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1Deceased
1 Introduction

Sleep-disordered breathing (SDB) comprises a wide spectrum of sleep-related breathing abnormalities; those related to increased upper airway resistance include snoring, upper airway resistance syndrome (UARS), and obstructive sleep apnea/hypopnea syndrome (OSAHS).1

First identified in 1976, Christian Guilleminault diagnosed children with a sleep apnea syndrome similar to that seen in adults by means of polysomnography. He found that excessive daytime sleepiness, a decrease in school performance, abnormal daytime behavior, recurrent enuresis, morning headaches, abnormal weight, and progressive development of hypertension suggested the possibility of a sleep apnea syndrome when any of these symptoms is associated with loud snoring interrupted by pauses during sleep.2

Primary snoring (PS) has been considered the most benign form of SDB, and treatment in the past has not usually been prescribed. Current studies suggest that PS may not be as benign as had formerly been considered. A significant proportion of children with PS persist with SDB symptoms even 5 years after the diagnosis.3

Children with non-hypoxic, non-apneic PS may exhibit significant neurocognitive and/or neurobehavioral impairments (Bonuck 2012). Consequences may be similar to those associated with UARS or obstructive sleep apnea (OSA). Compared with children who had never snored, children with PS had more hyperactive (39% vs. 20%) and inattentive behavior (33% vs. 11%), as well as poor school performance in mathematics (29% vs. 16%), science (23% vs. 12%), and spelling (33% vs. 20%; all P values <0.5). PS was a significant risk factor for hyperactive behavior and inattentive behavior, as well as daytime sleepiness. PS was also an independent risk factor for poor school performance in mathematics, science, and spelling.

Along with daytime impairments, snoring can be associated with sleep problems. An increasing prevalence of sleep problems was found with increasing snoring frequency for sleep-onset delay, night awakenings, and nightmares.
craniofacial features will be developed by 4 years of age, and 90% by around 12 years of age.7

OSA in children has emerged not only as a highly prevalent condition, but also as a disease that can impose a large array of comorbidities, some of which may have long-term implications extending past adolescence and well into mid/late adulthood. Major deleterious sequelae of pediatric OSA can include neurologic issues, cardiovascular and endocrine disease, and dysfunction of other metabolic systems such as appetite dysregulation and risks associated with excessive body weight (e.g. SDB/OSA, type 2 diabetes, etc.). The underlying pathophysiologic mechanisms of OSA-induced end-organ injury are now being unraveled and clearly involve oxidative and inflammatory pathways. However, the roles of individual susceptibility, such as might be dictated by single-nucleotide polymorphisms, and of environmental and lifestyle conditions, such as diet, physical, and intellectual activity, may account for a substantial component of the variance in phenotype.8

2 Evolutionary Considerations

2.1 Cultural Industrialization and Genomic-Environmental Mismatch

The relatively recent appearance of chronic Western diseases, or so-called noncommunicable diseases (NCDs) in humans, such as obesity, type 2 diabetes, cardiovascular disease, etc., is not accurately explained as somehow resulting from recent, and therefore anomalous, macro-genomic change over the past few centuries when prevalence of these health problems has grown to near-epidemic proportions, most notably in many industrialized societies. A more plausible explanation is likely to be found when evaluating the problem from an evolutionary perspective, that is, when critically evaluating why natural selection, the Darwin-proposed force driving either adaptive change or extinction,9 has apparently left contemporary humans vulnerable to NCDs. An argument can be made that human malocclusion, the term most often used to describe underdevelopment of various craniofacial structural components, specifically of the jaws, facial bones, and associated crowding and misalignment of teeth, is yet another NCD that also has only recently reached near-epidemic proportions worldwide and afflicts people of all ages, but seldom appears in extant aboriginal populations until after exposure to modern industrialized influences. It is now clearly understood that most forms of craniofacial maldevelopment can often be bidirectionally associated with under-development of upper respiratory anatomic structures, or simply stated, genetic and/or environmental factors affecting growth and development of the masticatory apparatus can also affect growth and development of the respiratory apparatus, and vice versa. For example, Harvold10 showed that under experimental conditions, impairing naso-respiratory competence in monkeys resulted in structural malformations of their mandibles, maxillas, and teeth alignment; in contrast to this, a postmortem analysis of several infants who had apparently died of respiratory complications showed that all were found to have retrognathic mandibles and high and narrow (ovigal) hard palates.

For the purpose of using terminology that better represents the intimate connection between craniofacial and respiratory structural anatomy, the term craniofacial-respiratory (CFR) would seem to be more accurate than is craniofacial alone. Accordingly, the term craniofacial anomalies, a commonly used term within the scientific literature when describing SDB/OSA-associated malocclusion traits in syndromic individuals (e.g. retro-positioned mandibles, mid-face deficiency, vertical jaw growth, etc.), can be substituted with CFR maldevelopments as a more encompassing descriptor, in that it better represents SDB-OSA malocclusion phenotypes that commonly occur with high frequency in both syndromic and nonsyndromic individuals.

As previously stated, malocclusion is a condition that had been nearly non-existent throughout the some 200,000+ years of anatomically modern human history but is now, according to Proffit,11 more highly prevalent than it was even a few hundred years ago. Larsen12 reports that as humans veered from the hunter-gatherer model toward a transitional shift to more intensified agricultural sustenance strategies, there was also a gradual increase in dental crowding, which seems to become more severe with cultural industrialization. Observations from Gilbert13 reveal that "jaw anomalies" (i.e. CFR maldevelopments) are relatively new to European populations, as skeletons from the 15th and 16th centuries show almost no malocclusion. Lieberman14 reports "there is much circumstantial evidence that jaws and faces do not grow to the same size that they used to."

Given that high prevalence of pediatric SDB/OSA and malocclusion are both relatively recent public health dilemmas that are often comorbid, it seems useful to posit a possible connection between these two maladies. Many children with SDB, often but not always, additionally have one or some combination of the following malocclusion traits: most notably, angle class II skeletal malocclusion with associated retro-positioned mandible, retrorotgnathic maxilla, narrow dental arches/dental crowding, steep mandibular plane angle/hyperdivergent craniofacial growth pattern, anterior open bite, anterior and posterior cross-bites, excessive dental attrition, excessive dental overjet, excessive dental overbite, and ogival (high and narrow-vaulted) hard palate.

As many modern NCDs are now better understood when viewed from an evolutionary perspective, that is, through learning and applying fundamental tools from the basic life sciences of evolutionary biology and anthropology, health care professionals can become adept at a unique approach to solving medical dilemmas in much the same manner as would a forensic anthropologist help solve a crime scene. Evolutionary medicine (EM), also known as Darwinian medicine, is a novel approach to understanding modern NCD pandemics; in fact, Randolph Nesse, considered to be one of the pioneering forces behind the EM educational framework, has often openly discussed the relative absurdity associated with ignorance in this most basic of life sciences.

Evolutionary oral medicine (EOM) is an application of the EM framework whose goals are to understand specific non-communicable oral diseases within an evolutionary context, most notably, the two plaque-mediated dental problems of early periodontal disease (gingivitis) and dental caries (cavities), and also skeletal-dental malocclusion. In much the same manner as exists with the EM model, EOM aims to use this understanding to develop diagnostic, preventive, clinical treatment and research strategies. The etiology of malocclusion is best understood in accordance with Nesse,
Williams, and others, who proposed the mismatch hypothesis for explaining modern human disease vulnerability. This hypothesis posits that the current high prevalence of certain NCDs, such as malocclusion in industrialized populations, is due at least in part to exposure to modern feeding regimens mostly consisting of softer processed and cooked foods and other environmental conditions that are vastly dissimilar or mismatched to an anciently derived genome that had been best adapted to prolonged chewing of minimally processed and seldom-cooked firm foods of the Paleolithic/pre-Paleolithic eras. A general lack of masticatory challenge from infancy and early childhood to a masticatory apparatus that had evolved to be constantly challenged over a lifespan is why many anthropologists and many other anthropologically informed dental and medical health professionals speculate that the relatively weak pressures posed by bottle feeding, weaning with soft purées, and later lifetime consumption of highly processed foods seems to offer a sound explanation for why worldwide prevalence of malocclusion, and associated airway problems, have only recently spiked in Western-exposed cultures.

### 2.2 Pathophysiology of NCDs

It is apparent that most NCD health disparities (i.e. mismatch phenotypes) apparently follow a distinguishable continuum of progression of pathophysiology:

1. They are often preventable; that is, the disease phenotype will actually never be expressed if a genetically susceptible individual can be identified before becoming symptomatic and somehow remain unexposed to harmful environmental triggers. For example, if the gene sequence by which an individual can be rendered susceptible to the disorder of lactose intolerance is identifiable before showing symptoms, the person can remain free of the intolerance for as long as dietary intake of lactose is completely avoided.

2. These conditions are sometimes reversible if a symptomatic person is identified very early in the disease state; for example, if a potential type 2 diabetic is still in the early stages of pre-diabetic insulin resistance/glucose intolerance, specific lifestyle changes in diet, physical activity, and improved sleep-airway hygiene can actually reverse an impaired glycemic-control phenotype back to a more healthy state.

3. These conditions can be controllable if the disease state has progressed beyond reversibility but can still be managed with appropriate intervention modalities; for example, if a maldeveloped CFR complex in early childhood is allowed to become progressively worsened beyond adolescence when CFR growth has ceased, associated esthetic issues (unattractive smile) and functional problems (impaired naso-respiratory competence) can be addressed respectively with orthodontic tooth alignment or continuous positive airway pressure (CPAP) with or without mandibular advancement devices.

4. When not reversed or adequately controlled with treatment intervention, NCDs can potentially result in increased morbidity and mortality; for example, if an individual suffering from any of the aforementioned lifestyle-modulated NCDs fails to follow a prescribed course of evidence-based therapeutic intervention, their health span and lifespan will become markedly reduced.

### 2.3 CFR Maldevelopment and SDB/OSA Intervention Strategies

If one accepts the premise that two highly prevalent health care problems, SDB/OSA and malocclusion, can often be accurately categorized as NCDs and thus will follow a pattern of pathophysiology that indicates possible preventability, reversibility, treatability, and disability, it stands to reason that health care professionals from all pertinent disciplines should collaborate in helping at-risk individuals get identified as early in life as is feasible. Specifically, because there is now ample published evidence in the peer-reviewed scientific literature to suggest a bidirectional pathophysiologic relationship between SDB/OSA and identifiable CFR maldevelopments (i.e. malocclusion), it becomes imperative that collaboration between allied medical and dental professionals begin earlier in childhood than is current practice. Specifically, according to a promotional pamphlet from the American Association of Orthodontists (AAO), the AAO recommends that a child's initial orthodontic screening should be on or before the age of 7 (when approximately two-thirds of CFR growth and development are already completed); however, they also state that most orthodontic treatment does not usually begin until about age 11 (i.e. when the majority of CFR growth and development have already been completed). In light of what is now well understood about how retractive and constricted CFR phenotypes are co-morbid with pediatric SDB/OSA and are also often first detectable in the primary dentition, and usually persistent and worsen in later childhood and beyond in the absence of appropriately timed and applied evidence-based intervention, it becomes apparent that the AAO should seriously consider revising this message to the public (Fig. 74.1).

### 2.4 Nonretractive Orthodontics

An effective strategy for treating CFR maldevelopments in the primary or early mixed dentition involves placement of a fixed or removable RPE as long as the second primary molars are erupted, usually by the age of 30 months old; before eruption of the second molars, orofacial myofunctional therapy, tongue training devices (e.g. Myo-munchies, Infant Trainers, Fresh Start, etc.), and/or mastication of firm foods can be utilized to improve lip and tongue posture. Maxillary protraction with a reverse-pull face mask, when indicated per maxillary sagittal insufficiency, can also be implemented due to its ability to create additional anterior tongue space and to move the posterior hard palate away from the posterior nasopharyngeal airway space. Also, mandibular transverse development with or without orthopedic mandibular advancement can improve deficient CFR-strucural components when indicated per persistent SDB/OSA symptoms and/or hypopharyngeal constriction (on CBCT). This protocol can be implemented either before or after T and/or A surgery as an adjunctive intervention per its ability to non-surgically enlarge pharyngeal corridors.

A likely obstacle for most orthodontic providers to treating children in the primary/early mixed dentition lies in their lack of confidence and experience in managing normal/
age-appropriate childhood anxiety in clinical settings. The American Dental Association's (ADA's) pediatric dentistry post-graduate curriculum requires that trainees spend a substantial portion of a 2- or 3-year post-graduate program learning clinical orthodontics/dentofacial orthopedics, cephalometric measures, and growth and development; yet the ADA's orthodontic curriculum has no requirement for residents to spend a single minute in the pediatric dentistry department for the purpose of learning how to appropriately manage child anxiety. If this particular void in the ADA orthodontic curriculum is allowed to persist, many children under the age of 7 will continue to be underserved by the orthodontic profession.

3 Summary and Conclusions

Specific CFR phenotypes are common findings among children raised in industrialized societies. However, these particular CFR traits are a rare finding in the pre-industrial skeletal and fossil records and are seldom seen in extant (modern-day) aboriginal societies who have not yet been Westernized. Certain CFR phenotypes known to be co-morbid with, if not causally related to, development of SDB/OSA are usually first detectable in very early childhood (primary dentition), and recent evidence suggests that they might even be detectable in utero (image). Changed dietary regimens associated with cultural industrialization during infancy/early childhood and beyond seem to have played a role in the observed increased prevalence of CFR maldevelopments since the Industrial Revolution in Western Europe and North America from the late 18th to the mid/late 19th centuries.

An argument can be made to support speculation that:
1. Specific CFR maldevelopments, now pandemic in industrialized cultures, is a relatively recent phenomenon that is associated with changed cradle-to-grave dietary strategies that are characteristic of cultural industrialization.
2. Antecedents to CFR maldevelopments can be initially detected in early childhood and possibly even during fetal development.
3. Early/very early detection of certain CFR maldeveloped phenotypes, and appropriately timed and applied dento-facial orthopedic intervention, can decrease risk for developing co-morbidities associated with pediatric SDB/OSA, such as attention deficit disorder/attention deficit-hyperactivity disorder (ADD/ADHD), and other airway-related systemic disorders.

4 Enuresis

Nocturnal enuresis (NE) is involuntary urination during night sleep; nocturia is the term for voluntary nighttime urination, and although known to be associated with apnea in adults, nocturia in children is uncommon. There is, however, evidence to suggest that NE in childhood might predispose to later nocturia in adulthood. The pathogenesis of NE is controversial. Developmental delay, genetic factors, stress and psychological factors, and sleep abnormalities are considered the etiologic factors. Various urodynamic studies showed bladder hyperactivity in enuretic children.11

It has been found that children referred for polysomnography with snoring but without concurrent NE were less likely to have moderate-to-severe OSA compared with snorers with NE.12 Children with NE are believed to have deep sleep with high arousal threshold. Studies suggest that OSAHS and NE are common problems during childhood. Patients with refractory NE had a significantly higher prevalence of OSAHS with no sex difference. The frequency of bedwetting was higher in patients with severe OSAHS.13

Although nocturnal voiding is frequently attributed to urologic disorders, nocturia and enuresis are important symptoms of SDB. The nocturnal polyuria of sleep apnea is an evoked response to conditions of negative intra-thoracic pressure due to inspiratory effort posed against a closed airway. Treatment of sleep apnea and airway compromise has been shown to reverse nocturnal polyuria and thereby reduce or eliminate nocturia and enuresis.14

Enuretic children are also found to have a higher-than-expected Apnea/Hypopnea Index (AHI), due to a high frequency of hypopneas. They were also noted to have a tendency for respiratory arousals. These children were found to have subclinical signs of disordered respiration, which may be one of the explanations for their high arousal thresholds.

Autonomic dysfunction is another one of the proposed mechanisms for NE in children. Study results suggest that sympathetic nervous system hyperactivity is present in enuretic children, and may explain why some enuretic children do not respond to anticholinergic medications.

5 Periodic Limb Movement Disorder

Restless leg syndrome (RLS), a common neurologic sleep disorder, occurs in 5% to 10% of adults in the United States
and Western Europe. Looking at the prevalence of RLS in children and adolescents, population-based data suggest that RLS is occurring more commonly than epilepsy or diabetes.

Also, children in the periodic limb movement disorder (PLMD)/ADHD group were more likely to have PLMs than were children with PLMD only. It is postulated that rather than a direct relationship between ADHD and PLMD, this link may be mediated by the presence of reduced rapid eye movement sleep and, more importantly, by the sleep fragmentation associated with PLM-induced arousals.15

RLS may have a behavioral association as well. Because PLMD and/or RLS can cause sleep disruption, we assessed whether these two specific sleep disorders are likely to occur in children with ADHD. Sleep disruption associated with PLMD and RLS and the motor restlessness of RLS while awake could contribute to the inattentiveness and hyperactivity seen in a subgroup of ADHD-diagnosed children.

6 Autonomics and Heart Rate Variability

Obstructive sleep apnea syndrome (OSAS) is associated with cardiovascular morbidity and mortality, and increased sympathetic activity is considered to be a causative link. Higher levels of sympathetic activity have been reported in children with OSAS. Sympathetic predominance is indicated on heart rate variability (HRV) analysis by increased heart rate (HR) and a higher ratio of low-frequency to high-frequency band power (LF/HF). Improvement in OSAS after adenotonsillectomy (AT) in children with OSAS could therefore be associated with reduced HR and reduced LF/HF. The proportion of sympathetic activity of the autonomic nervous system (ANS) declines in children with OSAS after AT in association with improvement in SDB.16

The activity of the ANS during sleep in children with OSA was assessed, as was HRV, to detect a possible cardiac ANS imbalance. A time domain index (R-apnea index) was developed to evaluate HRV strictly related to obstructive events during sleep. Poincaré plot of RR intervals during the whole night was calculated. Our findings suggest an autonomic impairment in children with OSA evidenced by the altered HRV, both in the very short term (R-apnea index) and in the short term (SD1). This is significant because the R-apnea index is an easy and cheap method to delay early ANS imbalance (Fig. 74.2).17

7 MRD

When looking at oral appliances (OAs) for SDB, more than 100 designs are available on the market, which differ in terms of the fabrication material, location of the coupling mechanism, titration capability, degree of customization, amount of vertical opening, and lateral jaw movement. Most of OAs cover the upper and lower teeth and hold the mandible in an advanced position with respect to the resting position. These appliances are further divided into titratable and non-titratable OAs based on the capability of the “dose-dependent” effect of mandibular protrusion. An OA is probably more conservative and predictable, as it has a similar mechanism as an orthodontic device for developing children. The treatment procedure also requires a sleep study in addition to the same baseline records (cephalometric and panoramic x-rays, study models, intra-/extra-oral photos) as used in traditional orthodontic patients. Although infrequently used in children, in two 6-month trials on children with OSA treated with two types of OA, significant improvements were noted.18

OAs are indicated for use in adult patients with mild to moderate OSA who prefer them to CPAP therapy or who do not respond to, are not appropriate candidates for, or who fail treatment attempts with CPAP.19

It has been demonstrated that mandibular advancement device (MAD) therapy improves the sleep assessment parameters in patients with mild to moderate OSA and reduces subjective symptoms and excessive daytime

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**FIG. 74.2** Serial images showing persistence of mandibular retrognathia in a non-syndromic child born to a healthy mother. (A) In utero at gestational age of 21 weeks/1 day. (B) Postnatally at 25 months. (C) Beginning of nonretractive orthopedic (NRO) treatment at 4 years old.
sleepiness.10-13 One study demonstrated significant improvements in cognitive and psychomotor performance, particularly in the domain of perceptive abilities, convergent thinking (constructing and solving simple mathematical tasks) and psychomotor reaction times, excessive daytime sleepiness, and quality of life in patients with mild to moderate OSA after MAD therapy.20

Multiple epidemiologic studies have shown a strong link between systemic inflammation and OSAHS. Repetitive hypoxemic stress from apneic and/or hypopneic episodes results in sympathetic activation, which causes persistent hypertension and increased levels of systemic inflammatory mediators, including intercellular adhesion molecules, coagulation factors, and C-reactive protein (CRP). Treatment with custom-made MADs significantly reduced elevated CRP levels in patients with mild to severe OSAHS. Therapy achieves reasonable response and cure rates in the observed patients with a significant reduction in AHI.21

Even though CPAP proved to be more effective at attenuating OSA, better compliance with MAD favored the reduction of one of the enzymes that participates in oxidative stress and better autonomic modulation during sleep. The main finding of this study was that MAD influenced blood pressure, oxidative stress, and HRV. The results suggest that although CPAP was a more effective treatment for OSA patients, MAD showed greater compliance, reduced catalase activity, and improved autonomic modulation during sleep. Even though CPAP proved to be more effective at attenuating OSA and its subjective associated complaints, better compliance was observed with MAD in this group of patients and was more efficient and favored autonomic modulation during sleep and the reduction of one of the enzymes that participates in reactive oxygen species removal.22

Treatment with OAs can be an effective alternative for mild and moderate-to-severe OSAS but requires strict monitoring due to differences in individual response to this therapy.23

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