Assessment and Therapies for Sleep and Sleep-Related Breathing Disorders Associated with Atopic Disease in Children: A Dental Perspective

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In 1992 the American Academy of Pediatrics (AAP) published a policy statement on their concept of the *medical home* in an effort to assure that all infants, children, and adolescents ideally should have accessible, coordinated, continuous, comprehensive, compassionate, and family-centered care [1]. Among the services that were listed as being important for qualifying a healthcare facility as a *medical home* included, but were not limited to, provision of preventive care, growth and development assessments, appropriate screening, healthcare supervision, patient and parental counseling about health and psychosocial issues, identification of the need for subspecialty consultation and referrals, and knowing from whom and where the referrals can be obtained. Following the AAP's example, in 2002 the American Academy of Pediatric Dentistry (AAPD) published their own version of the medical home concept in a landmark paper, *The dental home: A primary care oral health concept* [2]. This paper described that the essential concept of dental care for children of all ages is best managed when there is an established relationship between a practitioner who is familiar with the child and the child's family, which should also be provided for pediatric dental patients by or before the age of 1 year. In fact, after the age of 2 or 3 years, dentists actually have greater frequency of patient encounters with children than do primary care medical providers [3]. This provides opportunity to discuss with parents/adult caregivers *traditional* (cavities, gum disease, etc.) and non-*traditional* oral health issues, such as how morphology of the craniofacial-respiratory complex (CFRC) (Fig. 30.1) can be comorbid with certain atopic disease phenotypes. Dentists might also discuss how atopy might be indicative of underlying breathing hygiene problems during wakefulness and sleep and/or predictive of possible future susceptibility to systemic issues associated with comorbid malocclusion (*maxilla-mandibular compromise*) and nasal disuse (chronic mouth breathing). They might also

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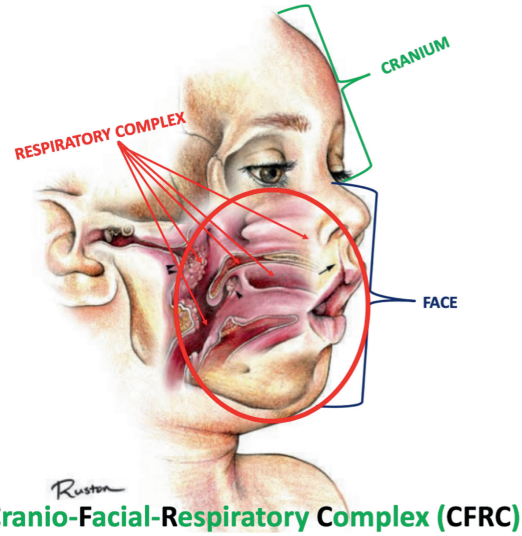

Fig. 30.1 The craniofacial-respiratory complex. (From Garg et al. [33], with permission)

consider making recommendations to seek further consultation from an appropriately trained and qualified healthcare professional, such as a pediatrician, otolaryngologist, allergist, orofacial myofunctional therapist or sleep medicine specialist.

Malocclusion as a Comorbidity of Atopic Disease

Human skeletal malocclusion (HSM), defined as the narrowing within the coronal plane, retracting within the sagittal plane, and lengthening of the horizontal plane of the mandible and maxilla (the maxillo-mandibular complex), with or without associated crowding and misalignment of teeth, is a condition that is prevalent in industrialized societies when compared to nonindustrialized cultures [4, 5]. This is usually first detectable before a child starts to shed their deciduous dentition (baby teeth), by approximately age 6–7 years. Some SM phenotypes known to be associated with atopic diseases of childhood [6, 7] can be diagnosed and appropriately intervened upon during school-age/preschool-age years. In addition to their known association with atopic diseases of childhood, pediatric SM correction, specifically with rapid maxillary expansion, is often associated with symptom mitigation of many non-atopic diseases as well, such as chronic otitis media, conductive hearing loss, and sleep-related breathing disorders (SRDB) [8]. Accordingly, it is important for dental and other healthcare professionals who provide services for children to be knowledgeable about screening for *all* types of SRDB comorbidities and risk factors, including not only physical(CFRC) and behavioral traits, but also pre-natal factors such as exposure to ethanol, first-/second- hand tobacco smoke and maternal (gestational) apnea.

HSM phenotypes most commonly associated with atopic diseases of childhood are unilateral posterior crossbite [7], higher gonial angles, shorter and retrusive

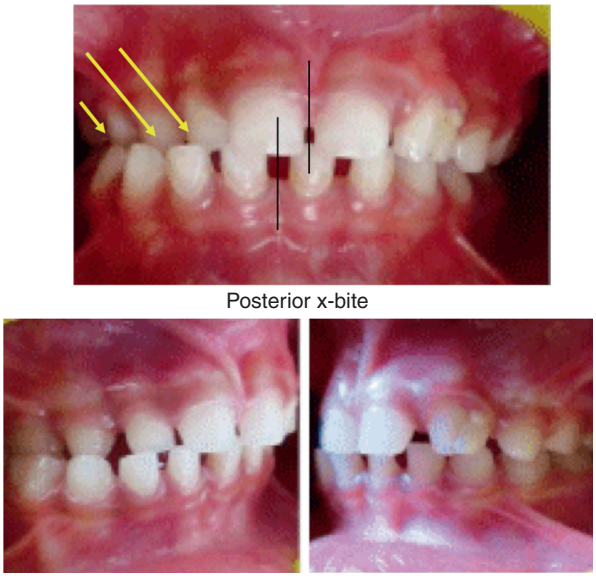

Fig. 30.2 Posterior x-bite

mandible and maxilla [9, 10], anterior open bite [11], vertical skeletal growth pattern (long face), higher palates, narrower maxillary molar widths, and excessive overjets and narrow pharyngeal airway space [6]. *Unilateral posterior crossbites* (Fig. 30.2) are characterized by an asymmetric shifting of a transversly constricted mandible (lower jaw) toward one particularly favored side of the opposing maxilla (upper jaw). They are usually first detectable in the primary dentition (baby teeth, under the age of 6). If left untreated after initial detection, they can precipitate to more serious skeletal involvement which can lead to noticeable facial asymmetries in adolescence and beyond. This in turn can lead to permanent mandibular asymmetry and structural changes within the temporomandibular joint (TMJ) complex [12]. Similarly, Class II SM which is most often characterized by mandibular retrognathia (Fig. 30.3) can also, albeit less frequently, involve coincident retrusion of the maxilla. Class III SM, most often characterized by maxillary retrognathia (Fig. 30.4), is usually first detectable during the earlier stages of dentofacial development (i.e, the deciduous and early mixed dentition). They are often left untreated until a child has shed most of their primary teeth somewhere between the age of 9 and 14, at which time most orthodontists recommend beginning orthodontic treatment (braces) [14]. We hypothesize this delay in SM diagnosis and timliness of intervention might result in a child becoming symptomatic from various atopic and nonatopic systemic health CNCD comorbidities such as SDB/OSA, ADHD, and other CNCDs due to inadequate pharyngeal airway space and mouth breathing/nasal disuse. *Vertical skeletal growth* (Fig. 30.5) pattern (long face) is characterized by lengthening of the lower face as the mandible grows downward and away from the cranial base and, again, similar to posterior crossbites and Class II retrognathia,

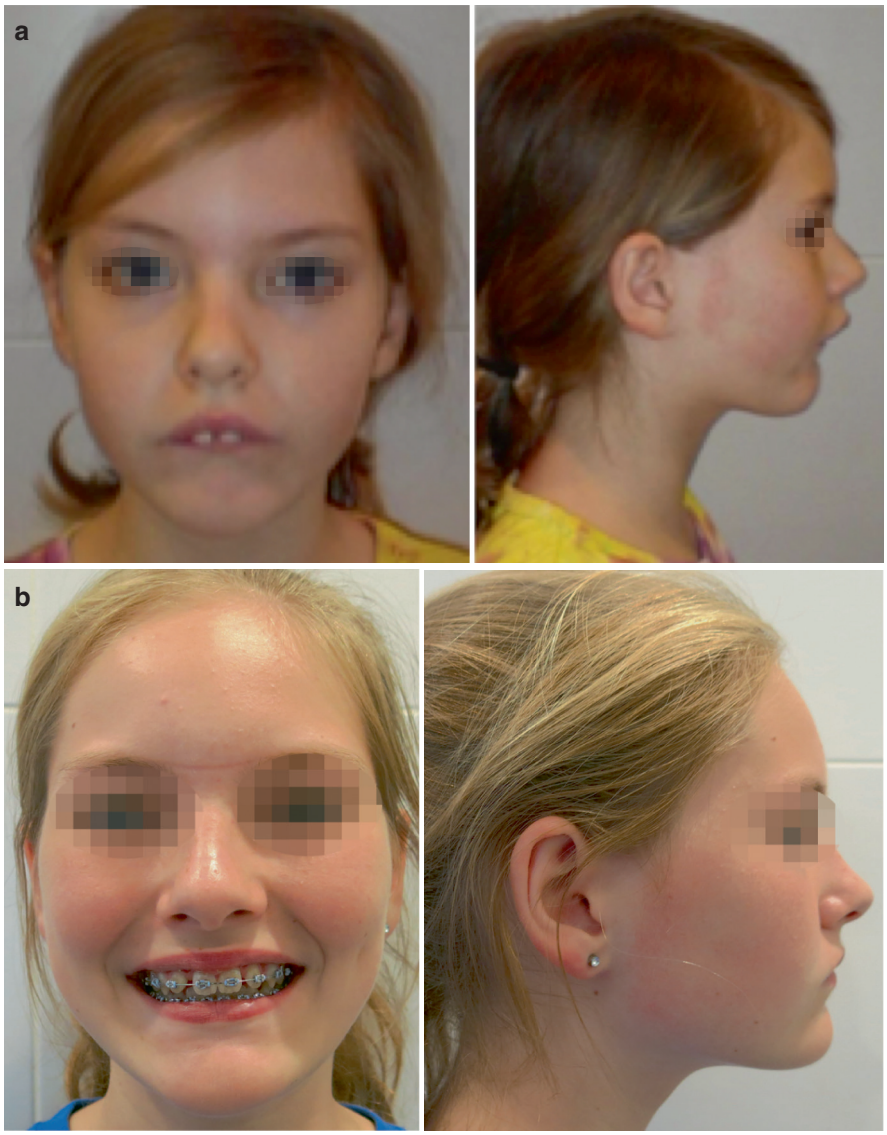

Fig. 30.3 (**a**) A-pre-Tx facial and profile and (**b**) progress Tx facial and profile. SM Class II profile

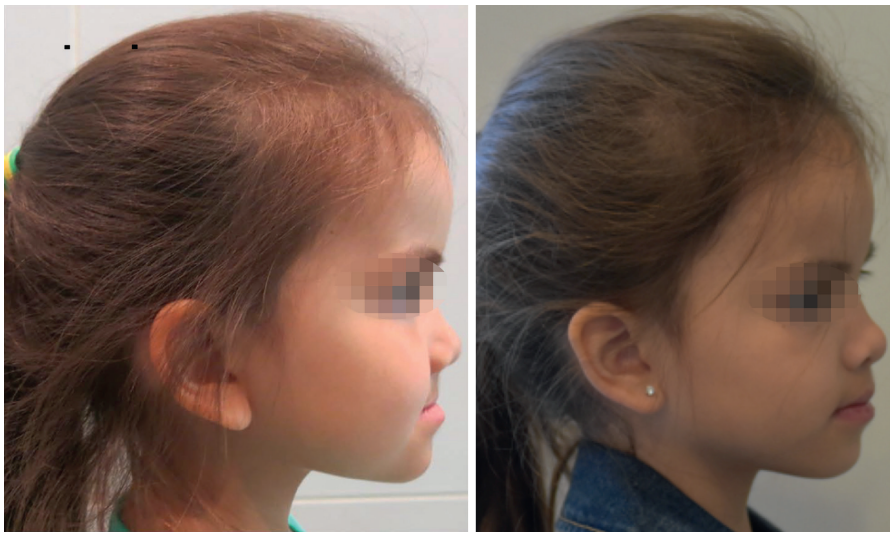

Fig. 30.4 HSM Class III profile

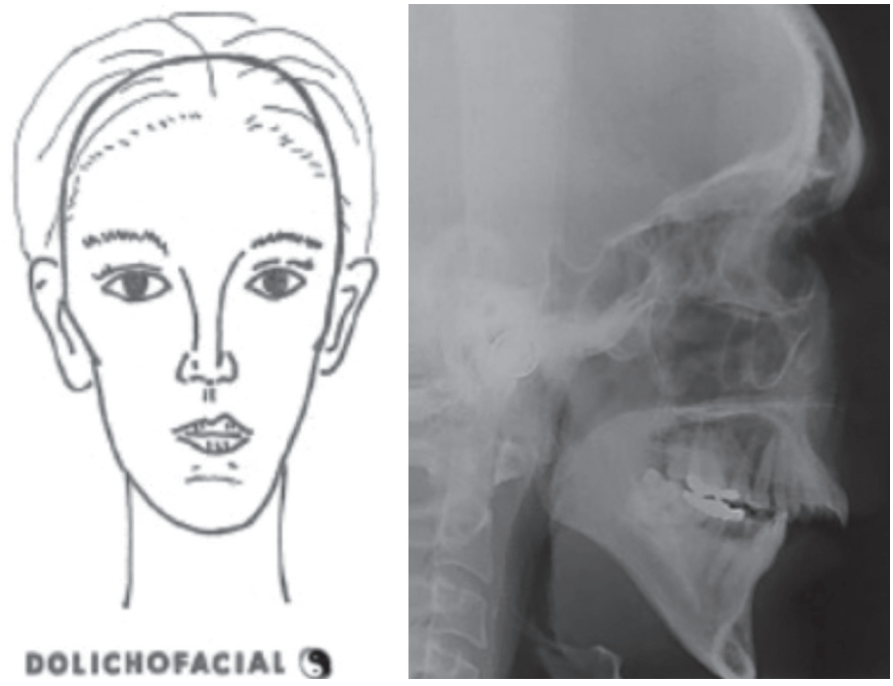

Fig. 30.5 Vertical facial growth (long-face phenotype)

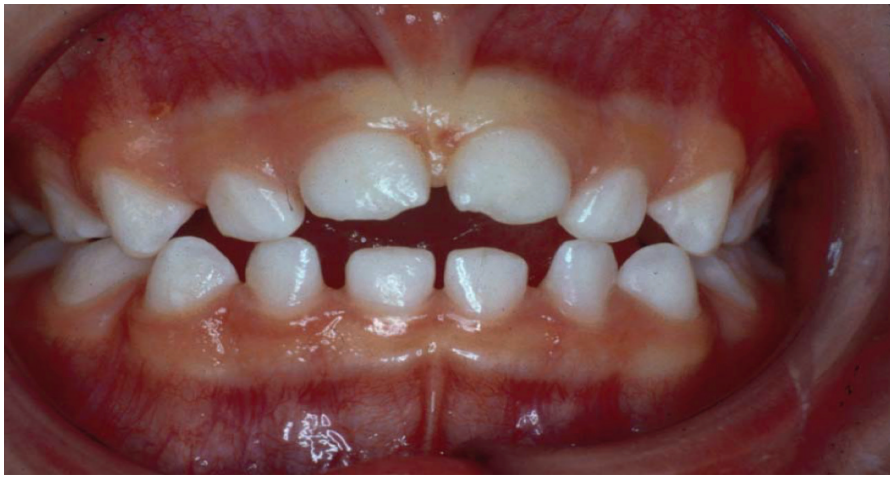

Fig. 30.6 Anterior open bite

is often initially detectable and treatable in the primary dentition and early mixed dentition. It can worsen beyond if left unaddressed with effective intervention strategies [15]. *Anterior open bite* (Fig. 30.6), as the name implies, is characterized by an inability of the upper front teeth to close over the lower front teeth. This is in an effort to establish a normal overbite when the upper and lower posterior teeth (molars) are occluding with one another. Again, anterior open bites are often first detectable during early childhood but also can frequently appear initially at more mature phases of dentofacial development. Prevalence of specific SM phenotypes in the primary and early mixed dentition is approximately 20% and rises to 70% upon the initial eruption of the permanent teeth around age 7 [16]. Because of this, we believe SM and possible existing or future comorbidities, should be screened for at much younger ages than is now considered as being *conventional*.

Using Evolutionary Medicine to Approach Dentistry

The relatively recent appearance of chronic *Western diseases* in humans, such as allergeric rhinitis, obesity, type 2 diabetes, cardiovascular disease, dental caries (cavities), skeletal malocclusion, etc., is very likely *not* the result of Mendelian genetic change over the past few centuries. A more plausible explanation is in evaluating modern health problems from an evolutionary perspective. Evolutionary medicine (EM), also known as Darwinian medicine, provides a useful framework for understanding modern systemic diseases [17, 18]; evolutionary oral medicine, or Darwinian dentistry, is the branch of EM whose goals are to understand the evolutionary origins of oral diseases and to use this understanding for development of diagnostic, preventive, clinical treatment and research strategies. Similar to obesity, obstructive sleep apnea, and type 2 diabetes, *oral* diseases of

civilization (DCs) also seem to follow a predictable pattern of pathogenesis. Specifically, if a susceptible individual is identified early, oral DCs can often be *prevented*. Furthermore, if signs and symptoms are detected early, oral DCs can often be successfully *reversed* and/or *controlled/treated*. Finally, if not prevented, reversed, and/or appropriately *controlled/treated*, systemic, and/or oral DCs can threaten health, well-being, and survival. *Boyd* and Sheldon [19] give a detailed explanation for how a genomic-environmental *mismatch* hypothesis (18) helps explain the relatively sudden appearance of interrelated modern CNCD epidemics such as HSM and SDB/OSA that are seemingly coincident with the phenomenon of cultural industrialization. SM is most accurately described as an epigenetically modulated (genetic-environmental) disorder and appears correlated with cultural industrialization.

Comorbid Malocclusion and Naso-Respiratory Compromise: Which Comes First?

Mostly based upon the work of Harvold [20] in the early 1980s, it is often reported that the naso-respiratory compromise (nasal disuse, habitual mouth breathing) is *the* primary factor in the development of narrowed and lengthened jaws and faces of so-afflicted children; that is, so-called *adenoid facies* (aka, *allergic face*, *long-face syndrome*), and other malocclusion phenotypes commonly associated with atopy in childhood including: crossbites, crowded teeth, open bites, short mandibles, and/or maxillas and retrognathia, might indeed be exacerbated by ATH, but it has been hypothesized that ATH is not necessarily the primary intiating etiological factor. In a recently published essay on the controversy surrounding whether ATH usually preceedes lengthening and narrowing of jaws and faces, or vice-versa, Stupak and Park [8] cite Christian Guilleminault, the Stanford University pediatrician and Sleep Medicine physician who in 1976 first identified OSA as a pediatric disease [21], as also one of the first to suggest that malocclusion precipitates adenoid/tonsillar tissue hypertrophy-related breathing problems rather than vice versa [8]. Through multiple studies, Guilleminault teaches that facial structure and abnormal function, tone and/or equilibrium of the tongue, lips and facial expression, and masticatory musculature may be the inciting event triggering mouth breathing and the eventual hypertrophied adenotonsillar tissue. This line of thinking that malocclusion precipitates breathing problems rather than vice versa challenges conventional teaching. This implies that appropriately applied orthodontic/dentofacial orthopedic forces in the primary or early mixed dentition (i.e., before the age of 7) can improve maladapted bony structures of the craniofacial and respiratory complexes. Additional intervention with orofacial myofunctional therapy [22], surgical revision/release of soft tissue restrictions such as ankyloglossia (tongue-tie) [23], and efforts to restore optimal naso-respiratory function, such as Buteyko therapy [24], might also help mitigate the symptoms of naso-respiratory resistance, nasal disuse, and habitual mouth breathing that are commonly associated with atopic and nonatopic disease in childhood. Further research in this area is needed.

Orthodontic/Dentofacial Orthopedic (O/DO) Strategies in Early Childhood

As previously stated, short retrusive mandibles (Class II SM) and maxillas (Class III SM), unilateral posterior crossbites, vertical skeletal growth, anterior open bites, and other SM phenotypes (e.g., highly vaulted hard palates, crowded teeth, obtuse nose-lip angles, etc.) are often comorbid with atopy and other non-atopic health conditions (e.g., preterm birth, intrauterine growth restriction, obesity, fetal exposure to alcohol and/or primary/secondary tobacco smoke, deviated nasal septum, etc.). Most, if not all of the aforementioned co-morbidities are usually first evident in the primary/early mixed dentition, and usually worsen in later childhood and adolescence without appropriate intervention are often comorbid with naso-respiratory compromise and associated neurological, somatic, and metabolic problems associated with nasal disuse, habitual mouth breathing, and intermittent hypoxia during wakefulness and sleep. If not adequately controlled or left untreated, it seems reasonable to suggest that delaying orthodontic intervention until adolescence/pre-adolescence might pose unnecessary health risk for such identified at-risk children. Furthermore, as published evidence suggests, appropriately timed and applied orthodontic/dentofacial orthopedic intervention treatment strategies can often yield the therapeutic benefit of decreased naso-respiratory resistance and conversion from a habitual oral to a habitual nasal mode of respiration [25–27].

Class II HSM (also called distoclusion in the orthodontic peer-reviewed literature since the mid-/late nineteenth century) when initially observed in preschool-/school-age years does not self-correct [28] and tends to worsen [13]. When therapeutic O/DO intervention strategies are implemented, they can indeed resolve Class II and Class III HSM phenotypes. We hypothesize they might also improve naso-respiratory competence.

Non-retractive O/DO Intervention Strategies

Maxillary insufficiency, defined by both constriction in the transverse (width) dimension and retrusion in the sagittal (length) dimension, also called maxillary sagittal insufficiency, is the skeletal phenotype that is most commonly associated with SM Class III. However, McNamara demonstrated that retrusion of the upper jaw and midface can also be strongly associated with distoclusion/HSM Class II [13]. Maxillary expansion as an adjunctive therapeutic intervention for recurrent OSA symptoms adjacent to surgical removal of hypertrophied adenoid tissue was early described in a 1918 article published in the *Boston Medical Journal* [29]: “It would be entirely useless to hope for permanent relief by removing the nasal obstruction if the jaws did not first receive proper orthodontic treatment. It is important to undertake this at an early age, the principal aim being not only to straighten the teeth, but to stimulate growth of the bones of the entire face.” Maxillary and mandibular protraction are both known to improve naso-respiratory competence in children with comorbid retrusive HSM phenotypes, of either or both jaws, and

naso-respiratory compromise. To date, there is not yet an accepted diagnostic descriptive classification for the HSM phenotype that is characterized by maxillo-mandibular skeletal retrusion (MMR).

MMR is a primary preoperative diagnostic indication for maxilla-mandibular advancement (MMA) orthognathic surgery in nongrowing (adult) patients with unresolved OSA. Further works might explore precisely when, in an adult patient's earlier life, did an MMR phenotype initially become evident.

Maxillary and Mandibular Retrusion Phenotypes as SRBD Comorbidities

In 1992 McNamara demonstrated that in Class III "occlusal (retrusion of the maxillary first permanent molar relationship)-skeletal" (retrusion of the entire maxillary base) in 6-year-old (± 9 months) subjects, nonsurgical protraction of the entire maxillary complex with a reverse traction face mask appliance, in combination with transverse expansion of the maxilla utilizing a rapid palatal expansion appliance (Fig. 30.7), resulted in advancement and counterclockwise rotation of the entire maxillofacial complex [30]. This is in much the same manner as surgical MMA in adult patents. A retrospective analysis of 277 cephalometric lateral head films in untreated Class II "occlusal-skeletal" in 8–10-year-olds showed that many Class II subjects are *not* maxillary *protrusive*, but rather, most are actually maxillary neutral or *retrusive* [13]. As evaluation of skeletal traits associated with the diagnoses of

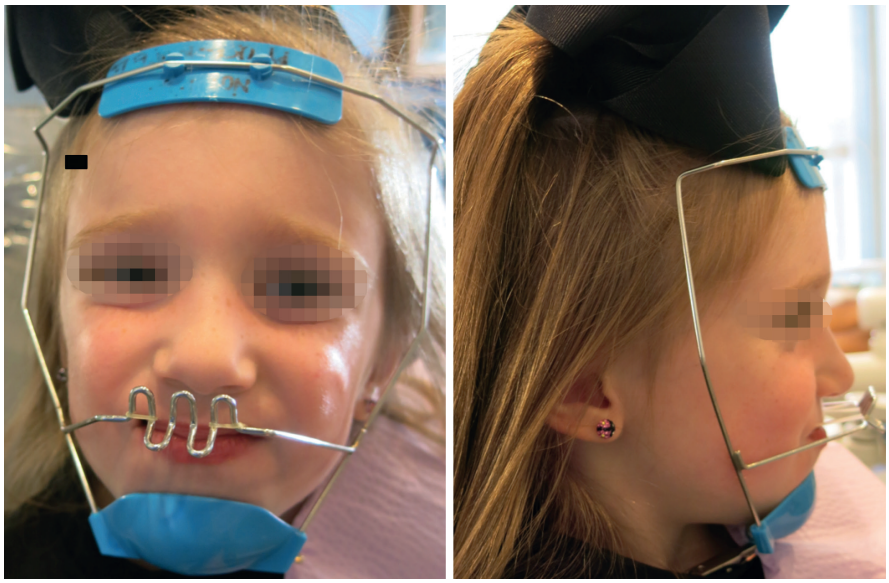

Fig. 30.7 Reverse pull face mask

Class II and Class III malocclusion has been the primary protocol objective, pre-/posttreatment effect data concerning airway health parameters has not been assessed.

As the mandibular retrognathia commonly associated with Class II HSM may also often be a diagnostic facial feature of SRBD, and that maxillary retrognathia commonly associated with Class III *and* Class II HSM may also be a comorbidity of SRBD, it is plausible to suggest that resolving the structural trait maldevelopments associated with both HSM types could lead to functional improvements such as improved airway competence [19, 31].

In 1976 Hershey [27] showed that rapid maxillary expansion is not only an effective method for increasing the width of narrow maxillary arches, but also reduces nasal resistance from levels associated with mouth breathing to levels compatible with normal nasal respiration. In 1980 both Subtelny [31] and Oktay [25] showed that with the addition of a reverse pull face mask, traction appliance to a maxillary retrusive patient undergoing rapid palatal expansion could increase the posterior pharyngeal airway space at the level of the naso- and upper oropharynx. Similarly, many other functional orthopedic appliance treatment strategies that are designed to protract retrusive mandibles, such as Twin Block, Herbst, Bionator, Biobloc [19], etc., also have the added benefit of increasing the posterior airway space at the level of the oro- and hypopharynx.

Conclusion

Many growing children afflicted with atopic diseases have comorbid structural maldevelopments of the craniofacial-respiratory complex (CFRC). Although the epidemiology is not well characterized, several characteristic patterns of human skeletal malocclusion in atopic patients have been noted: constricted width of the maxilla (unilateral posterior crossbites), excessive vertical jaw growth (long face), anterior open bites, and decreased length (short) of mandibles and maxillas. These particular HSM traits represent dysmorphology in all three planes of the CFRC space and are all associated with habitual mouth breathing/nasal disuse. As many HSM phenotypes are first identifiable in early childhood, we believe that in the presence of respiratory dysfunction, treatment for HSM should be discussed at much earlier ages than is currently considered common practice (i.e., before the age of 7).

Providing assurances of cavity-free teeth, disease-free gums, healthy TMJ/jaw joints, and uncrowded straight-toothed smiles, have all rightfully long been the primary criteria for defining optimal pediatric oral health and orthodontic treatment success. With the recent "rediscovery" from within past medical and dental literature dating back to the nineteenth and early 20th centuries [32] that HSM, naso-respiratory competence, and neurological health are all interrelated, it has become increasingly apparent that the *current* list of criteria necessary for defining optimal oral health and orthodontic success should be expanded to include the optimizing the ability to breathe habitually through the nose during wakefulness and sleep, at the earliest stage of a child's CFRC development as might be feasible.

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