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Childhood Sleep-Disorder Breathing: A Dental Perspective

Kevin L. Boyd and Stephen H. Sheldon

Chapter 34

INTRODUCTION

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p0010 With their 1981 publication *Western Diseases: Their Emergence and Prevention*,[1] authors Hugh Trowell and Denis Burkitt essentially launched a new paradigm in medical education; many modern diseases are now better understood when viewed from an evolutionary perspective. As healthy circadian sleep cycling during childhood would have been absolutely necessary for our ancestors' survival and reproduction (collectively referred to as *evolutionary fitness*) over the vast time span of human evolutionary history,[2] sleep-related breathing disorders such as obstructive sleep apnea (OSA) were likely not a part of the human experience until fairly recently, and thus can be appropriately categorized as *Western* diseases (WDs).

p0015 Evolutionary medicine (EM), also known as Darwinian medicine,[3] is a new approach providing a useful framework for understanding modern diseases from an evolutionary perspective. Evolutionary oral medicine (EOM), or Darwinian dentistry, describes how EM principles can be applied to exploring the evolutionary basis of modern dentofacial maladies such as dental caries, periodontal disease and malocclusion. For example, one proposed explanation by EM/EOM proponents for why humans have only *recently* begun to become vulnerable to many modern diseases such as type 2 diabetes and dental malocclusion, for example, is the *Mismatch* hypothesis,[4] which postulates that current high prevalences of WDs in *industrialized* populations are due, at least in part, to exposure to modern feeding regimens and environmental conditions which are vastly dissimilar, or *mismatched* to, the Paleolithic/pre-agricultural diets and environments to which the human genome has been best adapted.[5]

p0020 Pediatric sleep-disordered breathing (SDB) is a pathological condition associated with a wide range of clinical symptoms, historical evidence, dentofacial physical examination findings, environmental components and genetic and/or epigenetic factors. Recently published controlled studies indicate a close association between pediatric SDB/OSA and neurocognitive impairments such as ADD/ADHD and other behavioral disorders.[6,7] Many of the various physical characteristics associated with high prevalences of pediatric SDB/OSA are also strongly associated with a number of pediatric dentofacial abnormalities; the relationship between pediatric SDB/OSA and the developing jaws and facial structures is also well described.[8,9]

p0025 Many craniofacial traits are, for the most part, alterable during the early childhood stages of dentofacial growth and thus likely to play a large role in the presence or absence of clinical symptoms associated with pediatric SDB/OSA and its associated clinical morbidities. Consequently, the role of the orthodontist, pediatric dentist and general dentist as an integral member of every child's comprehensive health care team has never been more important.[10,11]

ETIOLOGY OF MALOCCLUSION

s0015

p0030 Anthropological studies confirm that dentofacial malocclusion (poorly aligned jaws and teeth), a known risk indicator of SDB/OSA,[12] was infrequently suffered by our pre-*industrial* ancestors, and seldom occurs with frequency in extant non-*Westernized* aboriginal cultures.[13] In fact, skeletal malocclusion didn't appear appreciably in humans until around the time of the *Industrial Revolution* of the mid-eighteenth century,[14] and wherever occasionally observed before that era, it was usually confined to privileged-class individuals.[15]

p0035 In order to most efficiently address the health problems known to be associated with untreated, and/or *inappropriately* treated malocclusion, it would first be helpful to have some idea about why our fairly recent ancestors seldom suffered from these unpleasant dentofacial dental and skeletal disharmonies. Anthropologists have understood for decades that human craniofacial volume has been steadily diminishing since around the time of the Agricultural Revolution some 10,000–12 000 years ago, and most rapidly over the past 350–400 years.[14] While there seems to be a definite observable trend towards increased prevalences of malocclusion over the last three to four centuries, to date there is not yet firm consensus amongst dental anthropologists as to precisely *what happened*, but there does seem to be a growing body of evidence that seems to suggest that feeding behaviors during infancy and early childhood are likely involved.[16] Specifically, *ancestral-type* breastfeeding and weaning are known to be protective against certain forms of malocclusion,[17] likely due to the physical challenges posed to the developing palatal–facial suture complex (P–FSC) during infancy and early childhood; furthermore, the highly processed/soft baby foods and artificial infant formulas/commercial nipples that are in so much use today were simply not readily available to children prior to the *Industrial Revolution*.

p0040 With ever accumulating physical evidence from anthropological studies, combined with advances in the newly emerging scientific disciplines of epigenetics and evolutionary medicine, it can be stated with a reasonable degree of scientific certainty that malocclusion is *not* primarily a *genetically determined* disease entity. Rather, malocclusion is better described as a WD that is primarily mediated through a gene–environment interaction that follows a fairly predictable pattern of pathological progression: initially, most WDs are *preventable* so long as genetically predisposed individuals are identified before early phenotypic expression of the disease is obvious, and where feasible, are allowed to thrive in a

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nurturing environment; next, WDs can be *reversible*, but only in the very early stages of disease expression, and only when the precipitating environmental pressures (e.g., unhealthy eating, sleep disordered breathing) have been eliminated; subsequently, in cases where a WD has advanced beyond reversibility, it can be still be *treated* with accurate diagnosis and appropriate therapeutic measures (e.g., dietary changes, pharmaceuticals) if the disease state is not too far advanced; and finally, advanced end-stage WDs can be *fatal* if not accurately identified, reversed and/or appropriately controlled.

p0045 While a cause-and-effect relationship between malocclusion and the pathophysiology of SDB/OSA is not yet proven,[18] a relationship does indeed appear to exist between the two disease entities. Similar to what is now understood about why diabetes and periodontal disease often coexist in the same host,[19] the underlying mechanism connecting SDB/OSA and malocclusion is more likely to be a *bidirectional* one rather than a unilateral cause-and-effect relationship. Simply stated, measures aimed at preventing the initiation and early progression of one disease entity will aid in preventing the initiation and early progression of the other. Given that many dentofacial physical risk indicators for malocclusion might also identify increased risk for SDB/OSA, it seems fairly obvious that measures aimed at prevention, reversal and/or adequate treatment of malocclusion might also help preclude the negative health outcomes that are often associated with SDB/OSA.

s0020 PEDIATRIC ORAL HEALTH AND SLEEP

p0050 Dentists who treat children are uniquely positioned to identify patients who might be at increased risk for SDB/OSA. Due in part to the successful implementation of the American Academy of Pediatrics and American Academy of Pediatric Dentistry's (AAPD) joint effort to assure that all children establish a *dental home* by the age of 1 year,[20] pediatric dentists now have a higher frequency of patient encounters than do most other allied health professionals. Additionally, postgraduate specialty training programs in pediatric dentistry and orthodontics purposefully prepare clinicians for identifying patients with interferences to normal dentofacial development, including children with special health care needs who might be at even higher risk for developing OSA, such as patients diagnosed with Down syndrome, sickle cell anemia, and Pierre-Robin sequence.

p0055 During dental visits, many warning signs that a child might be experiencing sleep disturbances can be ascertained from both a thorough oral–medical health history interview with parents/primary caregivers and a comprehensive clinical dentofacial examination. In order to best assure a comfortable and safe dental visit, questions usually asked during a detailed pediatric oral–medical health history interview are designed not only to obtain information about the child's overall dental/medical health status, but also to acquire information about dietary/feeding history and previous dental and/or medical encounters that might impact a child's possible expectations about the dental appointment. A typical list of questions asked might include, 'Was your child breastfed, and if so, for how long?' and 'What beverage does your child typically drink when thirsty?'

p0060 While not necessarily a component of a *typical* pediatric medical–dental health history, it is certainly easy, useful and appropriate for dentists to incorporate into the parent/caregiver interview a short series of questions specifically designed to gain valuable information about a child's possible risks for both malocclusion and/or SDB/OSA; some examples might include, but not limited to: 'Does your child grind his/her teeth at night?', 'Is your child a noisy open-mouth breather and/or snorer during sleep?', 'Does your child occasionally wet the bed?', 'Does your child ever wake up with either a sore jaw, headache, dry mouth and/or sore legs?', 'Is your child at a healthy weight?', and 'Does your child have night terrors or nightmares?'

p0065 In addition to the detailed medical–dental health history interview, a comprehensive dentofacial clinical examination might yield warning signs that a child might be suffering from impaired ability to breathe properly during sleep.

s0025 SURGICAL VERSUS NON-SURGICAL TREATMENT OPTIONS FOR PEDIATRIC SDB/OSA

p0070 It is well established that surgical removal of the tonsils and/or adenoids is the most common treatment for pediatric OSA. For extremely severe cases of OSA for which adenotonsillar surgery might not be indicated as the best treatment, maxillomandibular advancement surgery (MMA) and/or tracheostemy placement are on occasion considered as better surgical options. According to a recently published guidelines paper by the American Academy of Otolaryngology,[21] craniofacial abnormalities of the maxilla and mandible are definite indications for recommending a PSG sleep study prior to T and A surgery.

p0075 Maxillary constriction (MC) is a common craniofacial abnormality that plays an important role in the bi-directional relationship between malocclusion and OSA;[22] MC is typically characterized by narrow/deep-vaulted palate (Figure 34-1), tapered dental arches and retro-position of the mid face relative to the anterior cranial base. Per the various comorbidities associated with various surgical interventions, wherever feasible, collaborative efforts aimed at preventing and treating pediatric OSA non-surgically should be given the highest consideration.

p0080 Two commonly implemented *non-surgical* medical interventions include inhaled nasal corticosteroids and usage of CPAP/BPAP devices. While correctly classified as a non-surgical treatment options, long-term usage of CPAP/BPAP facial masks can markedly reduce mid-facial development potential in growing children[23] in much the same manner as adult orthognathic surgical reduction procedures such as mandibular setback and anterior segmental maxillary osteotomy. Other common examples of non-surgical prevention and treatment options for pediatric OSA include, myofunctional training oral appliances (e.g., Infant Trainers, Myo-Munchies, etc.), oral myofunctional therapy (OMT)*, conjunctive dietary counseling for overweight and obese OSA patients, functional orthodontic mandibular advancement appliances (e.g., Bionator), rapid maxillary expansion (RME) appliances (e.g., bonded Schwartz Plate, Hyrax, etc.) with, or without, reverse pull maxillary protraction appliances (MPA) (e.g., Delaire facemask) and more recently, Biobloc Orthotropic (BBO)* postural appliances that are capable of non-surgically increasing posterior airway dimensions through sequential advancement of both the mandible *and* maxilla with a series of removable acrylic mouthpieces.

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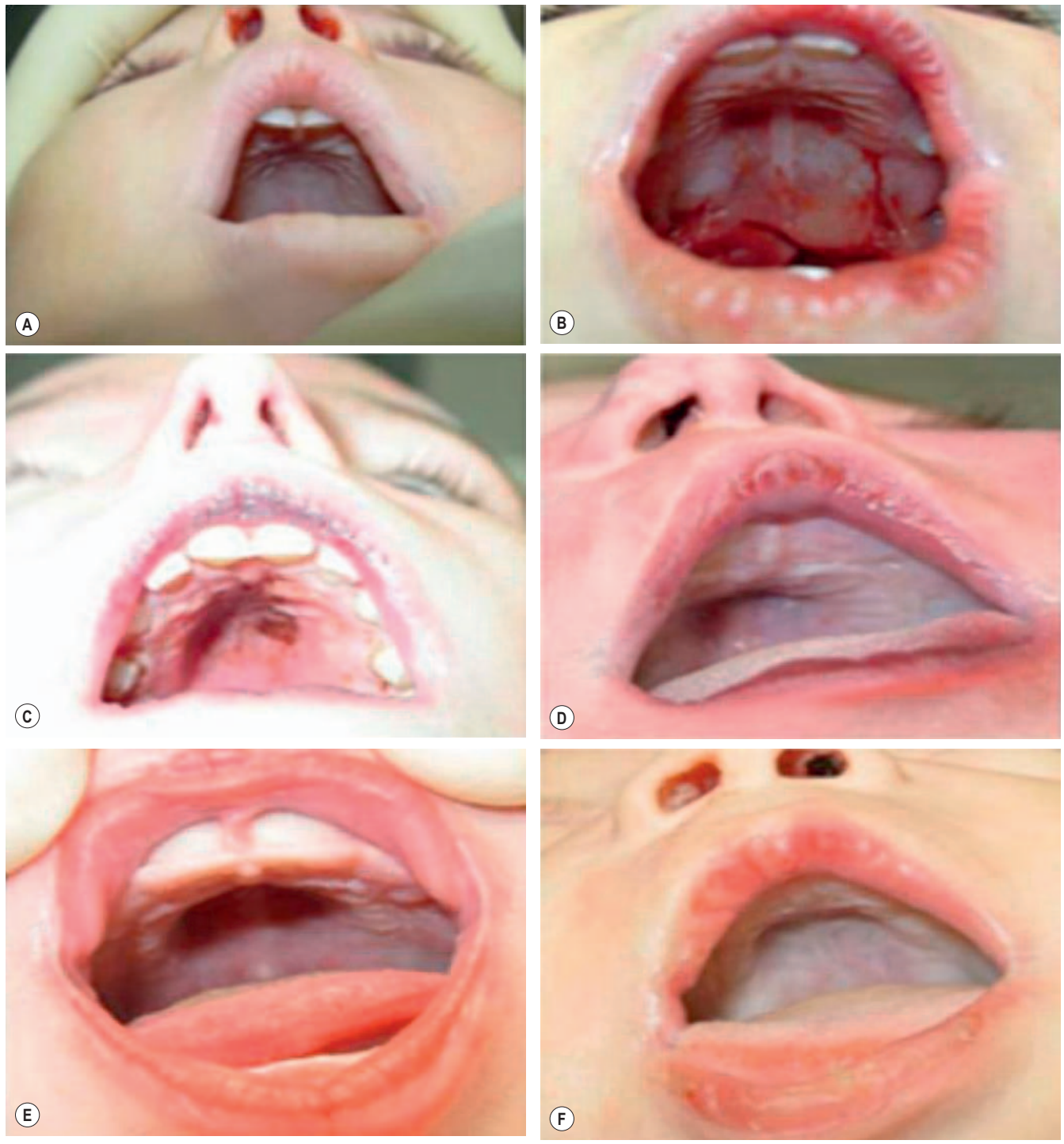

FIGURE 34-1 Montage of abnormal high and narrow hard palate in Cases 1–6. Note that all children present a visually recognizable abnormal high and narrow hard palate which is related to the development of the naso-maxillary complex during embryonic development considering age of children. On the first row on the left, on the second row in both cases, and on the third row from the top on the right, note the abnormal noses presented by the patients. The asymmetry of the nostril may not be obvious at first investigation; using photographs taken below the nose may help performing better analyses. Asymmetrical opening is often associated with asymmetrical septum and change in nasal resistance. When associated with high palatal vault, they indicate presence of a higher upper airway resistance and greater risk of abnormal breathing during sleep with addition of infectious or inflammatory reaction. ***From Rambaud C, Guilleminault C., Death, nasomaxillary complex, and sleep in young children. Eur J Pediatr. 2012 Sep;171(9):1349–58, with permission of Springer-Verlag 2012.***

p0085 Until the recognition of BBO as a non-surgical option for improving posterior airway volume in actively growing children,[24] rapid maxillary expansion (RME) was considered the primary non-surgical orthodontic treatment of choice for treating OSA in children.[25]

Rapid Maxillary Expansion (RME)

s0030

p0090 Rapid maxillary expansion (RME) is well established as an effective non-surgical treatment option for decreasing upper airway resistance through increasing airway volumes within the nasomaxillary complex.[26,27] Depending on the age of the

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patient, by exerting orthopedic forces upon the entire maxillary suture complex, primarily with the use of fixed maxillary expansion appliances, RME orthopedic movement will occur when the relatively light forces applied to the teeth and the maxillary alveolar process eventually exceed the forces required for orthodontic tooth movement alone. Following RME, there is an increase in the transverse width of the nasal cavity and hard palate, most notably at the floor of the nose near the mid-palatal suture. In cases where the narrow maxillary arch is also retrognathic (relative to the cranial base), RME can be assisted by reverse-pull headgear (e.g., Delaire facemask) to provide additional nasal airway volume through increasing the anterior dimension of the nasorespiratory space.[28]

s0035 3 ***Biobloc Orthotropics (BBO) and Oral Myofunctional Therapy (OMT)**

p0095 As a key participant at the 2012 NESCent Catalysis Conference, Professor Robert Corruccini, a dental anthropologist from Southern Illinois University, was recently cited in *Science*:[29] 'As for malocclusion and jaw disorders, Corruccini noted that a branch of evolutionary dentistry has emerged in which children do mouth exercises and wear devices that put stronger force on their growing jaws.' The branch that Professor Corruccini was referring to is called orthotropics. The orthotropic premise was originally developed in England in the late 1950s by Dr. John Mew, a dual-trained oral surgeon–orthodontist, as an alternative to the then and still commonly held belief that malocclusion is primarily a genetically inherited condition; Mew, also a student of anthropology, studied ancient skulls at the Natural History Museum in London where he was further convinced that malocclusion is an environmentally influenced *disopoly* (*disease of civilization*) which had been brought about by factors related to increased industrialization. Specifically, the orthotropic premise implicitly states that improper tongue and head posture will invariably lead to malocclusion and other associated negative systemic health outcomes.

p0100 United by their common focus on assuring optimal dentofacial growth potential and healthy wake–sleep nasorespiratory ability for their young and growing patients, the number of clinicians, mostly orthodontists, pediatric dentists, general dentists, OMTs, sleep medicine physicians and other allied health professionals, who are recommending BBO treatment as a viable non-surgical intervention for SDB/OSA patients is growing rapidly.

p0105 The Biobloc appliance system utilizes a series of acrylic intraoral appliances to first develop the upper jaw (maxilla) and mid face to its optimal width and forward position within the cranial base, after which the mandible is *postured* forward with a subsequent appliance to reunite both jaws to a more forward post-treatment position within the cranial base. This maximally forward jaws–facial position provides not only for better esthetics and facial balance, but is also more conducive to development of increased posterior pharyngeal volume and less nasal airway resistance.

p0110 There are many other more traditional types of orthodontic appliances such as Twin Blocs, Frankels, Bionators, MARA's, Herbst appliances, class II elastics and others, which all attempt to do the same thing, but most of these appliances are often only begun in the late-mixed to early adult dentition and can exert a backward force, or *headgear effect,* on the growing maxilla that can actually worsen esthetic appearance and/or an already compromised airway.

p0115 Biobloc Orthotropics (BBO) differs mainly from conventional orthodontic treatment modalities in that BBO: (1) does not utilize treatment mechanics that place retrusive forces on the jaws, teeth and face; (2) is usually begun in the primary or early mixed dentition when maximum impact upon a child's naso-respiratory competence and neurological, craniofacial and somatic growth is most easily accomplished; and (3) often in conjunction with OMT regimens, is chiefly designed to create a lifelong optimum oral environment for a properly postured and functioning tongue, which is also conducive to lifelong stable and well-aligned adult teeth.

s0040 ***Biobloc as an Alternative to Mandibular Distraction Surgery for Severe OSA: Case Report***

p0120 The case study described below is a good illustration of how BBO can be utilized as a safe non-surgical alternative to mandibular distraction osteogenesis for severe OSAS. Note the improved cervical spine posture and posterior airway volume seen at the end of BBO Tx (Figure 34-2) and the supportive PSG result. At baseline (Ba) (Figure 34-3), the AHI was elevated at 12.4 events per hour of sleep with the majority of events occurring during REM sleep. Over the course of time, the AHI and REM AHI both decreased steadily, to a degree that at point 1 on the graph the AHI was only 3 events per hour of sleep. Interestingly, as treatment continued from baseline and as the AHI fell, there was a concomitant increase in $EtCO_2$ with a peak when the AHI reached only 3 events per hour. Interpretation of these findings was initially difficult since there was numerical improvement in the frequency of occlusive and partially occlusive respiratory events, but apparent worsening of gas exchange exemplified by increasing obstructive hypoventilation. Etiology of elevation of CO_2 and presence of obstructive hypoventilation related to presumptive development of extremely prolonged partially occlusive respiratory events. For example, if each hypopnea lasted 20 minutes and they were continuously periodic, the AHI will be only 3 events per hour of sleep despite persistence of partial upper airway obstruction. At point 2 at the end of the graph, AHI continued to fall to its nadir of 0.2 events per hour of sleep with concurrent decrease in CO_2 levels to normal and resolution of obstructive hypoventilation. At the same time, there was clear clinical evidence of improvement in symptoms with absence of snoring, resolution of restless sleep, and resolution of daytime sleepiness. At this point, based on both clinical resolution and polysomnographic evidence, sleep-disordered breathing had resolved. Continued follow-up is still warranted and required in order to assure resolution. One final polysomnogram will be conducted to provide objective evidence.

SUMMARY AND FUTURE CONSIDERATIONS s0045

p0125 1. OSA is a chronic respiratory disorder that is clearly linked to retrognathic skeletal malocclusions that force the hyoid bone, base of the tongue and its supporting musculature too close to the posterior pharynx. Given what anthropologists have shown with regard to shrinking human craniofacial volume over the past 300 years or so, currently accepted cephalometric normative values are not completely reflective of our true genomic craniofacial growth potential. As a result of this disparity, many clinicians are

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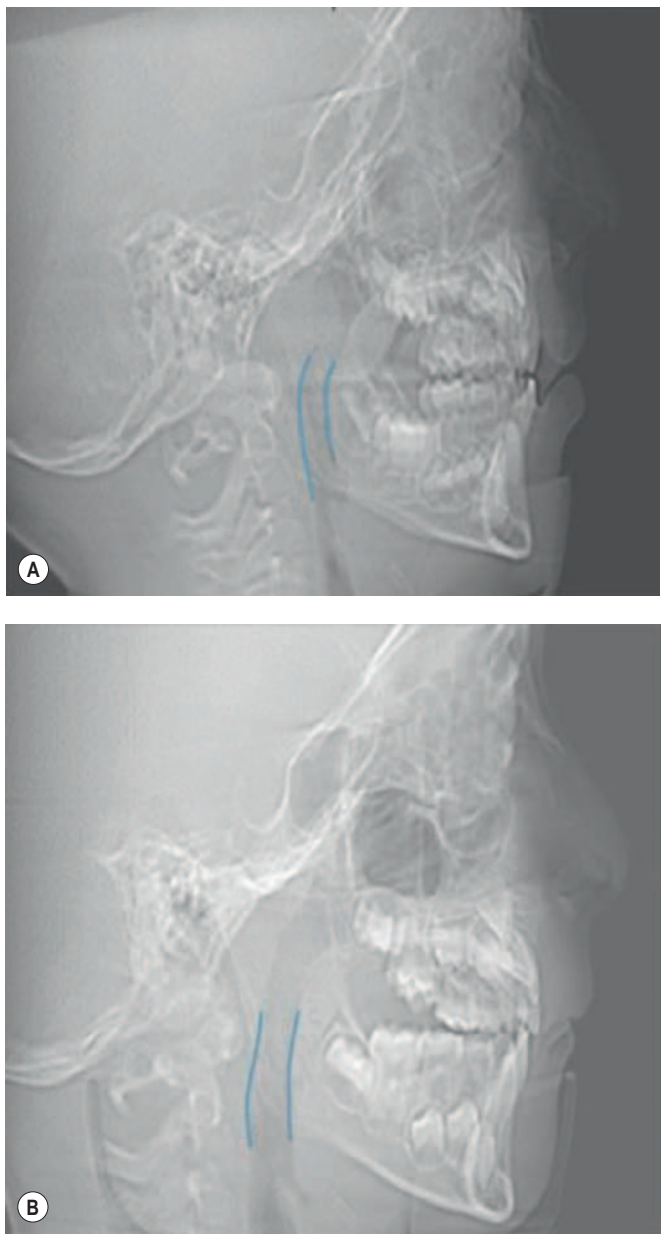

FIGURE 34-2 Note differences in cervical spine erectness and posterior airway area in pre-BBO Tx image (left) vs. post-BBO Tx image (right).

currently being trained to diagnose, treat and evaluate orthodontic treatment progress and final outcomes in accordance with cephalometric norms that are *anthropologically uniformed*, especially with regard to the baseline assessment of maxillary position relative to the anterior cranial base; this is potentially dangerous as this error can sometimes lead to diagnostic and treatment failures which in turn can have negative overall health implications related to inadequate posterior airway volume; e.g., cervical-pull headgear treatment and incisor retraction exacerbating compromised posterior airway volume in class II retrognathic patients.[30,31] Cooperative efforts between anthropologists, dentists and other concerned health care professionals should be undertaken to revise currently used cephalometric standards so as to better reflect the true forward growth potential of the human dentofacial complex.

2. Medical and dental educational programs should incorporate more cross-curriculum activities and include evidence-based content into their teaching curriculums within the disciplines of evolutionary medicine, sleep medicine, orofacial myology and nutrition. o0015
3. Overconsumption of sugar and other refined (fermentable) carbohydrates are clearly implicated in recent increases in nationwide prevalences of both early childhood caries (ECC)[32] and childhood obesity.[33,34] Given that childhood obesity[35] and pain associated with untreated caries[36] are both known risk factors associated with fragmented sleep, it seems reasonable to suggest that medical and dental professionals should implement diet counseling as an adjunctive component to their existing preventive and therapeutic treatment protocols. o0020
4. Orthodontists, pediatric dentists and general dentists should collaborate with efforts to raise awareness, amongst themselves and their patients, about the importance of early recognition of pediatric patients who might be at risk for SDB/OSA. o0025
5. Guidelines for identifying SDB/OSA dentofacial risk indicators (Table 1) should be established and disseminated to all members of the allied pediatric health care team. o0030

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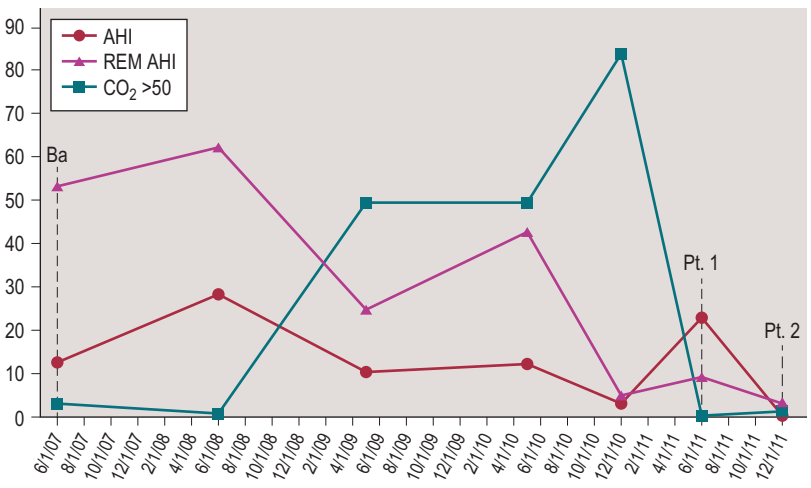

FIGURE 34-3 Longitudinal polysomnograph assessment comparing the patient's AHI, REM AHI, and CO_2 levels as BBO treatment progresses.

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o0035 6. Future well-designed prospective trials will be necessary in order to validate existing scientific and circumstantial evidence that early childhood feeding environments, dentofacial development, naso-respiratory competence, pediatric sleep hygiene and neuro-cognative development, are all inter-related.

o0040 7. When indicated by the presence of multiple medical and/or dentofacial risk indicators for SDB/OSA, orthodontists, pediatric dentists and general dentists will need to be properly trained to deliver effective non-surgical modes of oral interventions, and be willing and able to intervene or refer for appropriate screening (e.g., PSG) and/or treatment while children are still in their primary dentitions.

o0045 8. Pediatric sleep medicine centers should have at least one dentist on their team who is experienced in dentofacial issues related to SDB/OSA.

o0050 9. New methods for earlier detection of children at risk for SDB/OSA, such as in utero 3-D ultra-sonography facial imaging, should continually be explored.

b0010 **Clinical Pearls**

p0175 • An increased emphasis on identifying malocclusion as more of a *symptom* rather than as a distinct *disease* entity can help medical and dental clinicians better understand and appreciate the inter-relatedness between malocclusion and SDB/OSA. Evaluating SDB/OSA and malocclusion from an evolutionary perspective helps clarify that, similar to other *Western diseases* such as type 2 diabetes, susceptible individuals need not fully express the disease phenotype if environmental triggers are identified and eliminated early in a child's life.

u0060 • The ability to recognize known dentofacial risk indicators of SDB/OSA in early childhood can help dentists, physicians and other allied health professionals better collaborate in providing comprehensive and coordinated care for their mutual patients.

u0065 • Both childhood obesity (CO) and pain from untreated early childhood caries (ECC) are known risk factors for SDB/OSA that can negatively impact sleep quality and quantity. As overconsumption of commercially processed fermentable carbohydrates (f-CHOs) in early childhood is a known etiological component of both CO and ECC, dentists and physicians should discourage unhealthy snacking on simple sugars and starches as a component of their CO, ECC and SDB/OSA prevention and treatment protocols.

u0070 • As breastfeeding is known to be protective against the development of SDB/OSA, adenotonsillar hypertrophy (ATH) and malocclusion, dentists and physicians should mutually provide consistent and accurate advice to parents regarding options for infant and early childhood feeding regimens.

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