are craniofacial morphologic factors associated with a narrow upper airway and SDB in children,[34-37] which are also particular to oral breathing.[38]

p0150 Studies measuring differences in position between the maxilla and mandible (ANB: A point, nasion, B point) show an increased ANB angle in children with OSA or with primary snoring compared with controls.[36,39-43] In the primary snorers, this was associated with a decreased SNB angle (sella, nasion, B point),[36,42,43] which is a measure of retrognathia. In children with OSA, a decreased SNB angle and lower hyoid bone position and mandibular volume[28] were observed with three-dimensional imaging.

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t0010 **Table 143-1 Craniofacial Morphology Associated with Obstructive Sleep Apnea**

	Adults	Children
Long and narrow face		Marino 2009,[34] Pirila-Parkkinen 2009,[35] Pirila-Parkkinen 2010,[36] Tsuda 2011[37]
Transverse deficiency	Johal 2004,[78] Poirrier 2012[80]	Marino 2009,[34] Pirila-Parkkinen 2009,[35] Pirila-Parkkinen 2010,[36] Tsuda 2011,[37] Cozza 2004,[40] Lofstrand-Tidestrom 1999,[44] Katyal 2013[43]
Retrognathia	Paoli 2001,[59] Guilleminault 1984,[60] Lowe 1995,[61] Lowe 1986,[62] Lyberg 1989,[64] Miles 1996,[65] Johal 2007,[69] Ishiguro 2009,[70] Chi 2011,[26] Iked 2001,[66] Sforza 2000,[67] Tangugsorn 2000,[68] Banhiran 2013,[71] Gungor 2013,[72] Okubo 2006,[75] Riha 2005[76]	Marino 2009,[34] Pirila-Parkkinen 2009,[35] Pirila-Parkkinen 2010,[36] Tsuda 2011,[37] Cappabianca 2013[28]
Increased ANB angle		Pirila-Parkkinen 2010,[36] Deng 2012,[39] Cozza 2004,[40] Zucconi 1999,[41] Katyal 2013[43]
Decreased SNB angle		Cappabianca 2013[28]
Lower hyoid bone position	Paoli 2001,[59] Guilleminault 1984,[60] Lowe 1995,[61] Lowe 1986,[62] Lyberg 1989,[64] Miles 1996,[65] Johal 2007,[69] Ishiguro 2009,[70] Chi 2011,[26] Iked 2001,[66] Sforza 2000,[67] Tangugsorn 2000,[68] Banhiran 2013,[71] Gungor 2013,[72] Riha 2005,[76] Lowe 1997[74]	Cappabianca 2013[28]
Increased mandibular plane angle		Linder-Aronson 1970,[30] Pirila-Parkkinen 2009,[35] Pirila-Parkkinen 2010,[36] Deng 2012,[39] Cozza 2004,[40] Zucconi 1999,[41] Lofstrand-Tidestrom 1999,[44] Zettergren-Wijk 2006,[45] Juliano 2009,[46] Ozdemir 2004,[47] Guilleminault 1989[48]
Increased lower anterior facial height		Linder-Aronson 1970,[30] Pirila-Parkkinen 2009,[35] Pirila-Parkkinen 2010,[36] Deng 2012,[39] Cozza 2004,[40] Zucconi 1999,[41] Lofstrand-Tidestrom 1999,[44] Zettergren-Wijk 2006,[45] Juliano 2009,[46] Ozdemir 2004,[47] Guilleminault 1989[48]
Retrusive maxilla	Paoli 2001,[59] Guilleminault 1984,[60] Lowe 1995,[61] Lowe 1986,[62] Lyberg 1989,[64] Miles 1996,[65] Johal 2007,[69] Ishiguro 2009,[70] Chi 2011,[26] Iked 2001,[66] Sforza 2000,[67] Tangugsorn 2000,[68] Banhiran 2013,[71] Gungor 2013[72]	
Palatal morphology, increased length and thickness of soft palate	Lyberg 1989,[64] Johal 2007,[69] Kurt 2011[77]	

p0155 An increased mandibular plane angle and increased lower anterior facial height are associated with OSA.* However, a meta-analysis showed significant heterogeneity across selected studies,† suggesting insufficient evidence of a strong association. Retrognathia creates a posterior displacement of the tongue base, which further narrows the upper airway and is associated with a high-arched (ogival) palate due to tongue position.[41,49] Although a narrow maxilla is associated with OSA and snoring, only a few studies have reported this from dental impressions of hard and soft tissues[35,40,43,44] as this cannot be measured from lateral cephalograms. Moreover, orthodontic correction of a narrow maxilla (rapid maxillary expansion) has been reported to reduce respiratory indexes.[50-52] These earlier studies suggesting an association between morphology and sleep apnea do not fully explain causality of dentofacial morphology in the pathophysiologic process of SDB.

p0160 In children with OSA, it was suggested that 50% also have sleep bruxism, a concomitant sleep movement disorder[53] that can affect their dentofacial health, although no causal relationship has been established. Parents report tooth grinding twice more often in children who are habitual snorers than in nonsnorers[54] and more often in younger children than in older ones.[54] Up to 60% of children with sleep bruxism but without sleep apnea have a retrusive mandible; 28% have short faces (brachyfacial type).[55] Adenotonsillectomy reduced sleep bruxism muscle activity in 75% of OSA children as reported by questionnaires.[56] A temporary maxillary occlusal splint of 3 mm in thickness was worn 3 months by children (aged 6 to 8 years) with a history of sleep bruxism, oral breathing, and snoring.[57] As reported by questionnaires, tooth-grinding noises were decreased in 89% of patients, whereas snoring was reduced in 55.5% of patients.[57] This could be explained by potentially restoring nasal breathing during sleep as all

*References 27, 30, 35, 36, 39-41, 44-48.
†References 27, 35, 36, 39-41, 43-45.

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participants adapted from oral breathing to nasal breathing after treatment.[57] The associations between SDB and sleep bruxism in children need further investigation and objective data. A full description of sleep bruxism is found in Chapters 147 and 150.

s0035 **Adults**

p0165 In adults, obesity is the main anatomic risk factor for SDB. Like children, dentofacial morphology can also contribute to a compromised upper airway, and this is more often observed in nonobese patients with OSA.[58,59]

p0170 Overall, studies have reported a retrusive mandible, macroglossia, lowered hyoid bone position, and retrusive maxilla to be associated with OSA[26,59-72] and snoring.[73] A lower hyoid bone position is suggested to be a proxy of tongue shape, posture, and tone, which could increase upper airway collapsibility.[26,74] Magnetic resonance imaging studies in Asian and white populations observed a shorter and smaller mandible in patients with sleep apnea than in controls.[26,69,75] This was significant in men with sleep apnea[26,75] and not reported in women.[26] Mandibular morphology seems to be a stronger risk factor than maxillary morphology for OSA in adults.[26] Taking into account the genetic influence on dentofacial morphology, comparison of a cohort of siblings with and without sleep apnea found a short mandible and a lower hyoid bone position to be the most important risk factors for sleep apnea.[76]

p0175 Palatal morphology and increased length and thickness of the soft palate are also risk factors for OSA[63,64,69,77] and for snoring.[77] In addition, patients with sleep apnea are reported to have longer soft palates than snorers.[73] In comparing lateral cephalograms and dental casts of OSA adults with those of controls, an increased palatal depth was seen as measured at the first and second premolars and molars,[78] although this was not a consistent finding.[79] By anteroposterior cephalometric measurements, patients with OSA had narrower maxillas.[80]

s0040 **Craniofacial Syndromes**

p0180 Multiple craniofacial syndromes can have a higher incidence of SDB because of modified craniofacial morphology or hypertrophy of soft tissues. Outlined in Table 143-2, among the mandibular hypoplasia syndromes are Pierre Robin, Prader-Willi, Treacher Collins, and Marfan. Moreover, some syndromes can be associated with neuromuscular disorders, which can further negatively affect breathing during sleep. In children with craniofacial syndromes and SDB, the causes of obstruction or restriction of the upper airway can be at multiple levels, requiring multidisciplinary management and multiple treatments.[81-85]

p0185 SDB is often associated in children with midface hypoplasia; 50% of nonsyndromic and syndromic craniosynostosis children,[86] such as children with Apert, Crouzon, and Pfeiffer syndromes, develop SDB.[82,87,88] SDB improves during the first 3 years of life in the absence of midface hypoplasia.[89] However, this is not observed in children with syndromic craniosynostosis and midface hypoplasia (Apert or Crouzon/Pfeiffer).[89] Midface advancement surgery was successful in the short term with improved respiratory outcomes in 55% but was ineffective in 45% of children.[82] In this latter group, endoscopy and volume measurements showed obstruction of the hypopharynx.[82] After adenotonsillectomy, 60% of children with syndromic craniosynostosis had less desaturation events (Sao_2 4%).[81] Achondroplasia, an autosomal dominant congenital disorder, is also characterized by midface hypoplasia, leading to increased risks for development of SDB. Approximately 35% of patients with achondroplasia have SDB,[90] associated with increased lower facial height and retrognathia.[91]

Table 143-2 Syndromic and Craniofacial Morphology Associated with Obstructive Sleep Apnea t0015

- **Maxillary hypoplasia or midface hypoplasia** u0030
  such as syndromic craniosynostosis (Apert, Crouzon, Pfeiffer), achondroplasia, trisomy 21, and cleft palate u9000
- **Mandibular hypoplasia** u0035
  such as Pierre Robin, Prader-Willi, Treacher Collins, and Marfan u9005
- **Mandibular hypoplasia or micrognathia** u0040
  such as Pierre-Robin, Smith-Lemli-Opitz syndrome, and trisomy 21 u9010
- **Orofacial hypotonia** u0045
  such as Smith-Lemli-Opitz syndrome and trisomy 21 u9015
- **Cleft lip and palate** u0050
  such as Pierre Robin u9020
- **Maxillary and mandibular hypoplasia** u0055
  such as Turner syndrome u9025
- **Neuromuscular disorders** u0060
  such as Duchenne muscular dystrophy, myopathies, Guillain-Barré syndrome, and myasthenia gravis u9030

p0190 In addition to midface hypoplasia, patients with trisomy 21 also have micrognathia and orofacial hypotonia, which predisposes them to SDB[92] in 50% of pediatric and adult cases.[83,93-95] Although suggested as the first-line treatment, adenotonsillectomy resolves SDB in only 27% to 34% of cases.[96,97] Other alternative treatments are positive airway pressure therapy, mandibular distraction osteogenesis, midface advancement, and oral appliances. Oral appliances with tongue-stimulating knobs have been shown to improve orofacial muscle function.[83,84,98-103]

p0195 Cleft lip and palate children showing midface hypoplasia have a significantly higher incidence of SDB (22% to 37.5%)[85,104] than do healthy children (5%).[105] Furthermore, 34% of syndromic children with cleft lip and palate reported symptoms, whereas 17% of nonsyndromic children with cleft lip and palate did.[85] Following various surgical interventions (e.g., adenotonsillectomy, flap takedown, tonsillectomy, and partial adenoidectomy), only 38.5% of patients had improved SDB.[85]

p0200 Syndromes characterized by micrognathia and orofacial hypotonia, such as Smith-Lemli-Opitz syndrome, or by smaller maxilla and mandible, such as Turner syndrome, can also have increased incidence of SDB.[106,107]

p0205 Pierre Robin sequence is characterized by a triad of craniofacial anomalies consisting of micrognathia, cleft palate, and glossoptosis leading to respiratory and feeding issues. Most infants with Pierre Robin sequence (85%) also have sleep breathing disorders.[108] Nonsurgical management of Pierre Robin sequence in infants includes positional therapy, placement of nasopharyngeal airway, and oral appliances with velar extension. Positional therapy is successful in 49% to 52% of cases.[109,110] Oral appliances with velar extension were reported in one study to effectively reduce OSA at hospital

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discharge and 3 months later, in the absence of any adverse events.[111] Surgical management includes tongue-lip adhesion or glossopexy, subperiosteal release of the floor of the mouth, mandibular distraction osteogenesis, and tracheotomy. Mandibular distraction osteogenesis is reported to have better outcome measures than tongue-lip adhesion in regard to oxygen saturation, apnea-hypopnea index (AHI), and number of postprocedure tracheostomies.[112] However, both surgeries had comparable complications.[112]

s0045

TREATMENT STRATEGIES

p0210 Management and treatment acknowledge that SDB has a cumulative positive feedback loop in which repetitive respiratory-related arousals cause changes to the properties of the upper airway, and this causes end-stage sequelae that exacerbate the initial stimulus.[113] This section focuses on the treatment strategies for the pediatric patient. For more information on adult SDB treatment, refer to Section 14 of this volume.

p0215 The indication for treatment underlies what is pathologic and what is normal. What constitutes disease that necessitates treatment? Recent evidence shows that simple snoring carries consequences of cognitive impairments, suggesting that benign primary snoring should be treated as a disease. The incidence of snoring is much higher than the incidence of OSA,[114] and snoring is now recognized as an abnormality in children.[115] If snoring is the start of the cascade of abnormal breathing, early treatment may be correlated to improved outcomes. Even with early intervention, the recurrence of SDB has been documented in studies of pediatric patients observed through adolescence.[116,117] So, whereas the timing of treatment is relevant, early detection and treatment of SDB children are only part of the solution because there can be a familial inheritance of both symptoms and anatomic risk factors.[118-120] Consequently, asymptomatic children with at-risk morphologic characteristics and a familial history of SDB should be monitored for further evaluation.

p0220 There is not a linear relationship of symptoms to severity of the disease, and similarly, if there is a disproportion in anatomic structures, it may not necessarily correlate with the symptoms or the severity of the disease.[121] A study that observed SDB children 4 years after treatment showed that despite the improvement in respiratory parameters, complaints of daytime sleepiness persisted in the treated and untreated SDB group compared with asymptomatic, non-SDB controls. For the health care practitioner treating children, there are screening measures to decide if a child needs further care. Again, see Section 14 of this volume for more information on SDB and OSA diagnosis and management. These factors can be distilled down to the presence of daytime or nighttime symptoms, the orofacial anatomy, and the familial history of SDB (Box 143-1). The treatments for pediatric SDB are multiple and listed in Box 143-2.[122,123] The intent of early treatment is to halt the continued cycle of worsening OSA and also to prevent early systemic complications. The consequences of neurocognitive deficits and cardiovascular changes are evident in children,[124,125] similar to the systemic changes seen in adults. It is possible that OSA in an adult could have begun in childhood or adolescence.[126] However, there are no long-term outcome studies that demonstrate the progression of these changes from childhood into adulthood, nor is there any evidence that these effects seen in children are severity dependent or evolve into the adult presentation with end-organ morbidity.

Box 143-1 SCREENING MEASURES FOR PEDIATRIC TREATMENT b0010

Presence of daytime and nighttime symptoms	p0225
Orofacial anatomy	u0070
Familial history	u0075

Box 143-2 TREATMENT OPTIONS FOR PEDIATRIC OBSTRUCTIVE SLEEP APNEA SYNDROME b0015

Soft tissue reduction or removal (tonsils, adenoids, turbinates)	p0245
Allergy management	u0085
Jaw expansion	u0090
Myofunctional therapy	u0095
Nasal continuous positive airway pressure	u0100
Skeletal surgery	u0105

Box 143-3 CURATIVE STRATEGIES FOR PEDIATRIC OBSTRUCTIVE SLEEP APNEA SYNDROME b0020

Increase in airway size (nasopharynx, oropharynx, hypopharynx)	p0285
Improvement in muscle response	u0115
Craniofacial growth modification	u0120

p0280 There are three general treatment strategies for SDB. The problem of collapsibility affecting airflow exchange in SDB can be primarily related to inadequate airway size creating airflow resistance, and the first line of treatment is directed at enlarging the airway. Magnetic resonance imaging studies of OSA children show a smaller upper airway cross-sectional area.[127] However, children with normal oropharyngeal anatomy may suffer from OSA,[128] and the AHI has not been shown to directly correlate with airway volume.[121] First-line treatment approaches to increasing space in the nasopharynx, oropharynx, and hypopharynx are reviewed. SDB may also encompass a component of muscle alteration that may be a primary (etiologic) or secondary effect.[113] The second component of therapy addresses the muscle remodeling and myopathy that may be associated with SDB. The third strategy incorporates the challenge to complete care, and that raises the question of whether a cure or complete resolution of the disease is possible by changing the underlying facial growth pattern and modified anatomy to eliminate this as an etiologic factor in the disease. This paradigm for care is summarized in Box 143-3.

Strategy 1: Increase in Airway Size s0050

Location–Nasopharynx s0055

p0305 Although the area of greatest collapsibility is the soft tissue of the oropharynx, the properties along the entire upper airway will affect this collapsibility. Each part of the pharynx (i.e., nasopharynx, oropharynx, hypopharynx) serves different

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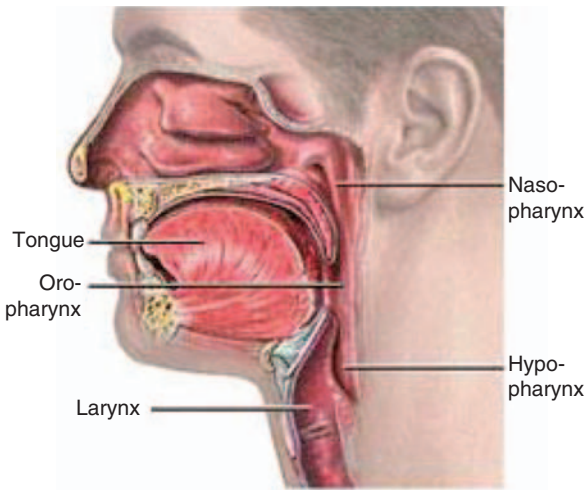

f0010 **Figure 143-1** The pharynx, where airway collapsibility occurs, is divided into three sections: nasopharynx, oropharynx, and hypopharynx.

b0025 **Box 143-4 INCREASING THE NASOPHARYNX SIZE**

p0315 Reduction of inflammation through medication
u0130 Orthodontic expansion
u0135 Surgical removal of soft tissue

functional roles, and so treatments to increase airway size are reviewed according to site in the pharynx (Figure 143-1).

p0310 The entrance to the airway at the initial site of airflow is the nasopharynx. The treatments to increase the size of the nasopharynx (Box 143-4) address either removal of obstructions inside the nose or enlargement of the space itself. The nasal influences on snoring and SDB are widely known because nasal obstruction can cause sleep disturbances that affect daytime performance.[129] The degree of nasal obstruction does not correlate with the severity of OSA,[130] probably because the extent of nasal resistance does not correlate with the amount of nasal airflow.[131] This is evident in patients with choanal atresia who have nasal obstruction as a clinical feature. In this population, it was reported that 65% are diagnosed with OSA[132] versus the entire patient group. Systematic review of the relationship between nasal obstruction and OSA shows that nasal obstruction plays a modulating role in OSA but is not a direct causative factor.[133] Whereas the relationship between nasal obstruction and SDB is not linear, it is thought either to be linked to an increase in nasal resistance initiating unstable oral breathing or to result from impaired nasal reflexes that hinder continued ventilation.[134] Increased nasal resistance depresses the critical closing pressure of the pharyngeal muscle walls, rendering the airway more collapsible. The critical closing pressure is correlated with the severity of SDB.[121] Pharyngeal compliance is impaired in a cycle in which SDB can both result from and be worsened by nasal obstruction.[135]

s0060 **Reduction of Inflammation and Medication Management.**
p0335 SDB is more prevalent in patients with allergic diseases.[136] Allergic rhinitis hinders nasal respiration by increasing nasal resistance and is considered a risk factor for SDB.[137] Allergic rhinitis is one of the major causes of impaired nasal function, and it affects up to 40% of the general population in developed countries, with an increasing prevalence. Although the evidence generally supports a connection between SDB and allergic rhinitis, this connection is not definitive, and the mechanism linking these two diseases and the mechanism of how nasal inflammation causes SDB are unclear.[138] However, nasal resistance measured by anterior rhinometry is increased in children with OSA compared with control subjects.[139] Larger population studies[140] and several smaller studies demonstrated an improvement in SDB by treatment of allergic rhinitis through medication management.[141] Nasal corticosteroids in children with moderate sleep OSA was effective in improving symptoms but not in eliminating the disorder,[140] thus suggesting the role of inflammation in the disease.[142] The cause and effect link is not yet fully understood or explained between allergic rhinitis and SDB. This topic is explored in more detail in Chapter 119.

s0065 **Orthodontic Expansion.**
p0345 As early as the 1860s, the midpalatal suture was separated within 2 weeks,[145] using an orthodontic screw-type expander device to widen the transverse dimension of the upper jaw either to correct a transverse deficiency or to create space for the permanent teeth. Early studies of maxillary expansion show that both dentoalveolar and craniofacial structural changes were created.[146,147] The amount, location, and rate of force application to the facial skeleton from expansion appliances create localized changes in the bony housing surrounding the teeth, with a potential effect at the sutural level of the maxilla.[148,149] Rapid maxillary expansion (RME) which refers to a rate of expansion of at least 0.25 mm/day, was described in the medical literature dating back to 1975[150] as a therapy for medical ailments and referenced in the dental literature for medical problems since 1974.[151] This early work described maxillary expansion to treat problems such as enuresis, nasal congestion, and asthma. These symptoms also describe the OSA syndrome, although the syndrome was not coined until 1976.[152]

p0350 In 1980, surgical widening of the maxilla to increase the lateral dimension of the nasopharynx was first described as a treatment for adult OSA. Nonsurgical rapid palatal expansion was first suggested as a therapy for OSA in 1998.[143] Pirelli et al[144] published seminal work in 2004 using rapid maxillary expansion (RME) to successfully treat OSA syndrome in children with narrow maxillas. Several other groups have corroborated this work, and now other published studies also describe the effectiveness of maxillary expansion in treating children with or without narrow palates, with or without retrognathic mandibles. These studies are outlined in Table 143-3, and the general appliance design is depicted in Figure 143-2. In the maxilla, the applied force from the expansion appliance creates midpalatal (also called median palatal or palatal) suture separation. This results in distraction osteogenesis across the palate, resulting in an increase in width of the maxilla (Figure 143-3).[147] Both oral and nasal volume is increased as the triangular nasal fossa is enlarged by widening of the nasal floor and outward movement of the lateral nasal walls.[153] The volumetric increase evident in the nasal fossa but not posteriorly in the nasopharynx[154] has an effect on nasal airflow, but this is not a proportional relation.

p9100 Several studies show that maxillary expansion reduces nasal resistance.[153,155-157] In a systematic review of all expansion studies across several different databases, there were

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t0020

Table 143-3 Expansion Outcome Studies for Obstructive Sleep Apnea

Author	Year	Mean Age (years)	N	Responders
Pirelli et al[144]	2004	8.7	31	31
Villa et al[51]	2007	6.6	14	12
Miano et al[249]	2009	4–8	9	9
Villa et al[52]	2011	6.6	10	8 (3-year follow-up)
Guilleminault et al[167]	2011	6.5	31	31
Marino et al[250]	2012	5.9	15	8
Guilleminault et al[117]	2013	7.5	24	24
Villa et al[245]	2014	3.7	22	18

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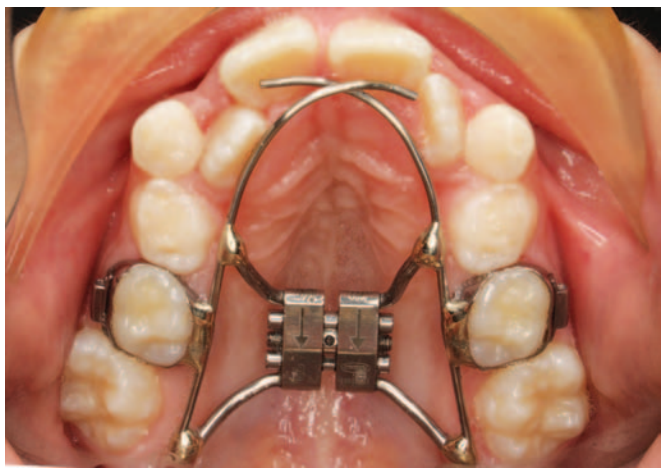

Figure 143-2 Example of maxillary expansion appliance in the mixed dentition.

f0015

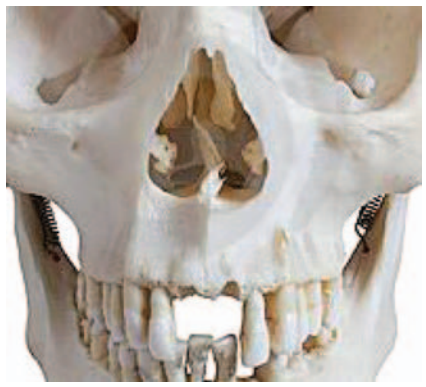

Figure 143-3 Skeletal boundaries of the nasal cavity. The maxilla forms the floor of the nose (F); the lateral walls are the sides of the maxilla (LW). This intranasal space is increased volumetrically with maxillary expansion.

eight controlled studies with 6-month follow-up after therapy that measured changes in airway dimensions and functions after RME. These studies support a moderate level of evidence that RME therapy in a growing child causes increases in nasal cavity width and in the posterior nasal airway, associated with reduced nasal resistance and increased total nasal flow.[158,159] The stability of the results can be expected for at least 11 months after the orthopedic therapy.[160] In one study, nasal airflow measured by rhinomanometry improved in the supine position in 65% of the patients.[161] Changes in nasal geometry of increases in intranasal width[153,162,163] and increases in both nasal cross-sectional area and nasal volume[153,164] were noted. Airway properties were examined, showing decreases in nasal resistance measured by rhinomanometry[153] and acoustic rhinometry,[164] along with changes in head position and decreases in the craniocervical angle.[161,165,166] RME therapy through sutural opening creates an increase in the nasal cavity width, area, and volume in children, which allows a reduction in nasal resistance.

p0355 The studies in Table 143-3 support maxillary expansion as a treatment modality for pediatric OSA syndrome. The majority of children responded to expansion therapy, and OSA was eliminated in a few children.[144,167] Most of the expansion studies looked at children with narrow upper jaws (selection criteria for treatment) and malocclusions, including crossbites, dental crowding, and mandibular retrusion. Expansion as a first-line therapy was initiated in a few of the studies. Mandibular retrusion was not specifically a factor in selection of patients, although it was noted in more than half of the patients studied. Two of the studies used bimaxillary expansion.[117,167] Bimaxillary expansion was employed because of the dental compensation in both the maxilla and mandible from a narrowed maxilla. The dentition is tipped inward toward the tongue, which creates a narrowed intraoral space. Dental expansion of the lower dentition aids in achieving maximum skeletal expansion of the upper jaw. The effectiveness of bimaxillary expansion as a treatment option for pediatric SDB was first described a decade ago.[168] The overall expansion data of all studies are varied, but mostly with a general improvement in both sleep parameters and subjective symptoms of SDB.

p0360 The therapeutic effects of maxillary expansion include increasing space for dental crowding,[169] increasing airway dimensions, and decreasing nasal resistance[153,155,156] in addition to treating SDB (Table 143-3). These studies also demonstrate a greater gain when RME treatment is rendered before the pubertal growth spurt peak, demonstrating an age-related phenomenon to long-term success. In the studies outlined in Table 143-3, expansion was started as early as age 3 years up to age 15 years. Whereas nonsurgical expansion of the palate is still possible beyond the age of 15 years, because of increased interdigitation of the suture in an older child, the widening that occurs is more often dental tipping that increases the intraoral volume and not true skeletal expansion

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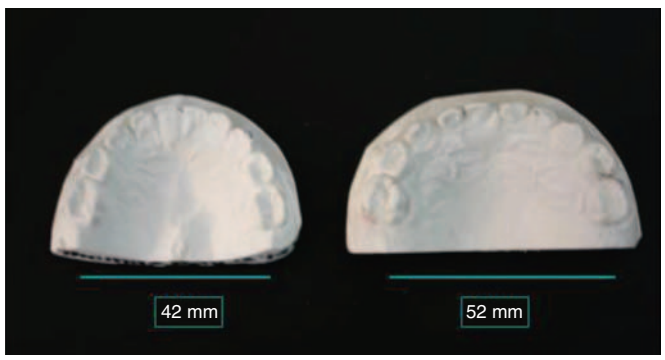
that increases the intranasal space. Greater changes in nasal width were evidenced when expansion was done early in maturation versus late in maturation.[162] By the age of 4 years, the craniofacial skeleton has reached 60% of its adult size; by the age of 7 years, 75% total craniofacial growth is complete, and by the age of 12 years, 90% total craniofacial growth is reached.[170] This suggests that RME therapy should be considered as an early-stage treatment in pediatric SDB, as the intervention timing seems critical for predicting RME orthopedics outcomes.

p0365 Maxillary expansion is a common noninvasive orthodontic treatment that is well tolerated by children. Advantages as therapy for SDB include little or no risk of morbidity or discomfort, with treatment performed in an outpatient setting during a 4- to 6-month period. There is a high level of acceptance for initiating this type of therapy, especially because many children with SDB also have concomitant dental crowding. However, some type of holding or retaining appliance is needed to maintain the expanded arch form, and so there is long-term ongoing care. If early expansion is advocated, a holding appliance may be in place for many years, and the timing is dependent on the eruption status of the dentition and the ability to sustain nasal respiration.

p9105 The reported risks of RME therapy include bite opening, relapse, microtrauma of the temporomandibular joint and the midpalatal suture, gingival recession, and root resorption.[158,160] Whereas these effects are not usually encountered, the incidence is also age dependent, seen more with maturation. There are no current guidelines for selection of patients, other than the studies that treated children with narrow palates and dental crossbites, in which teeth of the upper arch do not horizontally overlap teeth of the lower arch. Future work will help identity which types of patients will benefit most, how much expansion can be gained, and at what age to start expansion.[171] An example of the shape changes and the amount of space that can be gained is shown in Figure 143-4.

p0370 Whereas the expansion results for SDB are promising, these studies were not controlled or randomized and were limited to only a few groups that reported data. Few new therapies of pediatric SDB have been validated with randomized controlled trials.[251] Current pediatric guidelines recommend referring children with maxillary transverse narrowing for orthodontic therapy and possible RME treatment for persistent OSA syndrome after adenotonsillectomy.[122] Orthodontic expansion therapy has three potential effects for the SDB child. It can widen the intranasal volume to reduce nasal resistance, which improves airway collapsibility and SDB; it can facilitate other SDB treatment, such as positive pressure therapy, by allowing improved nasal airflow; and it can facilitate the transition from oral to nasal respiration, which can have a secondary effect on oropharyngeal growth. Because maxillary expansion is so well tolerated by children, it is expected that in the coming years, larger scale trials will examine the efficacy of jaw expansion as one component of improving the properties of the developing airway in a child with SDB. The effect of RME on orofacial growth and facilitating nasal respiration is discussed in the next part of this chapter.



f0025 **Figure 143-4** Example of the amount of expansion that can be created. Preexpansion model shows a width of 42 mm, measured from the central groove of the upper primary second molar, compared with postexpansion width of 52 mm.

Surgical Expansion: Removal of Soft Tissue. An alternative mode of increasing airway size is through the surgical removal of structures or obstructions. This is further described in Chapter 149, and for children, these procedures may include reduction of the inferior nasal turbinates, sinus surgery, or adenoidectomy. There is a nonlinear relationship between the level of nasal resistance and the severity of SDB. This may explain why the reported cure rate of adenotonsillectomy for OSA in meta-analyses of children with a mean age of 6.5 years was only 59.8%, showing that adenotonsillectomy as the first-line and most common therapy for pediatric OSA may be insufficient.[172] s0070 p0375

Location–Oropharynx s0075

The retropalatal area is represented here, which is often the site of greatest airway narrowing in children. SDB children have more fluctuation in airway size, with narrowing during inspiration that is more prominent in higher oropharyngeal levels.[127] Surgical adenotonsillectomy can enlarge this space, as shown in Box 143-5. Whereas maxillary expansion increases the intraoral space, imaging studies using cone beam computed tomography and lateral cephalography do not depict any changes at the oropharyngeal level with RME. OSA children have narrowed space at this level compared with controls, but after RME therapy, there was no evidence of increased oropharyngeal airway volume.[173,174] However, it has been postulated that the increased oral volume resulting from maxillary expansion induces a postural change in the tongue position.[175] p0380

Surgical Expansion: Removal of Soft Tissue. Hypertrophy of the tonsils (pharyngeal and palatine) is the second major cause of respiratory obstruction in childhood, followed by allergic rhinitis, and is found to be associated with allergic rhinitis in many children, exacerbating the respiratory symptoms.[176] As described earlier in this chapter, tonsils generally initiate hypertrophy within the first 3 years of life, the period of highest immunologic activity during childhood. Because tonsil growth outpaces craniofacial growth from 3 to 7 years s0080 p0400

Box 143-5 INCREASING THE OROPHARYNX SIZE b0030

Orthodontic expansion p0385
Surgical removal of soft tissue u0145

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b0035 **Box 143-6 INCREASING THE HYPOPHARYNX SIZE**

p0410 Oral appliances (Chapter 147)
u0155 Surgical expansion (Chapter 149)
u0160 Nasal continuous positive airway pressure (Chapter 115)

of age, most symptoms are observed during this period, coinciding with the peak incidence of childhood OSA syndrome.[177] Tonsil atrophy starts after 10 years of age and is completed in adulthood.[178] The first-line therapy for pediatric SDB is adenotonsillectomy, which has been associated with a decline in the critical closing pressure of the muscles along the pharynx, rendering the upper airway less collapsible.[121] Surgical therapies are further described in Chapter 149.

s0085 ***Location–Hypopharynx***

p0405 Hypopharyngeal airway obstruction can be caused by the prominence or relaxation of the base of the tongue, the lateral pharyngeal wall, and, occasionally, the aryepiglottic folds of the epiglottis. The treatments to increase the size of the hypopharynx are outlined in Box 143-6.

s0090 **Oral Appliances.** Several case report studies show the effec-
p0430 tiveness of oral appliances that hold the lower jaw forward in treating SDB and OSA in children. The use of oral appliances in the treatment of adults is well established, and the specific mechanics of how these appliances work is further discussed in Chapter 147. Oral appliances that advance the jaw forward are similar to the functional appliances used in orthodontics that address problems of mandibular deficiency. In children, these appliances create changes in the dentition and the growth of the maxillomandibular complex, and these alterations are thoroughly described in the orthodontic literature. A 2007 Cochrane review[179] examined studies in children aged 15 years and younger using mandibular advancement appliances. It concluded that there was not sufficient evidence to support treatment of pediatric OSA syndrome using oral appliances. In the more than 200 studies, only one study was recognized for inclusion,[180] which examined children aged 4 to 10 years against a control group of no treatment, showing a reduction in respiratory parameters in the majority of the treatment group. It must be highlighted that oral appliances can affect the forward growth of the upper and lower jaw, which likely makes their use inappropriate unless the child presents with retrognathia. The long-term side effects of this growth on the pediatric developing airway warrant further examination.

s0095 **Surgical Expansion: Skeletal Surgery.** Orthognathic advance-
p0435 ment surgery is not considered a treatment option for the pediatric patient until after jaw growth cessation. Because of the late-stage timing, other therapies would be enacted before orthognathic surgery is planned. Mandibular advancement surgery enlarges the hypopharyngeal space, whereas maxillary advancement surgery enlarges the oropharyngeal cavity. Bimaxillary or maxillomandibular advancement surgery creates expansion across the entire pharynx, primarily at the oropharynx and hypopharynx, with a secondary impact on the nasopharynx. Expansion/advancement surgery is described in Chapter 149.

B

s0100 **Continuous Positive Airway Pressure.** Nasal continuous posi-
p0440 tive airway pressure (CPAP) stents the pharyngeal airway open and prevents the muscular walls from collapsing. It is not a curative strategy as it does not increase the airway size or change the neuromotor properties of the surrounding musculature. Studies in children[181] demonstrate the efficacy of CPAP therapy in reducing or eliminating symptoms and improving respiration but also acknowledge the challenges of compliance with and adherence to routine use. Consequently, CPAP is used as a secondary measure when adenotonsillectomy, bimaxillary expansion, or pharmacologic management has not improved SDB or as a primary option for obese children or children with craniofacial syndromes. This form of therapy is further outlined in Chapter 115.

Strategy 2: Improvement in Muscle Response

s0105

p0445 If OSA is modeled as a disease of progressive muscle degeneration, one strategy for treatment would counter these resultant muscular changes that stem from pharyngeal muscle damage. The collapsible pharynx is a tube composed of paired muscles with no bony perimeter, mediating airflow from the nose in the upper airway to the lungs of the lower airway. With repetitive collapse in OSA, the sustained pressure in the collapsible pharynx can result in repetitive microtrauma to the pharyngeal muscles.[182,183] Over time, these insults can lessen muscular neural control of the upper airway muscles that regulate airway opening and collapse, the interplay of the airway dilating opening muscles against the airway contracting or closing muscles. The pharyngeal muscle activation can become altered such that the response to neurochemical (hypoxemia or hypercapnia), neuromechanical (respiratory effort), or sensory (afferent input) stimuli becomes diminished or absent.

p0450 The tongue muscle is one of the largest structures defining the oropharyngeal airway and bounds its anterior aspect with motor innervation by the hypoglossal nerve and sensory innervations by the lingual nerve and glossopharyngeal nerve. As the largest and most studied pharyngeal dilator muscle, it almost directly controls airway patency by its forward movement, enlarging the airway. It is composed of extrinsic muscles (genioglossus, hyoglossus, and styloglossus) that alter its position and intrinsic muscles that alter its shape, both of which can affect airway size and shape.

p9110 Although SDB is often an anatomic problem of small size, the tongue volume in children with OSA syndrome does not differ from that of controls.[184] Similarly, the other muscles that border the pharyngeal airway, such as the pterygoid muscles, pharyngeal constrictor muscles, and parapharyngeal fat pads, were similar in size in both normal and OSA syndrome–affected children. In children with OSA syndrome, there was no muscle thickening of the lateral pharyngeal wall as reported in adults, suggesting that the surrounding craniofacial structures create the deficient anatomic volume differences and not the muscular soft tissues. In contrast to OSA children, OSA adults show evidence of muscle remodeling in phenotype of muscle size or fiber type,[185] which affect the sensory properties and strength performance of the muscle system. So, whereas the size of the muscle tissue mass is not enlarged in children with OSA syndrome, it implies that repetitive activations to the muscle can create muscle enlargement and changes in muscle properties (i.e.,

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length, force) and innervations (i.e., spindle, motoneuron) that then can become problematic in adults.[186] It is not known if the upper airway neural abnormality is related to muscle activation or muscle recruitment; much more work is need to understand such complex interactions and impact on breathing.

p0455 At sleep onset, pharyngeal muscle activity is reduced, and it becomes slightly atonic during rapid eye movement sleep (see Chapters 21 to 23 for more information on muscle physiology and breathing). During adolescence, the dilating upper airway reflexes show a gradual reduction of responsiveness, and so the collapsibility of the upper airway increases.[187] Some amount of airway reflex attenuation or blunting occurs with age. Normal children and adolescents show neuromuscular compensations of increasing genioglossus electromyographic (EMG) activity in response to increased resistive loading, indicating that these children and adolescents have active upper airway neuromuscular reflexes during sleep. OSA syndrome children and adults show higher EMG activity during wake than normal controls and lower EMG activity during sleep.[188,189] These daytime increases in EMG activation impart a resistance to collapse,[190] and these mechanisms have been described in obese non-OSA children[191] as a neural compensation. Snoring may or may not be present in children, but over time it creates vibratory stress as mediated in the upper airway and is hypothesized to induce change or injury to the affected pharyngeal muscles.

p0460 Because of this eventual blunted airway muscle reflex to motor and sensory stimuli, efforts have been targeted toward muscle rehabilitation, specifically to address deficiencies in muscle activation. It is thought that these changes in the upper airway muscles are reversible. Electrical neurostimulation as a form of muscle training activation on animals and human subjects was experimented to maintain pharyngeal airway patency during sleep. Stimulation was applied directly on the hypoglossal nerve[192] or the dilator muscles.[192-195] These studies show mixed results, but current investigations are ongoing.

s0110 *Myofunctional Therapy*

p0465 More recent work in muscle rehabilitation explores pharyngeal muscle exercise therapy to change muscle strength, posture, and responsiveness. These oropharyngeal exercises of myofunctional therapy (MFT, also called orofacial myofunctional therapy and orofacial myology) are intended to improve the neuromechanical performance of the pharyngeal dilators. History dates MFT to the field of speech pathology, with references to the orthodontic literature in 1906, recognizing the role of the facial musculature in the development of malocclusions. The term myofunctional therapy was coined in 1935.[196] MFT by definition is a tongue and lip muscle exercise therapy to treat malocclusions and other dental and speech disorders, usually as a result of an anterior tongue displacement pattern.[197] It has also been implemented as a cotherapy for orthodontic management of craniofacial growth along with the treatment of crossbites and anterior open bite malocclusions.[198,199] With a high level of evidence, a form of oral MFT has been used to treat dysphagia resulting from neuromuscular movement disorders or stroke patients.[200] The potential mechanisms implicated in the growth deceleration of OSA include dysphagia resulting from adenotonsillar hypertrophy.[201]

p0470 The tenets of MFT address muscle hypotonia and weakness by strengthening the nasal mode of respiration using resistance and range of motion exercises. Performance of these exercises during the day supposes that airway collapsibility during sleep will be reduced. As the pharyngeal muscles are involved in creating speech, swallowing and deglutition, and breathing, therapy will treat dysfunctions in abnormal drinking, chewing, and swallowing while correcting the resting posture of the lips, tongue, and jaw by repatterning of facial muscles.[202] This is thought to restore the altered sensory input and proprioception to the affected musculature. Swallowing as an upper airway reflex is impaired in OSA patients,[203] suggesting that targeted therapy for swallowing could improve upper airway motor tone, although the degree of swallowing impairment does not correlate with OSA severity.[204] Preliminary studies of muscle rehabilitation exercises combined with oral shields were effective in reducing reports of snoring in normal and overweight (but not obese) middle-aged individuals as measured on an analog scale in non-OSA adults.[205] OSA young adults in a small-scale trial using MFT for 2 months showed overall improvement in AHI, oxygen saturation, and strength of the perioral musculature. The small sample size of six patients warrants further investigation to strengthen these findings.[206]

p0475 Randomized and controlled studies using MFT in adults reinforce the efficacy of muscle exercises on decreasing airway collapsibility in the middle-aged population with moderate OSA. One study with almost daily didgeridoo instrument playing for 4 months' duration in combination with breathing training yielded improved results of AHI and SDB symptoms as measured by various scales.[207] Another investigation using a highly specified regimen of oropharyngeal exercises examined an overweight, middle-aged, and predominantly male cohort that underwent 3 months of upper airway muscle rehabilitation. Compared with controls, the treatment group showed improvements in all outcome variables of improved AHI severity, lowest oxygen saturation, and symptoms and thus laid the foundation for further exploration.[208]

p0480 The muscle remodeling phenomenon seen in adults has not been evidenced in children, but the tenets of muscle strengthening can be applied to pediatric SDB. A retrospective study of adolescent youth showed resolution of the breathing abnormalities seen during sleep, but only after MFT was supplemented to the treatment regimen. Prior adenotonsillectomy and orthodontic expansion were other treatments used to increase nasopharyngeal and oropharyngeal space; however, flow limitations and daytime symptoms of mild OSA persisted after these therapies.[123] A prospective study of MFT and twice-daily nasal lavage was instituted for a 2-month period for children older than 4 years with residual moderate to severe OSA after adenotonsillectomy. This randomized and controlled work showed considerable improvement in AHI severity in all 14 children using MFT.[209]

p0485 These studies provide evidence to identify MFT as a potential adjunctive treatment for SDB in adults and children. MFT yielded improvements in both respiratory parameters and symptoms of SDB. The aspect of inadequate muscle activation or recruitment is not completely managed with mechanical (CPAP), surgical, orthodontic, or medication therapy. MFT may address these problems of diminished pharyngeal muscle motor tone from neural motor and

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sensory deficiencies in all parts of the pharynx, extending from the nasopharynx to the hypopharynx. Future work in this area will likely outline screening and treatment parameters and identify the most effective muscle rehabilitation techniques for managing SDB. Most patients who undergo MFT do not have imaging to demonstrate an abnormal swallow reflex or have EMG tests to show abnormal muscle activation, and so identification of selection criteria is ambiguous. Longer term studies can address questions of treatment timing, age, and severity appropriateness and lend more definition to the initiation, end point, and duration of MFT. (See also Chapter 150 for other approaches to improving SDB.)

s0115 **Strategy 3: Craniofacial Growth Modification**

s0120 ***Changing the Pattern of Respiration***

p0490 Does oral breathing cause abnormal orofacial development, and if so, does this cascade of altered growth exacerbate SDB or predispose a growing child to SDB? A simple cause and effect relationship between upper airway respiratory function and dentofacial and skeletal morphology has not yet been determined, despite the myriad studies in the clinical specialties of otolaryngology, oral surgery, and orthodontics—all areas that are specific to treating the upper airway, the jaws, or the teeth. However, there are associations between OSA syndrome, craniofacial anatomy, facial growth, jaw size and shape, and malocclusions as outlined earlier in this chapter. The genesis of these relationships stems from the hypothesis that oral breathing from impaired nasorespiratory function can affect craniofacial growth. Although controversial, the relationship between nasal obstruction and facial growth has been demonstrated in animal experimental and human clinical trials.[210]

p0495 Because the nasal respiratory pattern is critical in the development of a normal upper airway, initial SDB treatment strategies in children are targeted to promote and to sustain nasal respiration. Removal of nasal obstructions can promote nasal respiration, during the day and night, but the relevance of establishing daytime nasal respiration in affecting nighttime upper airway properties is not known. If nasal obstruction is sufficiently severe, a transition to oral breathing may occur. Nasal breathing is the primary route of airflow, responsible for most inhaled ventilation during wake and sleep states. The transition from nasal to oronasal breathing at night increases with age.[211] Transitioning to primarily oral airflow can be detrimental because it can change the upper airway properties. Open mouth breathing during sleep can create lengthening of the pharynx and lowering of the hyoid bone,[212] which increases the upper airway resistance from increased collapsibility of the pharyngeal lumen and posterior retraction of the tongue. Upper airway resistance during sleep is significantly lower during nasal breathing than during oral breathing,[213-215] which may further compromise the airway and increase the effort of breathing if oral breathing is sustained during sleep.[135]

p0500 Oral breathing in children can also have a potentially dysmorphic effect on the developing orofacial complex. Based on the earlier described functional matrix theory of Moss, it is thought that nasal respiration provides continuous airflow through the nasal passage during breathing to induce a constant stimulus for the lateral growth of the maxilla and for lowering of the palatal vault. The position and shape of the bone are the result of air pressures through the nasal and oral cavities.[216] The second effect of oral breathing on the facial skeleton is mediated by changes in muscle recruitment patterns, which result in an altered soft tissue and skeletal morphology and posture.[217] Increased nasal resistance can yield a more collapsible airway, and it potentiates mouth breathing that can create the postural and thus muscular changes that lead to unfavorable jaw growth. So the oral breathing cycle is perpetuated once it is initiated. This suggests that one of the tenets of SDB therapy would be to change the respiratory pattern from oral breathing to a predominantly nasal breathing mode.

p0505 Before strategies to create oral breathing can be created, it is challenging to understand what oral breathing means. There is not a standard well-accepted test to define an oral breather. The controversy stems from the inability to quantify nasal versus oral respiration or if spontaneous transitions from oral to nasal breathing occur and the lack of long-term data with growth maturation. Reliable tests to assess continuous airflow through the nose and mouth are lacking, and often the assessments are made on clinical presentation and subjective perceptions of the patient. Tests that measure airflow and nasal resistance, such as anterior rhinometry, acoustic rhinometry, nasal peak flow, rhinomanometry, and pneumotachography, are not routinely used because the testing results do not consistently correlate with the patients' subjective complaints.[218,219] Oral breathing is thought of as an oral habit or as an adaptive response to a perturbation. Despite the associations to maxillomandibular growth, few studies explore how to cure oral breathing. The most common respiratory mode is a combination of simultaneous oral and nasal airflow.[220] During sleep, normal subjects without nasal disease or SDB are nasal breathers, with only 4% of total ventilation time spent breathing orally.[221] Neonates are born as obligate or preferential nasal breathers, but as outlined earlier in this chapter, this changes as the upper airway matures. Even patients with severe nasal obstruction from either allergies or soft tissue enlargement display some measure of nasal respiration.[222]

p0510 Given the multifactorial nature of the nasorespiratory pattern and function on the expression of SDB and facial growth, relief of nasal obstruction to create nasal respiration is only part of the solution. There are likely other multilevel variables that should also be addressed, such as the development of nasal breathing at the habitual level. Some children with adequate upper airways breathe through the mouth because of habit.[222] Nasal obstruction can be the stimulus for oral breathing, but removal of nasal obstruction to create nasal patency does not always induce a spontaneous change in the respiratory pattern.[223] Although RME therapy can enlarge the intranasal space and reduce nasal resistance, the oral respiratory mode does not automatically revert,[155] suggesting that a patterned mode of breathing develops.[224] Increased nasal airflow is not enough to achieve nasal breathing because other factors, such as nasal concha hyperplasia, nasal polyps, adenoidal hypertrophy, and septal deviation, are responsible for oral breathing.[225] Even after surgical removal of enlarged tonsils in children, changes in airway muscle tone were variable after surgery,[226] demonstrating a spontaneous but partial motor tone and posture improvement.

p0515 Oral breathing is associated with tonic changes in the orofacial musculature but also in head posture, where the head is extended and forward of the spine.[227] In this position, the

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cervical musculature shows higher muscle activity, measured by electromyography, in at-rest oral breathers compared with nasal breathers, showing that there is cervical musculature recruitment.[228]

p0520 This modification of the muscle recruitment pattern when the mode of respiration changes from nasal to oral breathing was demonstrated in experimental animals.[217] Therapy exercises aimed at these cervical muscles can create reduced EMG activity as seen in nasal breathers.[228] So the postural mode can influence the respiratory mode as in habitual oral breathers,[229] and this affects muscle balance. This suggests that muscle exercise therapy treatment be incorporated to reinforce a habitual mouth-closed mode of breathing. MFT could target the orofacial and cervical musculature either to reverse the adapted muscle recruitment patterns or to strengthen tonicity to change the upper airway caliber. Use of MFT as adjunctive therapy to firmly establish the nasal mode of respiration is an area to be explored.

Passive Force Application s0125

Shape Changes in the Upper Jaw. As described earlier in this chapter, the muscle pattern exerts a passive tension on the developing airway through the force mediated on the maxilla. Numerous studies have shown a relationship between nasal airway obstruction and aberrant facial growth. In monkeys, the nasally obstructed animals had longer faces and unusual dental malocclusions. In children, these same morphologic effects of impaired nasal respiration are evident, including narrowing of the maxilla, reduced development of the mandible, increased vertical growth of the lower face, dental crowding and malocclusion,[30,230] and altered head posture. A new posture compensates for the decrease in nasal airflow to allow respiration. However, these are not consistent phenomena in children with oral breathing tendencies.[231] s0130 p0525

These changes are typified in Figure 143-5 and occur passively because there is no directly applied force or load. In response to chronic oral breathing, three distinct p0530

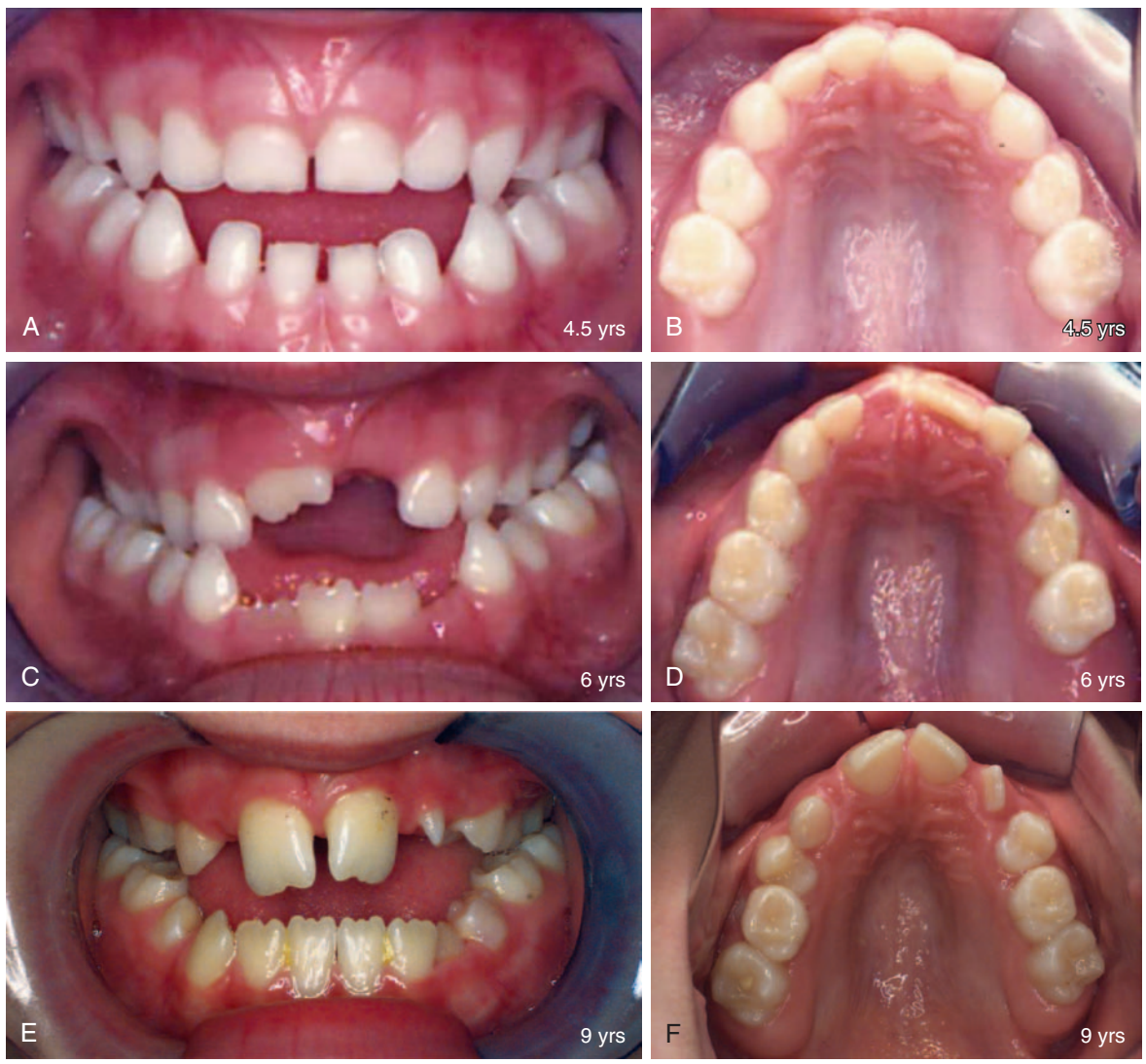

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Figure 143-5 A–F, Natural growth of the maxillomandibular complex in response to adenotonsillar hypertrophy and oral breathing. **A** and **B,** Taken at age 4.5 years. **C** and **D,** Taken at age 6 years. **E** and **F,** Taken at age 9 years. Note the gradual constriction of the upper jaw from the age of 4.5 to 9 years, while the lower jaw remains an intact shape.

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developmental changes may be manifested. The upper jaw narrows, suggesting that the volume of the nasal cavity becomes smaller as the upper jaw continues to narrow. As nasal volume decreases, the nasal resistance increases, which can exacerbate upper airway collapsibility, causing a cycle of progressively worsening SDB. This induced distortion in morphogenesis could partly explain the pathogenesis of SDB. The second developmental change is seen in the dentition, as the teeth add a second layer of insult to this distortion by compensatory inward tipping in both the upper and lower jaws. This restricts the intraoral volume even more, which potentiates a postural and neuromuscular adaptation of the genioglossus muscle. It can be argued that aggressive efforts are needed to combat the inherent tendency for upper jaw narrowing in oral breathers, even after the obstacles to nasal airflow are removed, because of the muscle alterations. The subsequent effect on the mandible is the third level of passive distortion that intensifies the pharyngeal collapsibility as the hypopharynx narrows with the backward rotation of the mandible. This narrowing is specifically evident at the retroglossal area.

s0135 **Shape Changes in the Lower Jaw.** In a growing child, abnor-
p0535 mal nasal resistance may affect the maxilla and the mandible.[232] During puberty, these deleterious changes can be magnified because of the growth velocity seen during teenage years. Deleterious effects of mouth breathing are the sensory stimulus that propagates abnormal tongue position and altered orofacial musculature tone. The muscle recruitment pattern is modified when nasal respiration changes to oral breathing.[217] Some of these growth responses can be exaggerated because of the concurrent effect of molar tooth eruption occurring at the same time.

p0540 In oral breathers, it has been reported that mandibular growth is redirected posteriorly. Oral breathing that develops from nasal obstruction has an effect on the largest pharyngeal dilator muscle that keeps the airway open, the genioglossus. In an open mouth posture, the genioglossus adopts a low-lying position in the floor of the mouth. The relationship of breathing to airway problems shows a lowered position of the hyoid bone in OSA syndrome patients. In this dorsocaudal position, EMG activation of the tongue on experimental animals shows a weaker protrusive force,[210,217,233,234] which suggests a possible mechanism for increased collapsibility as decreased genioglossus tension led to an increased muscle length in experimental animals.[233] The increased size of the genioglossus would narrow the pharyngeal airway, and this increases collapsibility. Genioglossal activity is increased with nasal compared with oral breathing, with supine versus sitting body position, and during inspiratory resistive loading,[235] probably because of an altered respiratory drive and reflex activation. This effect coupled with the potential edema from the negative airway pressure generated during sleep[236] exacerbates the disorder, exemplifying the necessity to treat the sequelae of the root cause. A cyclic pattern of distortion develops in which the lower jaw rotates backward because of mode of breathing. This changed positvion narrows the retroglossal airway, which could potentiate increased repetitive activation stimuli to keep the airway open, leading to an increase in pharyngeal muscle mass. Further muscle injury can result from snoring, vibration, or increased airway resistance.

p0545 The term adenoid facies has historically been used to describe the long, narrow, flat face of the individual with oral hypotonia and protruding teeth and lips that are widely separated at rest, often accompanied by an open bite malocclusion.[30] The original work on experimental animals by Harvold et al[210] demonstrated that there is a variable response to this insult of forced oral respiration. Oral breathing was not linked to a specific skeletal structure. Similarly, there is not a defined facial type or malocclusion that accompanies adenotonsillar hypertrophy in an oral breather. Several studies corroborate this conclusion because there is not a consistent relationship between nasal resistance and dentofacial morphology, as described earlier in this chapter. These differences are illustrated in Figure 143-6, which shows varied facial phenotypes with OSA. Medical intervention has not been shown to influence the pattern of facial growth in children with allergies. Surgical therapy to relieve nasal airway obstruction in children (whether adenoidectomy or turbinectomy) has not been shown to predictably affect ultimate facial form.[237]

p0550 The three cases illustrated in Figures 143-7 to 143-12 demonstrate the variable response of mandibular growth to impaired nasal respiration. All three patients had therapy for their malocclusion, but the problem of allergies and oral respiration persisted. The records shown were taken during a period of adolescent growth, illustrating a lack of forward upper jaw development. Whereas there is an inherited growth pattern that accounts for the familial tendency or inheritance in OSA, there can be different epigenetic expressions of craniofacial malformation overlaid on the inherent preexisting growth presentation.

p0555 Comparison of the general superimposition tracings, from three subjects, shows a general descent and slight rotation of the maxilla but variable responses of the mandible (Figures 143-8, 143-10, and 143-12). All three subjects had nasal obstruction that persisted after expansion treatment, and the disproportionate jaw growth was expressed during adolescence. The mandible showed posterior rotation (Figure 143-8) and straight vertical descent with no forward growth (Figure 143-12) or forward growth rotation, versus forward anterior mandibular growth rotation (Figure 143-10). The dentoalveolar development from tooth eruption can affect the forward (Figure 143-10) or backward rotation (Figures 143-8 and 143-12) of the mandible, and this important effect cannot be discounted. These are three different expressions of mandibular growth, from the same stimulus of increased airway resistance from nasal obstruction causing differences in muscle recruitment patterns that changed muscle tone and function. This created variable adaptations of soft tissue, which influence the skeletal morphology. Thus, the oral breather may present with a normal appearance to severe skeletal and dental irregularities. "Nasal obstruction presents the trigger factor, but it is the deviant muscle recruitment which causes maldevelopment."[210]

p0560 These cases illustrate the answer to the question about disease propagation if craniofacial malformation is not only a cause of SDB but a consequence of SDB. It is a variable response, depending on how the effect of impaired nasal respiration affects the muscle recruitment and activation. For some patients, this paradigm of propagation would hold true. The challenge is to identify those patients who are more at

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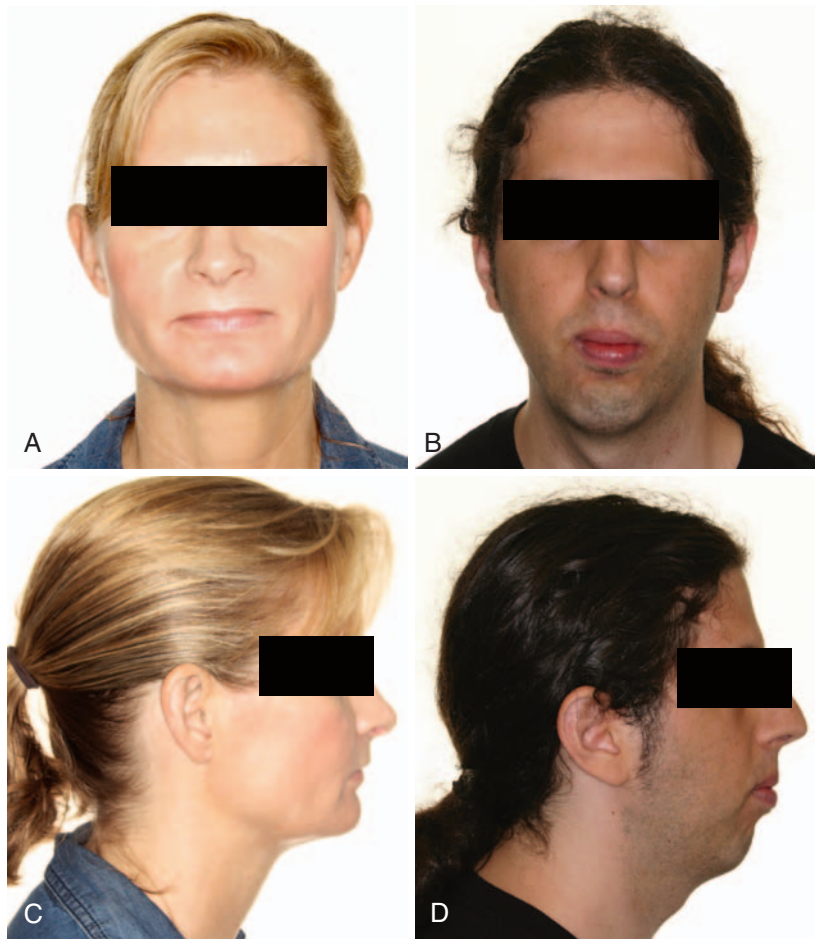

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Figure 143-6 Contrasting facial morphologies associated with OSA. **A, C,** and **E** Decreased lower facial height of an individual with a closed gonial angle and bimaxillary retrusion. **B, D,** and **F** Increased lower facial height of an individual with bimaxillary dental protrusion and an open gonial angle.

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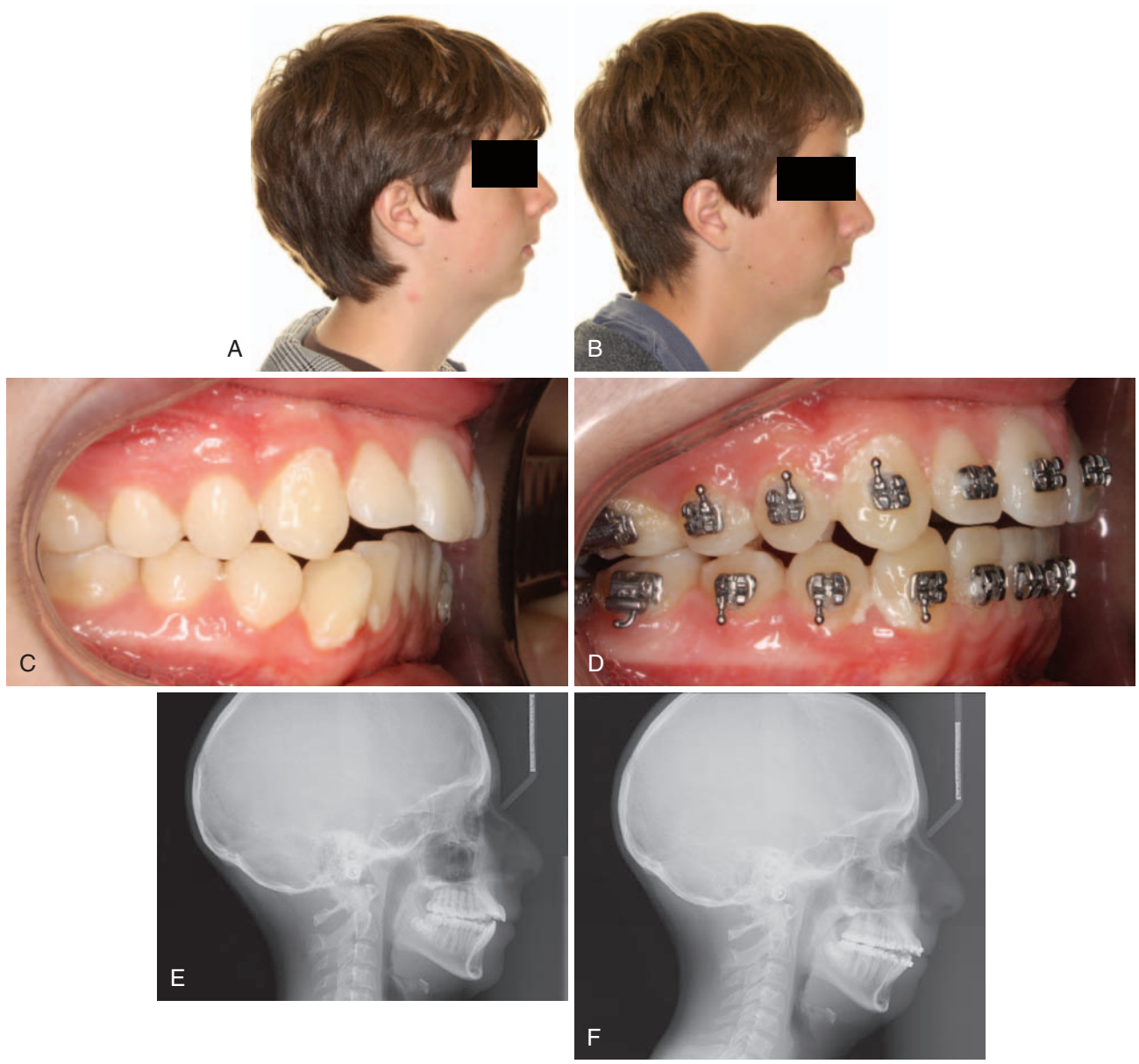

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Figure 143-7 Growth changes during adolescence. **A, C,** and **E** Presentation at the age of 14 years before the initiation of orthodontic therapy for malocclusion correction. **B, D,** and **F** Growth and dental changes 12 months later.

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f0045 **Figure 143-8** General growth superimposition during a 7-month period in adolescence of the patient in Figure 143-7. Method of Bjork, superimposed on cranial base. Vertical lowering of the maxilla is noted, with backward growth rotation of the mandible.

risk for aberrant muscle alterations, and for these patients muscle rehabilitation may be beneficial.

s0140 ***Active Force Application***

s0145 **Induced Bone Remodeling.** If bone remodeling is the result
p0565 of passive force application, active force application as an epigenetic effect to direct bone development could be a strategy to increase airway size or to redirect unfavorable growth patterns, beyond the inherent genetic expression. Not all patients develop a dysmorphic growth pattern as a result of impaired nasal respiration, so interventional therapy to modify existing growth patterns may not be justified when an aberrant growth pattern cannot be predicted. However, if these therapies help improve the critical closing pressure, the pharyngeal collapsibility would ultimately be improved or even normalized.

p0570 The consideration for this direction of care lies in the shortcomings of current therapies. The "gold standard" for treatment of OSA in adults is nasal CPAP. Whereas adenotonsillectomy is the first-line therapy for children, nasal CPAP is considered after other therapies have not completely improved respiration. Nasal CPAP is burdened with problems of compliance due to mask fit, comfort, or initiation of CPAP use. As an extraoral appliance, it will exert a molding force on the facial skeleton and the dentoalveolar bone. This force can cause remodeling and redirection of maxillomandibular jaw growth in the pediatric patient.[238-241] This retrusive remodeling force from the positive pressure from the mask or machine can create a skeletal jaw imbalance and negate all earlier efforts to enlarge the airway size, as seen in Figure 143-13, *A* and *B*. As increased nasal resistance is both a cause and consequence of SDB, an often prescribed option for treatment not only treats the problem but can perpetuate the problem too. However, induced bone remodeling through active force application can rectify these iatrogenic craniofacial alterations (Figure 143-13, *C*). There remains the question as to how long this imbalance is to be sustained.

p0575 Placing applied pressure (or force or loading) on the craniofacial bone causes shape and size changes. Future directions in care will test these boundaries. RME appliances use the dentition for force delivery, but there can be unwanted tooth movement side effects that accompany the skeletal change. Removable appliances that attach to the teeth to change the muscle tension on the underlying bone have been used with a small degree of success. One future avenue for force delivery uses temporary attachment devices, called TADs, anchored directly into the bone, that bypass the dentition.[242,243] The advantage of this type of direct force application to the bone is the direct and possible increased effect of loading on bone modeling while minimizing the side effects to the adjacent teeth and periodontium.

p0580 Through the combination of therapies, it may be possible to modify the inherent growth pattern. Efforts to target therapy at the site of greatest airway narrowing or obstruction may not be adequate because airway narrowing and collapse are thought to occur at multiple sites.[244] There are currently no studies that detail the effectiveness of a comprehensive and combined approach of treatments and growth modification to the nasopharynx, oropharynx, and hypopharynx while also implementing muscle rehabilitation to address the concomitant neuromuscular adaptations of an impaired airway. Although there are studies that document the efficacy of adenotonsillectomy and jaw expansion together as a treatment modality[167] and studies that describe adenotonsillectomy and MFT,[245] there remains to be examined the full complement of creating the largest patency of the airway using the modalities described in Box 143-3. These types of studies would be difficult to design, and longitudinal data across several different populations would be needed. The 11 known collections of longitudinal craniofacial growth records of untreated children in the United States and Canada could serve as the comparison of treated to untreated populations. This monumental effort would necessitate the collaboration of the multiple specialists involved in the treatment of upper airway disorders.

p0585 Prophylactically treating the entire pharynx, instead of targeting the site of greatest obstruction or narrowest opening, may seem aggressive and extreme. However, children with SDB have blunted responses to hypercapnia,[246] demonstrating early neural deficits that are thought to be reversed with treatment, but it is not known if there is complete recovery. Reports of a high recurrence of SDB in later teenage years[117] suggest that some neural dysregulation persisted and perhaps worsened after previous treatment. Treatment is usually initiated from daytime or nighttime symptoms. The onset of symptoms may underlie a larger problem, as it is not known how long the primary cause needs to be present before symptoms develop. The most viable treatment may be consistent early screening to identify patterns before the onset of symptoms. By the time treatment is rendered, even if it is done at any early age, there may already be alterations in muscle recruitment and tone that initiate the development of a craniofacial malformation. A recent population-based longitudinal study observing children from 6 months to 7 years of age found that early symptoms of snoring, mouth breathing, and witnessed apneas had statistically significant effects on behavior in later

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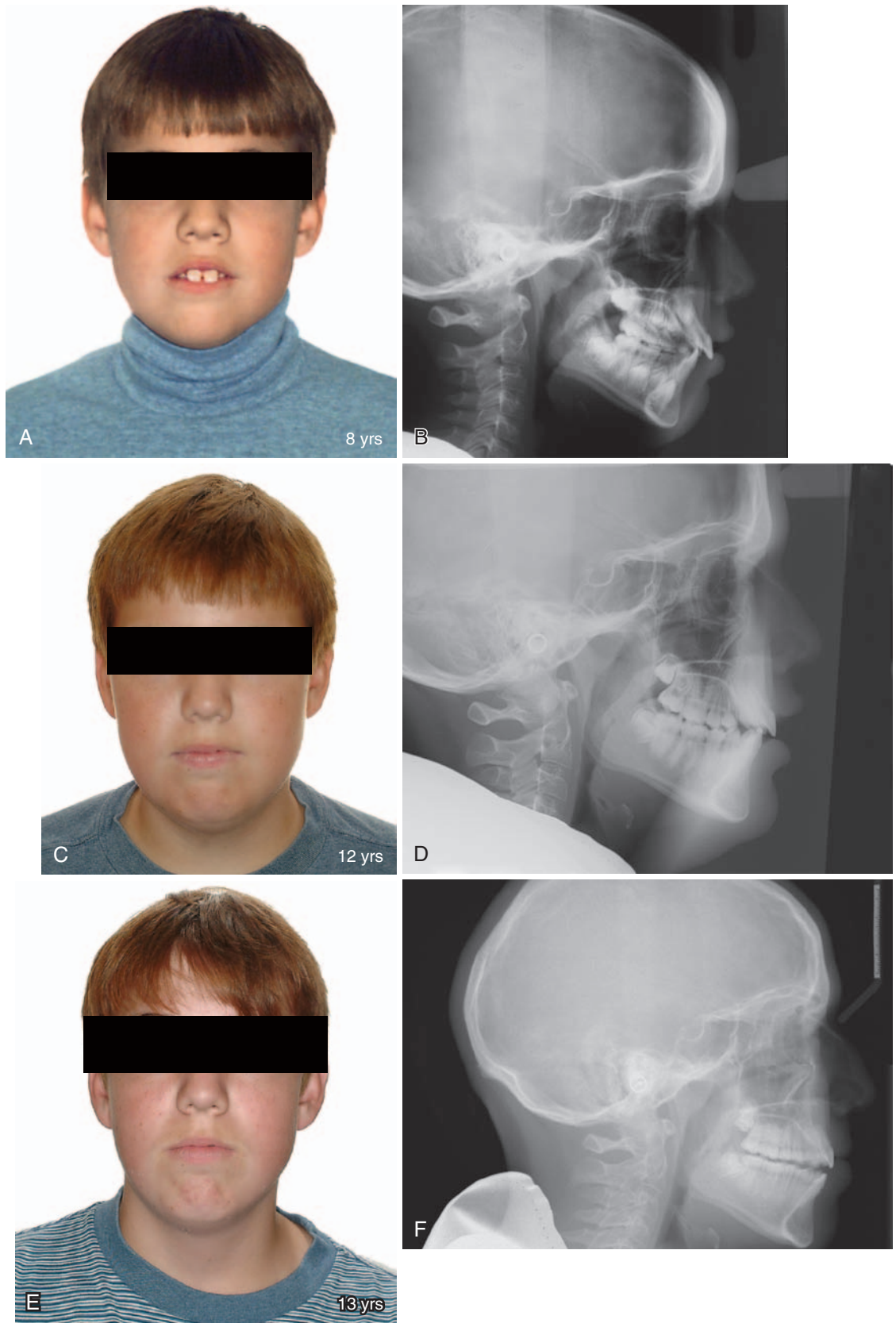

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Figure 143-9 This patient had orthodontic intervention of maxillary expansion for the malocclusion. **A** and **B,** Facial proportions and jaw relationship at the age of 8 years, initial presentation. **C** and **D,** Vertical lowering of the mandible after 4 years. **E** and **F,** Narrowed posterior airway space, continued vertical lowering of mandible, and forward jaw rotation after 5 years. No treatment interventions were rendered between 12 and 13 years of age. Note the dramatic change in growth velocity of the lower jaw from the age of 12 to 13 years.

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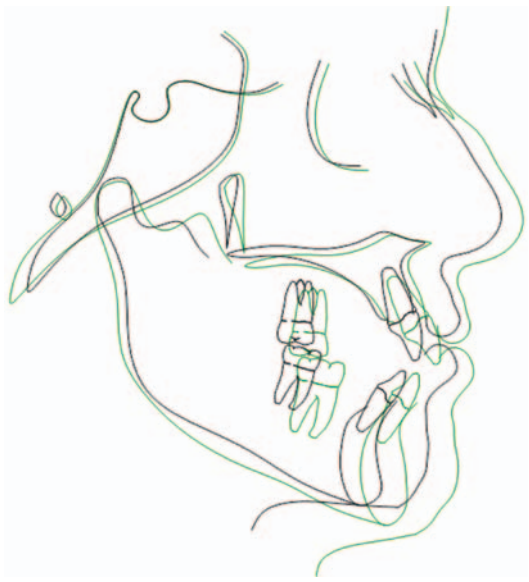
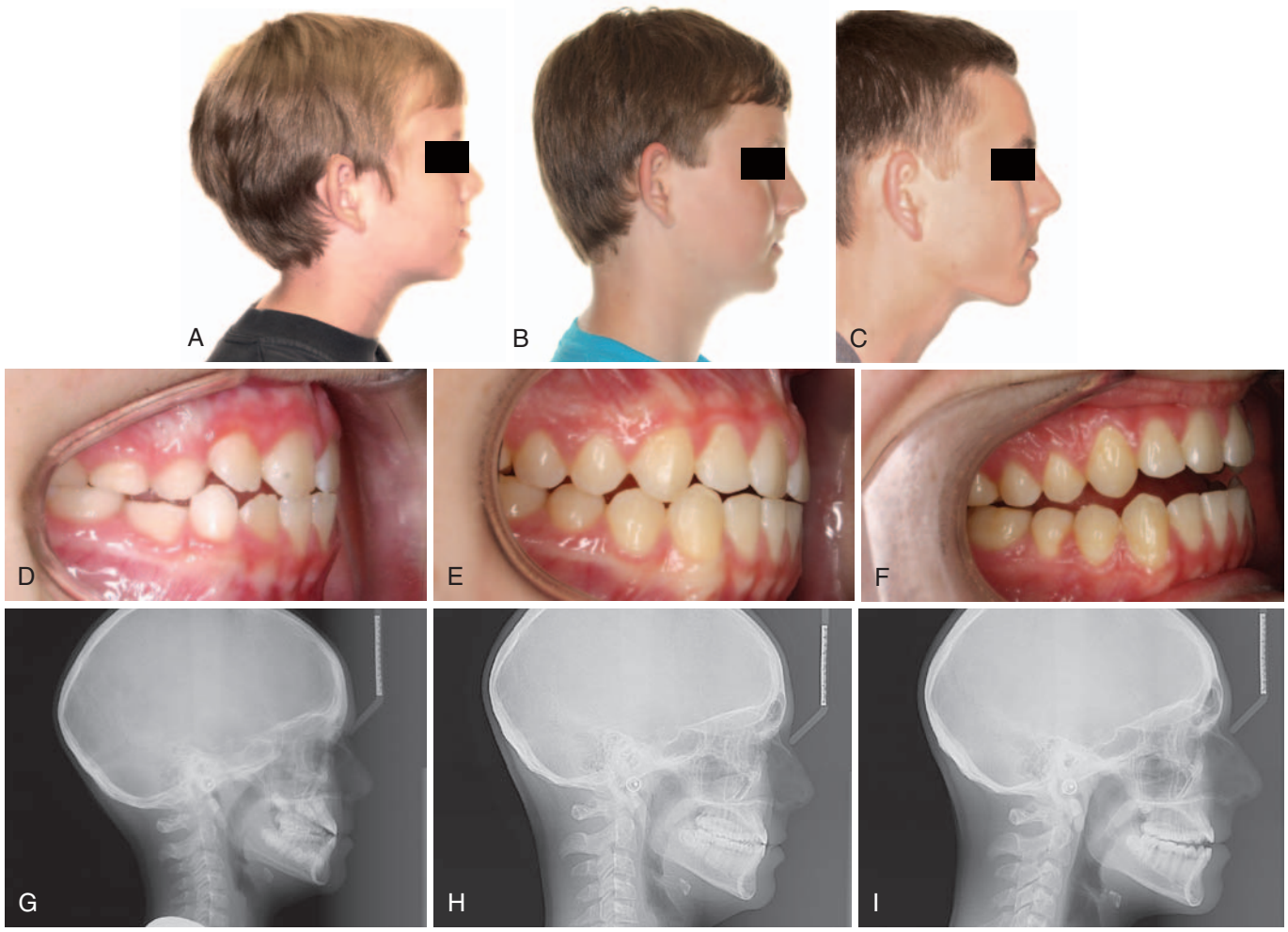


f0055 **Figure 143-10** General superimposition from 12 to 13 years of age of the patient in Figure 143-9. Method of Bjork, superimposed on cranial base. Forward growth rotation of the maxilla and mandible are evident.

childhood, suggesting that early symptoms warrant examination as early as the first year of life.[247] Herein lies the challenge of defining reliable screening patterns that are predictive of the disease.

p0590 A stepwise evidence-based approach for the diagnosis and multitherapeutic management of childhood SDB has been recently presented.[248] This approach, starting with weight control followed in succession with nasal corticosteroids, adenotonsillectomy, dentofacial orthopedics such as mandibular advancement or maxillary expansion, CPAP, and maxillofacial surgery, is illustrated in Figure 143-14. This case demonstrates that despite early best efforts at recognition, diagnosis, and intervention of multiple therapies, including adenotonsillectomy, allergy management, multiple rounds of bimaxillary expansion, and nasal CPAP, the upper airway problems may still persist. For this particular case, problems of CPAP adherence rendered it ineffective as long-term treatment. Ultimately, on growth cessation, maxillomandibular advancement was the final treatment rendered, which normalized respiratory parameters and symptoms. Anatomic insufficiencies were improved to maximize the enlargement of the pharyngeal space.



f0060 **Figure 143-11 A–C,** Growth changes during adolescence. Initial presentation at the age of 8 years, when upper jaw expansion was done. **D–F,** Growth presentation 5 years later. **G** and **H,** Growth presentation with no treatment interventions between the ages of 13 and 15 years.

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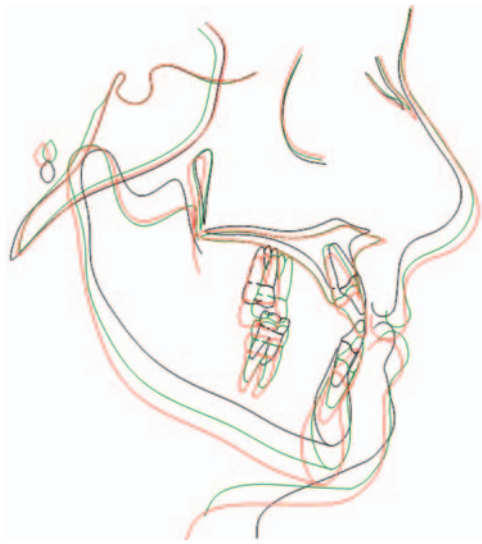

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Figure 143-12 General superimposition, method of Bjork, superimposed on cranial base of the patient in Figure 143-11. The lowering of the mandible for oral respiration was followed by a downward displacement of the maxilla with an increased extrusion of the teeth.

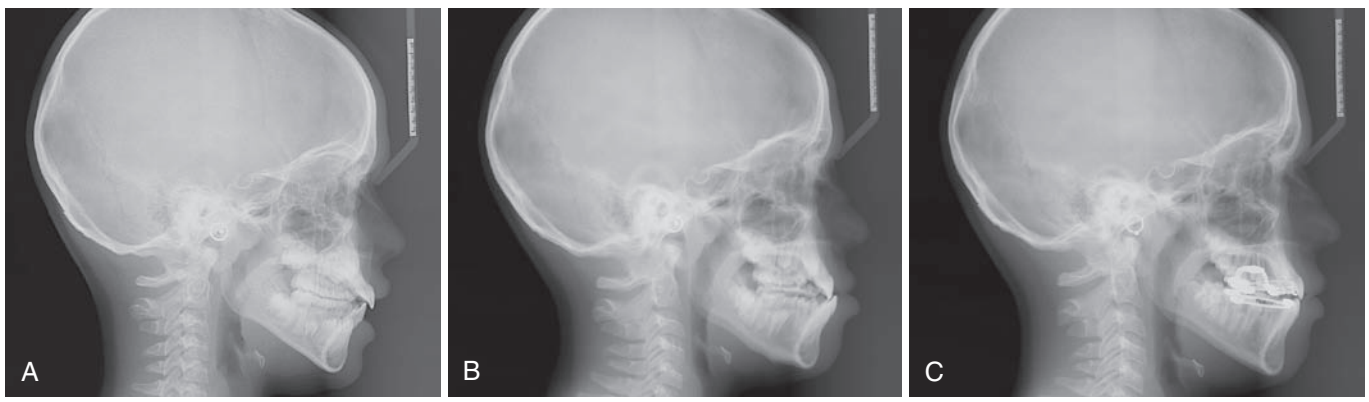

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Figure 143-13 Effect of CPAP on facial growth. **A,** Lateral cephalogram of an adolescent boy before initiation of nasal CPAP. **B,** Lateral cephalogram showing retrusion of the upper jaw in response to nasal CPAP (mask type: nasal mask) for 12 months. **C,** Lateral cephalogram showing protraction of upper jaw from orthodontic orthopedic forward traction of 8 months to the maxilla, while still using CPAP (mask type changed to nasal pillows).

b0040

CLINICAL PEARLS

- The altered craniofacial morphology associated with SDB has been well classified and trends to growth of longer and narrower facial structures. The exact mechanism by which this maladaptive plasticity of growth occurs is not well understood but is most likely related to impaired function that is both caused and perpetuated by genetic and epigenetic factors.
- Nasal CPAP as an extraoral appliance will exert a molding force on the facial skeleton and the dentoalveolar bone, causing remodeling and redirection of maxillomandibular jaw growth in the pediatric patient. This retrusive remodeling force from the positive pressure from the mask or machine can create a skeletal jaw imbalance and negate all earlier efforts to enlarge the airway. Pediatric mask selection should minimize face contact to reduce the retrusive molding pressure to the maxilla.
- When children with SDB present with the specific abnormality of narrowed palate or posterior dental crossbite, treatment with rapid palatal expansion is an effective, noninvasive therapy to improve disease symptoms. Another common means to orthodontically increase airway volume, using a mandibular advancement device in children, has limited but promising evidence in retrognathic SDB patients.
- Improvement of both subjective and objective measures of SDB by MFT has been demonstrated in children and adolescents through the mechanisms of muscle strengthening and reinforcing nasal respiration. Despite impressive results, the overall number of patients is small, and future research should develop the full extent to which this form of treatment can be effectively applied.

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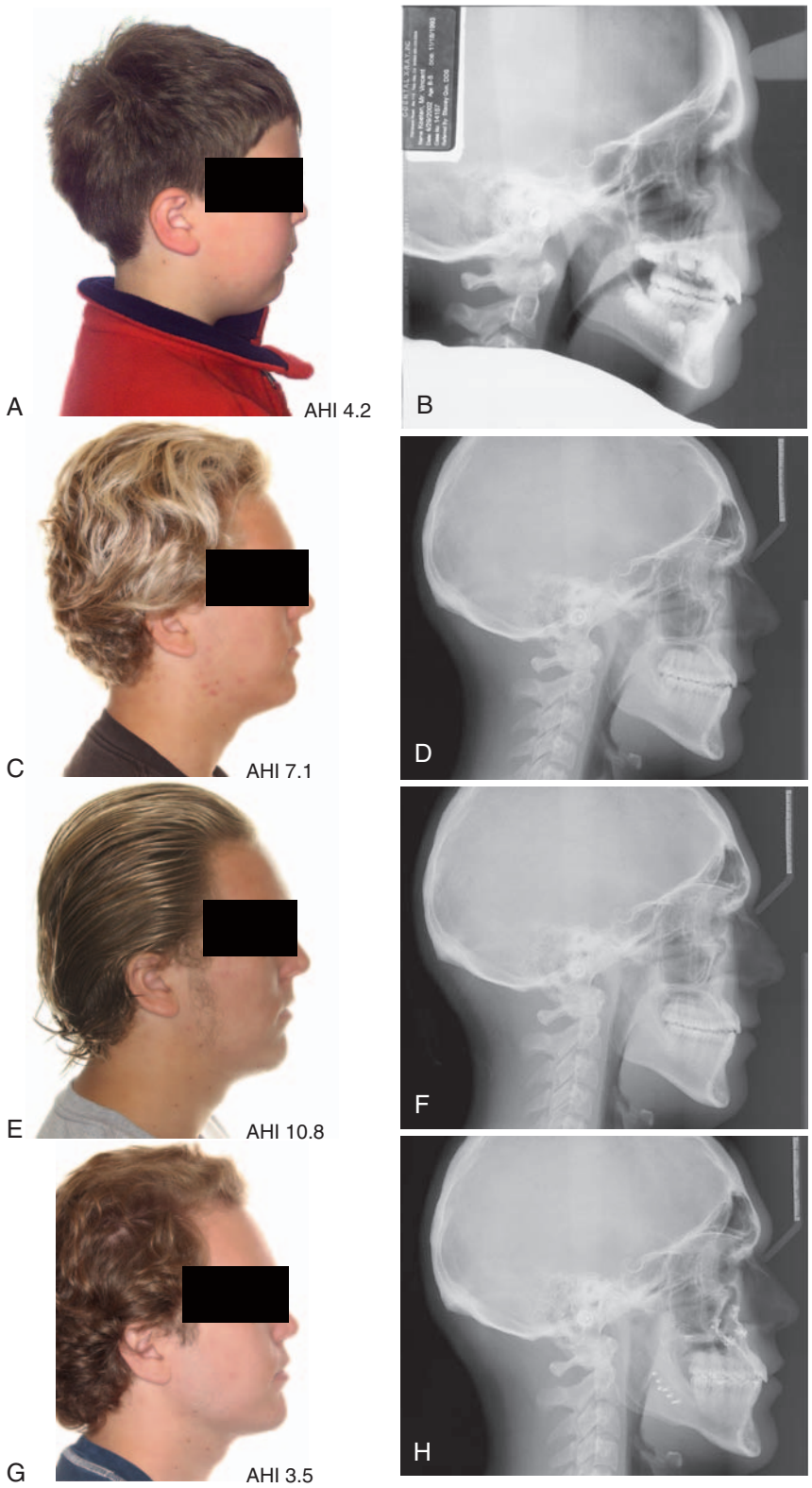

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Figure 143-14 Craniofacial development from the ages of 8 to 19 years. **A** and **B,** Records at age 8 years show airway and facial proportions after adenotonsillectomy, indicating residual SDB/OSA (AHI 4.2, residual symptoms). Bimaxillary orthodontic expansion initiated. Initial diagnosis of OSA at age 5 years (AHI 2.9 with daytime symptoms). **C** and **D,** Age 15 years, after bimaxillary expansion was completed at age 8 years and again at age 10 years. AHI increased to 7.1. **E** and **F,** Age 17 years, in preparation for maxillomandibular advancement. AHI increased to 10.8 with disproportionate increase in symptoms. **G** and **H,** Age 19 years after maxillomandibular advancement, with normalization of AHI and resolution of symptoms. Note the pharyngeal airway enlargement and accompanying facial soft tissue profile changes.

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s0150 ## SUMMARY

p0615 Many of the factors leading to pediatric SDB create secondary morphologic changes that exacerbate and perpetuate the syndrome. The approach to establish normal daytime and nighttime respiration is a multidisciplinary endeavor, involving the treatment of all three components of the pharynx and the attendant surrounding musculature. As multiple treatments may act synergistically, a greater degree of collaboration between specialists in sleep medicine, otolaryngology, allergy, and orthodontics is warranted to establish the contribution made by each discipline to the outcome of pediatric OSA. To achieve successful treatment of upper airway disorders, future efforts aimed at modifying the anatomy can hold the promise of prevention of problems of constricted size and impaired muscle function because oropharyngeal malformation may be not only a cause but also a consequence of SDB.

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p0620 ***A complete reference list can be found online at ExpertConsult.com.***

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REVIEW QUESTIONS

p0625 1. Greater nasal width is obtained when expansion is done earlier in maturation.
o0170 A. True
o0175 B. False
o0180 2. Children and adults with obstructive sleep apnea syndrome have higher upper airway muscle activity during wake and lower muscle activity during sleep than individuals without this syndrome.
o0185 A. True
o0190 B. False
o0195 3. Dentofacial morphology can fully explain the pathophysiologic mechanism of sleep-disordered breathing.
o0200 A. True
o0205 B. False
o0210 4. Adenotonsillar tissue is largest relative to the surrounding anatomy between the ages of 4 and 6 years, which is the peak of obstructive sleep apnea incidence in children.
o0215 A. True
o0220 B. False

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ANSWERS

p0690 1. **A.**
o0230 2. **A.**
o0235 3. **B.**
o0240 4. **A.**

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