Orthodontics and Sleep-Disordered Breathing

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Contents

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The topics of disturbed sleep and the airway have drawn interest among orthodontists since the beginning of the profession. In fact, these issues were discussed more than a century ago in the very first issue of the *American Journal of Orthodontics & Dentofacial Orthopedics* in 1915 (then *The International Journal of Orthodontia)*, in which physician Daniel M'Kenzie discussed their potential relation with craniofacial structure and malocclusion [1]. As discussed in the previous chapter (▶ Chap. 9), adenoid hypertrophy, mouth-breathing, and other related issues are surmised to have some effect on craniofacial growth, malocclusion, and respiration. Many orthodontists have taken a general interest in these issues and their potential relationship with obstructive sleep apnea (OSA). This chapter focuses on an evidence-based discussion regarding these topics as they relate to OSA.

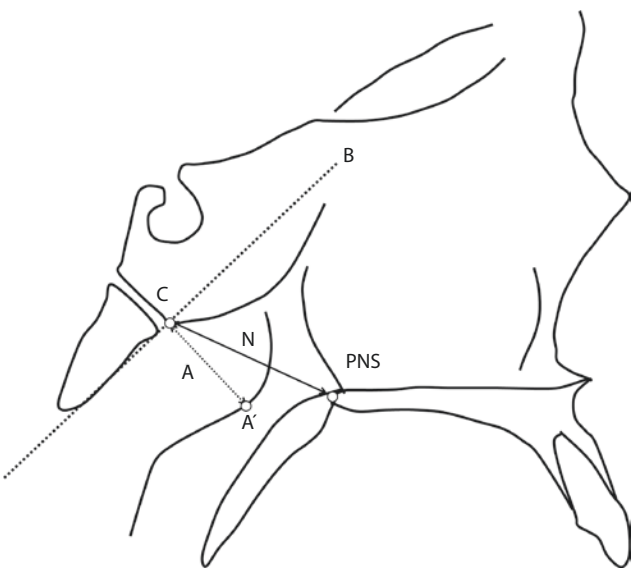
10.1 The Diagnostic Value of Cephalometrics for Airway Evaluation

The lateral cephalogram is the part of the standard orthodontic records and the most commonly used imaging modality. Because the diagnostic process using cephalometric radiographs and cone beam computed tomography (CBCT) was discussed in the previous chapter, evaluation of adenoid hypertrophy and obstruction in the nasopharyngeal airway using lateral cephalography will be discussed in this chapter. The relationship between facial growth and breathing has been a subject of controversy in orthodontics, particularly relating to how adenoid tissue and mouth-breathing affect craniofacial growth. A variety of imaging techniques have been used to diagnose adenoid hypertrophy [2–7].

Nasal endoscopy is the most common method in otolaryngology to evaluate adenoid hypertrophy and nasopharyngeal airway obstruction [8–12]. In addition, rhinomanometry [13, 14], acoustic rhinometry [15], fluoroscopy [12], computed tomography (CT) [16], cone-beam computed tomography (CBCT) [17–21], and magnetic resonance imaging [22, 23] have been used as well. Besides cephalometrics and CBCT, however, the remaining imaging techniques are not commonly used in orthodontics because of their invasiveness, high radiation, and cost.

Many researchers have used cephalometrics to identify key craniofacial characteristics of OSA patients, and several studies have investigated its diagnostic value in identifying adenoid hypertrophy and upper respiratory tract obstruction [24–29]. In 1979, Fujioka et al. [4] introduced the adenoid-nasopharynx (A/N) ratio to determine adenoid size using cephalometrics. Its advantage is the assessment is not impacted by changes in horizontal or vertical position of the patient [30]. McNamara's analysis, or McNamara's line, has become one of the most important and common analytical tools for orthodontists to evaluate and describe structural relationships that affect the airway and is fundamental for diagnosis of many conditions, including adenoid hypertrophy [28]. (See ◘ Figs. 10.1, 10.2, and 10.3).

Caylakli et al. [8] reported on the reliability of the A/N ratio calculated by a lateral cephalogram (evaluated by a blinded author) and nasal endoscopy for measuring the size of adenoid tissue. A total of 85 patients (52 males, 33 females; mean age: 5.0 ± 2.2 years; range: 2–12 years) with a suspected prediagnosis of adenoid hypertrophy between June 2007 and March 2008 were included. The average A/N ratio was 0.87 ± 0.1, which was reported to have a statistically significant Pearson correlation with nasal endoscopy ($r = 0.511$; $P < 0.0001$). However, Feres et al. [31] questioned the value of the lateral cephalography regarding detection of adenoid hypertrophy and nasopharyngeal obstruction in their systematic review, citing spectrum bias in the evaluation of patients with the disease and those without. They noted that the study by Caylakli et al. was the only one among all studies citing the A/N ratio that recruited patients with *suspected* adenoid hypertrophy, whereas the other four studies [6, 15, 32] included patients with an previously confirmed diagnosis.



◘ **Fig. 10.1** A/N ratio. Adenoidal measurements (A): distance from A′ point of maximal convexity, along inferior margin of adenoid shadow to line B, drawn along straight part of anterior margin of basiocciput. Nasopharyngeal measurement (N): distance between posterior nasal spine (PNS) and C, anteroinferior edge of sphenobasioccipital synchondrosis

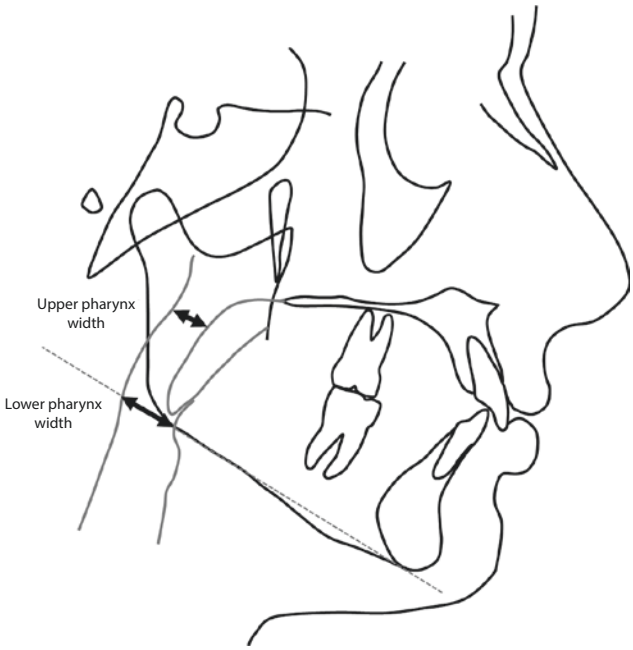


Fig. 10.2 McNamara Analysis. Airway widths according to McNamara analysis, upper pharynx and lower pharynx widths

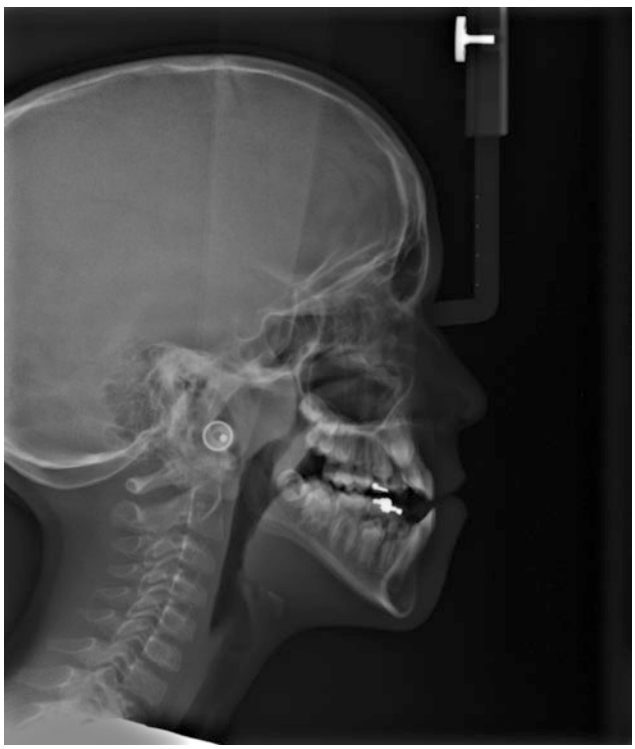


Fig. 10.3 Example of hypertrophic adenoid cephalometrics

Saedi et al. [33] evaluated the diagnostic efficacy by comparing patient's symptoms with nasal endoscopy and lateral cephalometrics findings. They found both cephalography and nasal endoscopy could adequately define the relationship between adenoid hypertrophy and associated symptoms, affirming that this method was useful as a treatment planning tool. Kurien et al. [34] also evaluated the reliability of lateral cephalography in the diagnosis of adenoid hypertrophy and determine if flexible nasopharyngoscopy validated findings. They showed statistically significant agreement was observed between the two techniques, although the accuracy of lateral cephalography was a suboptimal 65%.

Wang et al. [35] compared 109 patients evaluated with both nasal endoscopy and lateral cephalometrics, finding a highly significant relationship ($P < 0.0001$) between both imaging methods. However, there was some disagreement between the two imaging methods. Notably, only 54% of patients who showed adenoid hypertrophy by lateral cephalography radiographs were confirmed by nasal endoscopy. In addition, radiographs revealed 25.4% of children had a large adenoid that nasal endoscopy could not confirm and, conversely, nasal endoscopy revealed a large adenoid in 13% of children that radiographs could not confirm.

Filho et al. [36] in 2001 reported that while lateral cephalography promised high sensitivity, specificity was low in the diagnosing hypertrophy of the inferior and middle turbinates vs. nasopharyngeal endoscopy. They suggested nasal endoscopy is a more suitable method for the diagnosis of diverse nasopharyngeal obstructions. Major et al. confirmed this in 2014. Even though lateral cephalography showed good to fair sensitivity, they found specificity widely varied, depending on the evaluation method used. Conversely, the clinical exam was found to yield poor sensitivity but good specificity [37].

Furthermore, cephalograms have many disadvantages, such as the use of ionizing radiation [38] and can only represent a 3D structure with a superimposed 2D image [39]. One 2006 systematic review by Major et al. [40] concluded that cephalograms can be used to evaluate adenoid hypertrophy, but they are less reliable for determining the size of the nasopharynx. They suggest lateral cephalography is best used as a screening tool for diagnosing obstructed upper airways before a more rigorous follow-up is performed.

10.2 Relationship Between Craniofacial Characteristics and OSA

Some orthodontists have suggested that the soft tissue of the airway should be considered when establishing an orthodontic treatment plan to improve the likelihood of orthodontic and orthopedic stability [41, 42]. As previously discussed, there is controversy surrounding what specific morphology of the craniofacial structures, as well as nasal obstruction and mouth-breathing, impacts craniofacial growth. Linder-Aronson reported that

hypertrophic adenoid tissue can cause the retrusion of maxilla and mandible relative to the cranial base, and can also cause narrow dental arches, posterior crossbite, retro-inclination of maxillary and mandibular incisors, short mandibular dental arches, increased facial height, and a low tongue position [43].

Yamada et al. [44] suggested that nasopharyngeal respiratory obstruction is associated with downward and backward rotation of the mandible, upward and backward growth of the condyle, a divergent gonial angle, and anterior open bite. They suggested that permanent craniofacial deformities form because of a nasopharyngeal obstruction that existed prior to and during puberty, causing a skeletal open bite. Trotman et al. [45] suggested different craniofacial morphological associations for lip posture, sagittal airway, and tonsils. However, there is a controversy surrounding the relationships between head posture and/or facial patterns in children with different malocclusions and structures of the pharyngeal airway [46–48].

Other studies report other issues may be related to respiratory problems, such as a lower facial height, a retruded mandibular position, a deep palatal vault, and a posterior crossbite [45, 49, 50].

Martin et al. conducted a study with Class I ideal occlusion patients without OSA, suggesting that different skeletal patterns have different airway dimensions [51]. Freitas et al. [52] evaluated 80 untreated adolescent patients initially divided into two equal groups (Class I and Class II), then separately dividing these groups on the basis of normal and vertical growth patterns. Patients with Class I and Class II malocclusions and vertical growth patterns are known to have significantly narrower upper pharyngeal airways than those with Class I/II malocclusions and normal growth patterns. However, malocclusion type does not appear to influence upper pharyngeal airway width, nor do malocclusion type and growth pattern influence lower pharyngeal airway width.

Similarly, other research reports that Class II patients and hyperdivergent patients had smaller airway size dimensions [53]. Sagittal malocclusion type does not appear to influence upper pharyngeal width; however, hyperdivergent subjects have statistically significant narrower upper pharyngeal width when compared to normodivergent and hypodivergent vertical patterns [54].

Muto et al. [55] reported that the diameter of the anteroposterior pharyngeal airway was largest in a patient group with mandibular prognathism, followed by groups of normal mandible and mandibular retrognathism. They suggested that the anteroposterior dimension of the PAS is affected by different skeletal patterns of the mandible. Adult OSA patients have been characterized by a retrognathic mandible, maxillary hypoplasia, inferior position of the hyoid bone, a greater flexion of the cranial base, with an elongated soft palate [56].

However, one study that employed CBCT imaging showed that patients with different anteroposterior jaw relationships varied in airway volumes and shapes; furthermore, while airway shape differs in various vertical jaw relationships, volume does not [57]. However, one study that evaluated 276 healthy adult subjects 17–27 years of age with CBCT found that SNB (the angle between the anterior cranial base [SN] and the NB line) and oropharyngeal airway volume had a weak statistical correlation with minimum cross-sectional area. Despite this, the authors concluded that craniofacial morphology does not appear to have a big impact on upper airway dimensions [58].

It is difficult to conclude that there is an increased risk of OSA just by observing decreased airway dimensions in cephalography and/or CBCT. A more comprehensive appraisal of OSA risk should be attempted that includes a clinical examination accompanied with a polysomnogram, as well as cephalometrics and/or CBCT examinations.

The 2014 systematic review by Indriksone et al. [48] concluded that there is insufficient evidence to prove that the dimensions of the upper airway differ in various sagittal skeletal patterns. Many studies have tried to elucidate how head and tongue posture affects pharyngeal airway dimension and shape. Furthermore, there have been methodological concerns in studies in which the posture of the head and tongue was not standardized during image acquisition [59–70]. For instance, a standardized posture might be to position the head naturally and then capture the image after the patient has swallowed *and* while the patient is holding their breath. However, it is still questionable if this method will reliably show airway dimensions.

10.2.1 Cephalometric Characteristics of Adult OSA Patients

The following craniofacial characteristics are reported to be different between normal, healthy adults and adult individuals with OSA.

10.2.1.1 Cranial Base

Some studies reported that the cranial base length is larger than the control for an OSA patient [71, 72], but others showed that there was a significantly shorter cranial base length [73–83]. (See ◘ Figs. 10.4 and 10.5).

According to a meta-analysis by Neelapu et al. [84] reported that SN length in adult OSA patients was 2.25 mm shorter than normal [84]. The authors concluded that a decrease in cranial base length strongly suggests shorter dimensions of the anteroposterior cranium, ultimately expressed as bimaxillary retrusion and a relatively smaller pharyngeal airway.

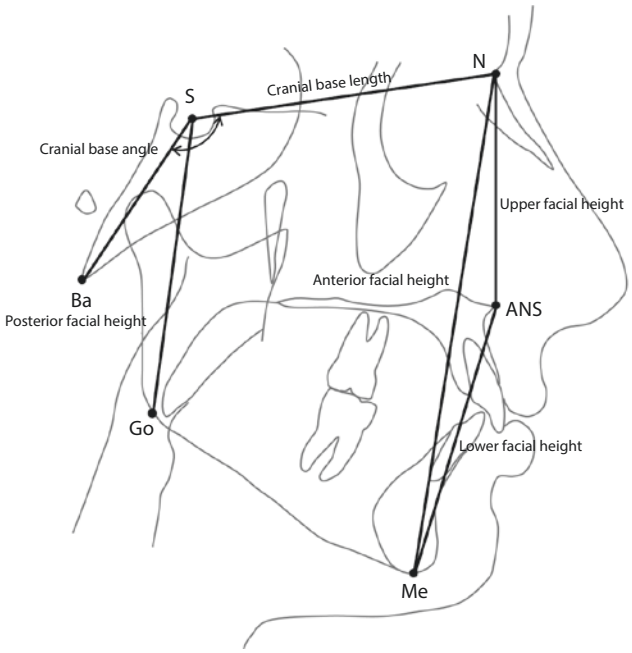


Fig. 10.4 Cranial base and facial height. Cranial base length: N (Nasion) to S (Sella), cranial base angle: N (Nasion) – S (Sella) – Ba (Basion), upper facial height: N (Nasion) to ANS (Anterior nasal spine), lower facial height: ANS (Anterior nasal spine) to Me (Menton), anterior facial height: N (Nasion) to Me (Menton), posterior facial height: S (Sella) to Go (Gonion)

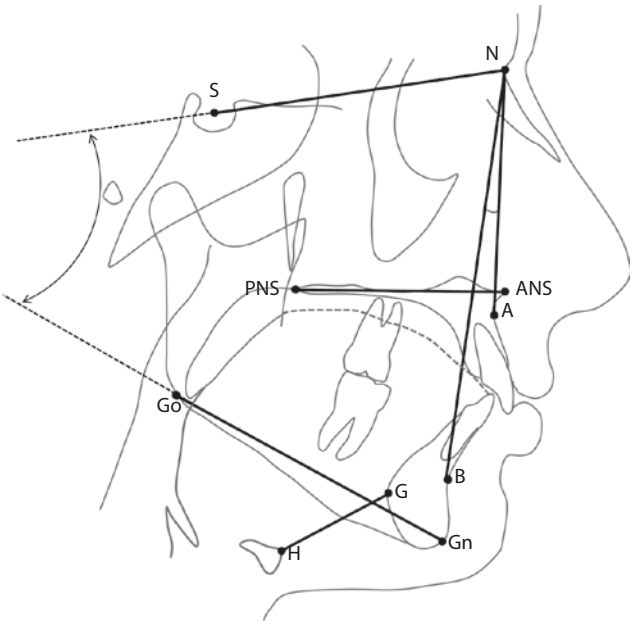


Fig. 10.5 Cephalometric landmarks and measurements. S (Sella), N (Nasion), A (A point), B (B point), SNA, SNB, ANB, PNS (posterior nasal spine), SN-GoGn (Gonion to Gnathion), G (Genial tubercle), H (Hyoid)

Several studies have reported significant decreases in cranial base angles in confirmed OSA patients [73, 74, 76, 78–81, 85–91]. The flexion of the cranial base has been correlated with pharyngeal dimensions. The reduced cranial base angle results in decreased anterior–posterior airway dimensions by a more forward position of cervical spine and posterior pharyngeal wall [92, 93]. Neelapu et al. reported in their meta-analysis that the SNBa angle of OSA patients was 1.45 degrees less than the normal control group [84]. One limitation of this meta-analysis was that age- and sex-matched control groups were limited (absent from 12 of 20 studies evaluating soft palate length and area in OSA patients), highlighting the importance of future research to better match control groups for comparison.

10.2.1.2 Facial Height

Another major finding of the meta-analysis by Neelapu et al. [84] was the increased lower anterior facial height in OSA patients vs. controls, [73, 74, 76, 78, 80, 84, 94, 95], which was found to be 2.48 mm longer than normal group (z-test for overall effect, $P = 0.004$).

10.2.1.3 Maxilla and Mandible

Mandible in OSA patients shows retruded position compared to a normal group [71, 72, 75] [76–79, 81, 83, 85, 89, 91, 94–101]. SNB angle in OSA patients was 1.49 degree smaller than normal group [84] (z-test for overall effect, $P < 0.00001$). The size of the mandible in OSA patients was observed to be significantly smaller than the control group [71–73, 76–79, 94, 97, 99, 100, 102, 103]. Go-Me was significantly shorter by 5.66 mm in OSA patients vs. controls ($P < 0.00001$), but while Go-Gn was 2.08 mm shorter in OSA patients vs. controls, this finding was not significant for overall effect ($P = 0.12$), though significant heterogeneity was found ($P < 0.00001$) [84]. For the SNA where the maxilla position could be assessed, no significant difference was found in the OSA group compared to controls [71, 72, 75–79, 81, 83, 85, 89, 91, 94–100], but the maxillary length evaluated from the distance of the ANS and PNS was smaller in OSA patients compared with normal group [71, 72, 77–79, 81–83, 96, 99]. Maxillary length was significantly shorter in the OSA group by 1.76 mm vs. controls ($P = 0.006$) [84].

10.2.1.4 Pharyngeal Airway Space

Several studies report that the pharyngeal airway in OSA patients was decreased compared to controls [76, 81, 83, 85, 91, 96, 99]; however, these reports should be prudently considered because of their reliance on cephalometrics, which cannot represent the dynamic motion of respiration.

10.2.1.5 Soft Palate and Tongue

The length, thickness, and area of the soft palate was found to be increased in OSA patients [71, 74, 76, 78, 79] [81–83, 89, 91, 94, 96, 97, 100, 102, 103]. There are many studies that show increased tongue length and tongue area in cephalometrics [71, 72, 76, 79, 81, 85, 91, 96, 97, 99], but these results should be cautiously interpreted because tongue position and breathing stage are known to be inconsistent during image acquisition.

10.2.1.6 Hyoid Bone Position

Hyoid position in cephalometrics remains controversial [69, 104]. Malkoc et al. in 2005 demonstrated that hyoid and tongue position are indeed highly reproducible on natural-head-position cephalogram [68]. Many studies reported that hyoid bone in OSA patients is inferiorly positioned [76, 79, 83, 87, 91, 94, 97, 100, 102, 103, 105–109]. A lowered hyoid position has also been associated with a posteriorly positioned tongue because the muscles that connect the tongue to the hyoid would pull the tongue posteriorly when the hyoid is more inferior.

All these craniofacial characteristics in adult OSA patients must be carefully considered. There is no uniform consensus between craniofacial morphology and airway dimension, albeit with weak correlation [58]. Moreover, it is also noteworthy that evidence is lacking that might explain a direct causal link between certain craniofacial characteristics leading to adult OSA or predisposing the risk of OSA.

10.2.2 Cephalometric Characteristics of Pediatric OSA Patients

Mouth-breathing patients have long been known to show a higher posterior crossbite, anterior open bite, and a Class II malocclusion. However, the existence of rhinitis and enlarged adenoid(s) and tonsil(s) have not been determined to be risk factors in the development of Class II malocclusion, anterior open bite, or posterior crossbite. Importantly, a large body of research suggests that certain craniofacial characteristics are associated with pediatric OSA patients [47, 55, 110–120]. One study reported that children with a posterior crossbite and convex facial profile were associated with a higher likelihood of a sleep breathing disorder [121].

Kim et al. [47] compared in 2010 the 3-dimensional pharyngeal airway volumes of healthy children with a retrognathic mandible to children with normal craniofacial growth. Total airway volume was observed to be significantly smaller in retrognathic patients compared to controls with a normal anteroposterior skeletal relationship. Alves et al. [120] assessed the pharyngeal airway dimensions in 50 children with different anteroposterior skeletal patterns using CBCT. They found that the pharyngeal airway space was significantly larger in several measurements of the normal skeletal group vs. the retrognathic group, suggesting that this airway space is influenced by varying anteroposterior skeletal patterns. Deng et al. [122] reported that children with OSA showed increased SNB angle, retrusive mandible, and small chin.

Few studies have reported that children with OSA present with a retrusive mandible is confirmed to correlate with an increased ANB angle [92, 112, 123–127], but several reports suggest that the hyoid bone in adolescent OSA patients is inferiorly located as adult OSA patients [80, 95, 124, 128, 129]. Children with OSA have also been reported with reduced anteroposterior width of the upper airway [126, 127, 130].

It is important to consider opposing evidence. Some researchers argue that craniofacial morphology and pediatric OSA are unrelated [54, 131, 132]. For instance, Oh et al. reported no statistically significant differences in airway volumes between Classes I, II, and III [133]. The dimensions of the anteroposterior pharyngeal airway were shown to not be affected by changes in the ANB angle. Furthermore, no significant differences were noted in a comparison of airway dimensions in different skeletal patterns [115]. Despite data suggestive of an association, there is currently insufficient evidence to definitively link differing upper airway dimensions in various sagittal skeletal patterns [48, 134].

Memon et al. [54] evaluated 360 healthy adolescents with no complaints of nasal obstruction. They reported that type of sagittal malocclusion had no bearing on the width of the upper pharynx. However, hyperdivergent subjects have a statistically significant narrower upper pharyngeal width when compared to other two vertical patterns.

It is still unclear whether or not there are meaningful differences in airway dimensions between hyperdivergent and hypodivergent subjects. Moreover, simply having a smaller airway does not necessarily mean the risk for a sleep breathing disorder is increased.

Katyal et al. suggest that the association between craniofacial disharmony and pediatric sleep breathing disorders is statistically supported by their data [135]. They reported children with OSA were observed with an increased ANB angle (>2 degrees), attributable to a decreased SNB angle. However, this result was regarded as having marginal clinical significance. Distance from the PNS to the nearest adenoid tissue was observed to be reduced in OSA children. This illustrates that reduced upper airway sagittal width occurs in children with OSA, but the authors could not confirm a direct causal association between craniofacial structure and pediatric OSA. At present, no solid scientific evidence has shown that Class II malocclusion with retrognathic mandible and hyperdivergent skeletal pattern increases the risk or causes sleep breathing disorders.

10.3 Relationship Between Craniofacial Characteristics and OSA

While there are arguments that support improved OSA via certain orthodontic treatment modalities, there also exist opposing arguments that suggest certain orthodontic treatments may worsen OSA symptoms or even cause OSA.

10.3.1 Maxillary Expansion

Rapid maxillary expansion (RME) was first introduced by Angell [136] in the 1860s, which was later reintroduced by Haas in 1965 [137]. This technique has been used for correcting posterior crossbite, a constricted maxilla, and to gain extra arch length [138–140]. (See ◘ Figs. 10.6 and 10.7).

Type of maxillary expansion is differentiated by rate and characterized as either "slow" or "rapid." Slow expansion is considered 0.25–0.5 mm per week, whereas rapid expansion is considered >0.5 mm expansion per day [141, 142]; the most common expansion protocol is rapid [143]. Orthodontists select expansion appliances based on expansion rate, patient age, malocclusion with vertical skeletal patterns, as well as their own clinical experience. Some expansion appliances have an expander screw in the center, adjustment of which depends upon the type of anchoring, bonding (acrylic covering over the occlusal surface of the posterior teeth), and banded (bands cemented to posterior teeth), and the Haas expander (acrylic cover the palatal tissue with bands cemented to posterior teeth) or tooth-anchored expander or bone anchored expander which uses mini-screw implants to anchor (◘ Fig. 10.8).

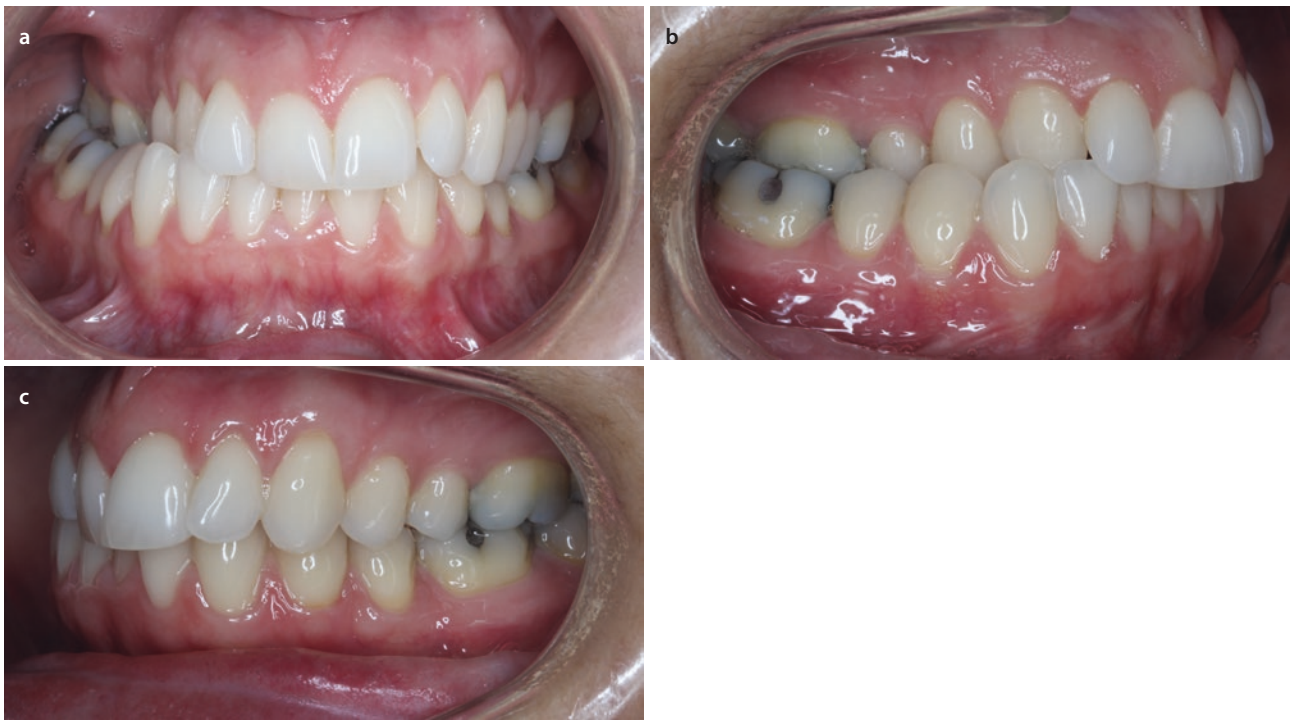
The primary purpose of maxillary expansion is to mechanically open the midpalatal suture with the intent to appreciably widen the maxilla. Therefore, this technique has been mainly used for adolescents whose sutures are not fully integrated. For adults, however, surgically assisted expansion is recommended [144, 145].

10.3.1.1 Effects of RME on the Nasomaxillary Complex

RME has a profound impact on the entire nasomaxillary complex [137, 139]. RME has been reported to increase the maxillary mid-palatal suture [146–152] as well as maxillary width [146, 147, 151, 152].

Starnbach et al. [153] in 1966 demonstrated that RME also separates the circumzygomatic and circummaxillary sutures. The technique results in significant bone displacement of the frontonasal suture, the intermaxillary suture, the zygomaticomaxillary suture, and the midpalatal suture [154]. (See ◘ Fig. 10.9).

A 2006 meta-analysis by Lagravère et al. [155] reported acute changes following RME. The mean expansion distance was 6.7 mm from the maxillary first molar crowns and 4.5 mm at the molar root apex. For



◘ **Fig. 10.6** Posterior crossbite. **a** Frontal view. Noticed a posterior crossbite on the right side and lower midline is off to the patient's right side. **b** Right buccal view. Posterior crossbite from upper right canine to upper right second molar. **c** Left buccal view. Non-posterior crossbite side

Fig. 10.7 **a** Initial records. **b** After RME. **c** Final records

Fig. 10.7 (continued)

the skeletal transverse changes, nasal cavity width was increased 2.14 mm. A study by Cameron et al. [156] evaluated the longer term effects of RME followed by comprehensive orthodontic treatment. The authors reported that transverse improvements from RME remained corrected at an average of 20 years and 6 months of age. The long-term stability of transverse dimension has been reported to be improved in prepubertal growth peak patients vs. pubertal and postpubertal growth peak patients [157].

10.3.1.2 Effects of RME on the Nasal Airway

The nasal valve area provides the greatest nasal airway resistance [158–160]. RME opens the midpalatal suture to separate the maxilla, which impacts the entire nasal airway passage structure and effectively increases nasal cavity volume [140, 161–168].

Thorne et al. [169] reported in 1960 that nasal width increased by a range of 0.4–5.7 mm post-RME. In 2000, Cross and McDonald evaluated posteroanterior cephalometric radiographs to determine nasal cavity width post-RME in a group of patients with maxillary narrowness (n = 25) compared to untreated age- and sex-matched controls (n = 25; 20 females and 5 males in each group). They reported the maximum nasal cavity width increased by a mean of 1.06 mm ± 1.13 mm ($P < 0.001$) compared to controls [170].

Li et al. [171] demonstrated that nasopharyngeal volume was increased 29.9% post-RME as measured by CBCT [172]. One prospective study showed that nasopharyngeal space was increased 12–15.2% after a mean 2.8–3.7 mm of expansion. Cameron et al. [156] performed a 5-year follow-up study, reporting RME patients sustained a nasal width increase of 4.16 mm vs. 1.52 mm in the control group.

Several studies report post-RME that the dimensions of the nasopharynx and oropharynx sustained no significant changes [173–176]. El and Palomo [177] found that nasal airway volume was significantly increased after RME; however, no significant change in oropharyngeal airway volume was observed. Smith et al. [178] showed that RME increased in nasal cavity volume and nasopharyngeal volume, but no change in oropharyngeal and hypopharyngeal airway. Chang

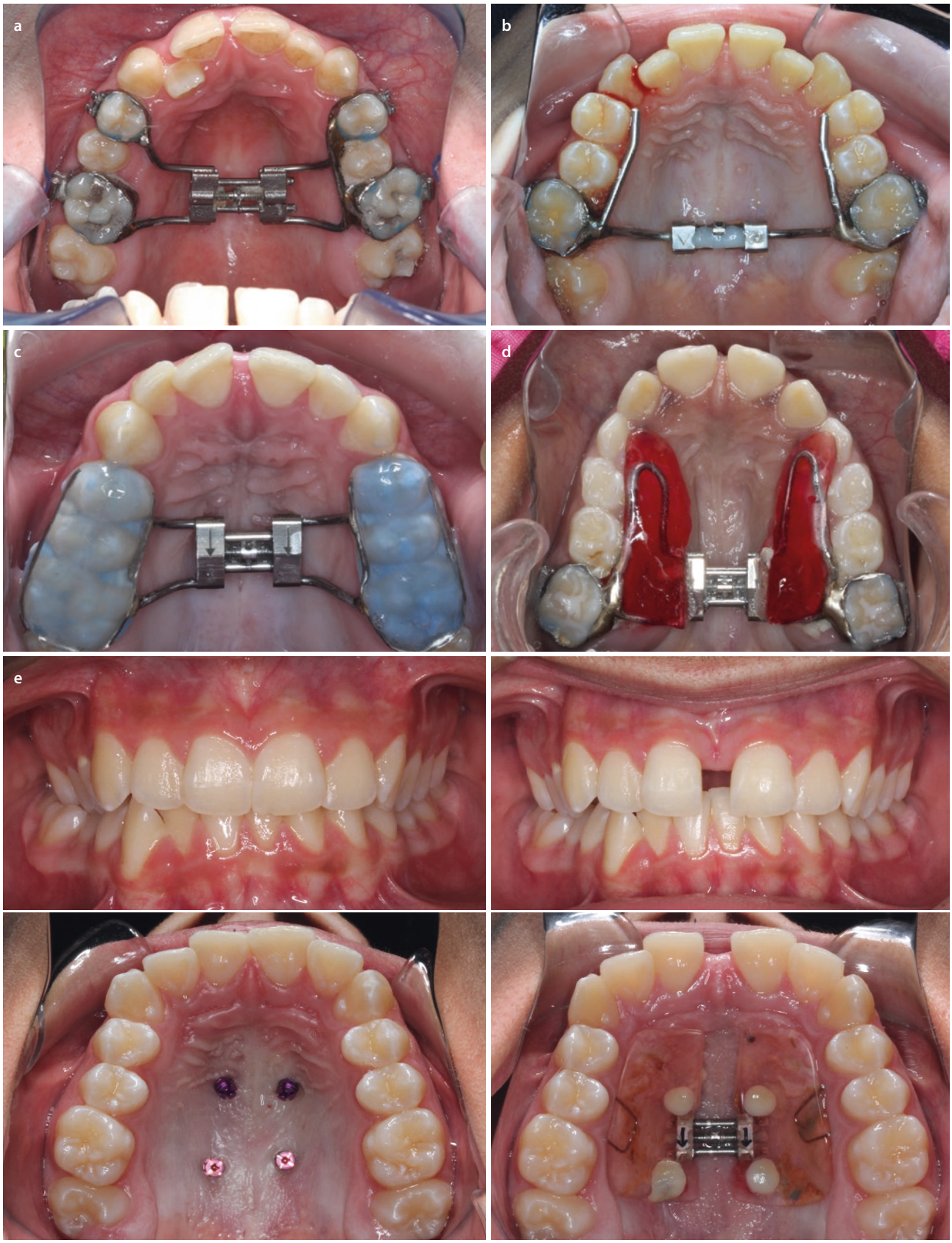
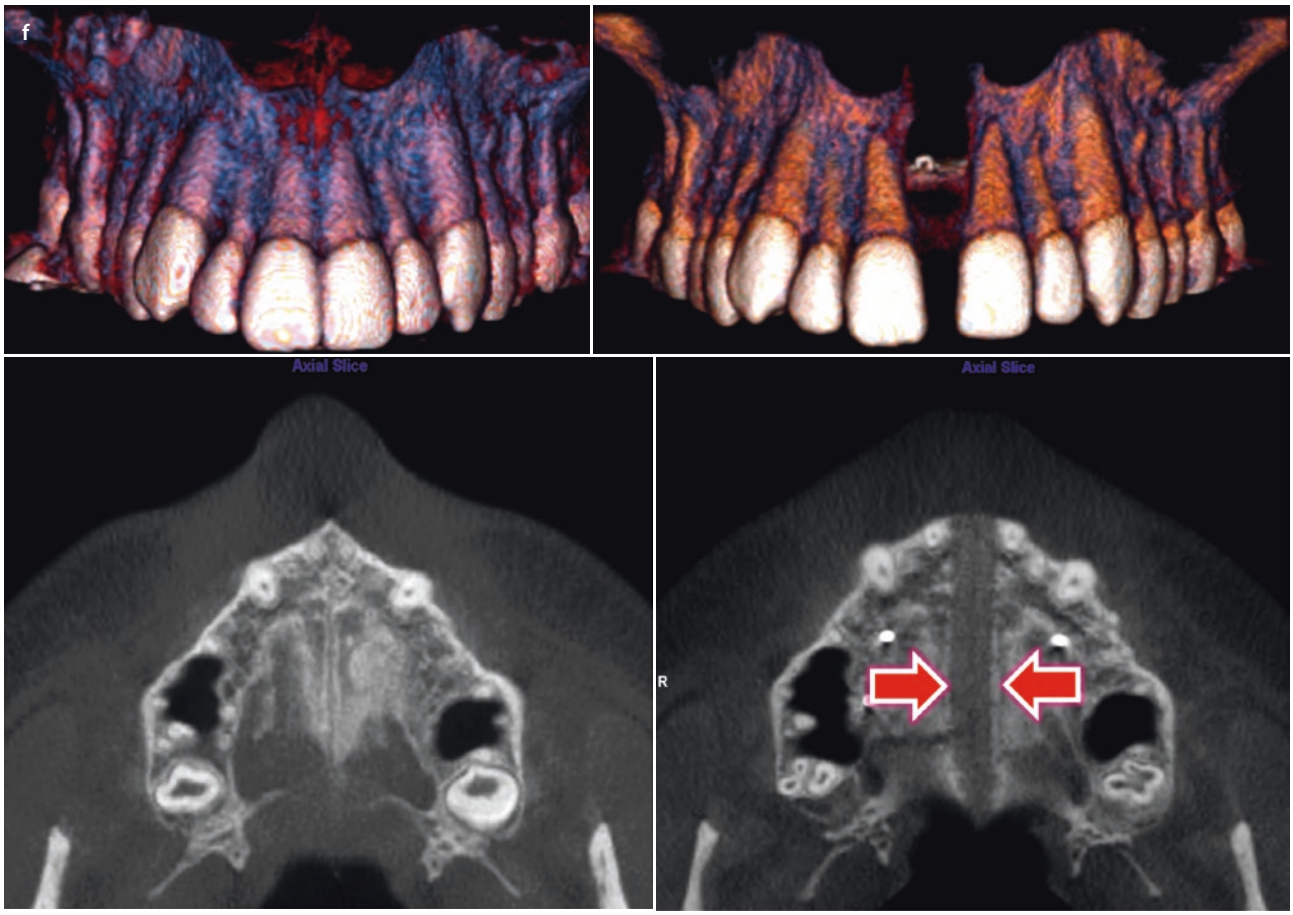


Fig. 10.8 Various expansion devices: **a** Four bands expander, **b** Two bands expander, **c** Bonded expander, **d** Haas expander, **e** Miniscrew-supported expander, **f** Before and after miniscrew-supported expander



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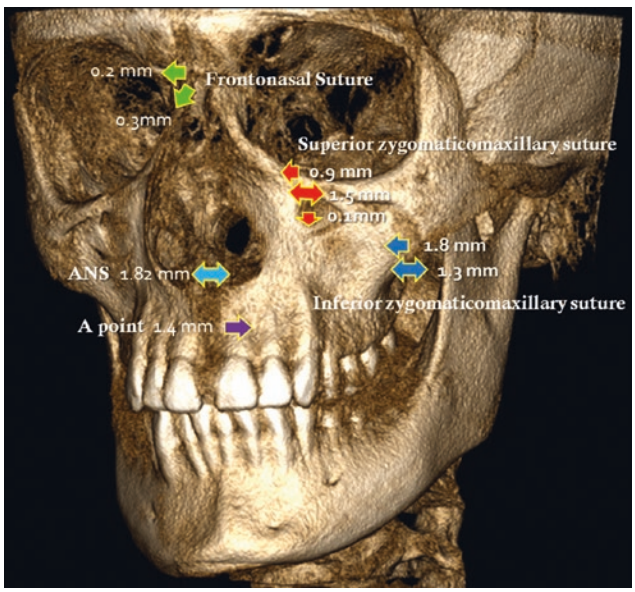


Fig. 10.9 Sutural changes after RME

et al. [179] also showed increased retropalatal airway after RME, but there was no difference in total airway volume.

Zhao et al. analyzed CBCT 15 months after RME and reported no significant differences in the nasopharyngeal and oropharyngeal volume [180]. Zeng and Gao [174] conducted a prospective study of 16 children post-RME using CBCT. Although a significantly increased nasal width was reported, there were no changes in either nasopharyngeal or oropharyngeal airway dimensions observed post-RME. Ribeiro et al. [173] showed that the nasal cavity and oropharyngeal volume increased, but did not find a difference in nasopharyngeal volume. However, they questioned if the oropharyngeal volume increase might be caused by inconsistent tongue posture, head position, and breathing and swallowing movements when the image was acquired. It is notable that all these cited studies (except Usumez et al. [175], 2000) were conducted this decade (2010 or later).

In their 2017 systematic review, Di Carlo et al. [181] cautioned interpretation of reported RME results because of inconsistencies in the CBCT protocols across studies, notably head posture, tongue position, breathing and swallowing movement, as well as segmentation protocols.

10.3.1.3 Effects of RME on Nasal Breathing

Several studies have reported positive outcomes following RME on respiratory disorders, such as mouth-breathing, asthma, colds/respiratory infections, nasal allergies, otitis media, and nocturnal enuresis [158, 182–190]. Stockfisch conducted a long-term study of 150 cases of children aged 6–31 years and with follow-up of 5–15 years post-RME, with general nasal airway improvements reported, including improvement with nasal allergies and asthma [191].

A few studies report that RME reduces nasal resistance [167, 182, 192]. Enoki et al. [193] showed that there was no difference in the minimal cross-sectional area at the level of the valve and inferior nasal turbinate; however, a significant reduction in nasal resistance after RME was observed. The 1987 RME study by Timms [182] reported nasal resistance was decreased by a mean of 37%. Monini et al. reported in 2009 their study of RME on patients who had maxillary constriction, snoring, or nasal obstruction, and reported overall improvements of nasal respiration [190].

Conversely, Giuca et al. [194] reported in 2009 that they failed to find any significant differences in nasal airway resistance post-RME. In 2010, Matsumoto et al. [195] reported an acute decrease in nasal resistance; however, the nasal resistance increased to its initial baseline value after 30 months after RME. Although Timms [196] reported a 36.2% decrease in nasal airway resistance, the correlation between the resistance reductions and the amount of expansion was weak. Patients who showed no change were those whose nasal airway resistance was close to normal.

Several studies have been conducted to determine how long the benefits of RME could be sustained [165, 167, 192, 197]. Oliveira et al. [192] reported that 61.3% of patients reported subjective improvement in nasal respiration 9–12 months after the expander was removed. In fact, most studies have demonstrated improvements remained stable up to 12 months [165, 167, 197]. According to a systemic review by Baratieri et al. in 2011, there is moderate evidence that growing children improve the conditions for nasal breathing, and that stability can be expected for at least 11 months after RME [198].

However, Langer et al. [199] studied RME in 25 children with posterior crossbite and used rhinomanometry to evaluate the nasal airway resistance 30 months after RME. They concluded that RME does not sustain a long-term impact in the nasopharyngeal area or in nasal airway resistance. To this point, Baratieri et al. cautioned that RME is not recommended alone if its primary purpose is to improve nasal breathing because of the wide variability of individual responses [198].

Once a nasal breathing problem is confirmed by comprehensive medical examination, RME could be considered as one of the treatment modalities. However, without solid evidence of long-term benefits, orthodontists should be cautious about using RME, especially for patients who do not have a constricted maxilla and/or posterior crossbite.

10.3.1.4 RME for OSA

Several studies have demonstrated that RME decreases the polysomnography apnea–hypopnea index (AHI) in adolescent OSA patients [185, 186, 188, 200–204]. In 2005, Pirelli et al. studied 42 children without adenotonsillar hypertrophy who received RME, with AHI substantially decreased from 12.17 ± 2.5 to 0.5 ± 1.2 [200].

Miano et al. studied the sleep architecture following RME in children with OSA. RME was found to nearly completely normalize sleep architecture and was noted to improve sleep respiratory disturbances. However, respiratory parameters and sleep microstructure failed to completely recover. In these patients, initial AHI was 17.4 ± 21.0 and 5.4 ± 6.25 post-RME. Nonetheless, no significant differences were detected in mean overnight oxygen saturation/desaturation [201].

One meta-analysis concluded that the mean AHI decrease after RME was 3.24 [205]. In 2016, Machado-Junior et al. [206] performed a meta-analysis on the relationship of RME and OSA 10 articles conformed to the inclusion criteria and were included in this meta-analysis. The total sample size across all these articles was 215 children (mean age: 6.7 years; 58.6% male). Mean AHI during follow-up post-RME was −6.86.

It is not clearly understood how RME positively affects OSA symptoms. In concert with increases in nasal cavity size and the decreases in nasal airway resistance, tongue posture is also raised and maxillary width is increased [186, 203, 207–209]. However, many of these studies using lateral cephalography or CBCT did not control tongue posture, breathing, or swallowing during image acquisition; therefore, the mechanism of improvement is unclear.

Huynh et al. [210] concluded in their meta-analysis that even though there are many studies demonstrate reduced AHI following RME, the considerable heterogeneity of these studies precluded direct comparability of the variable interventions or patient populations. In addition, most study samples were not randomized and lacked a control group. Further well-controlled, randomized controlled trials are needed.

In terms of the long-term effect, Villa et al. [202] reported that after RME, AHI decreased. Twenty-four

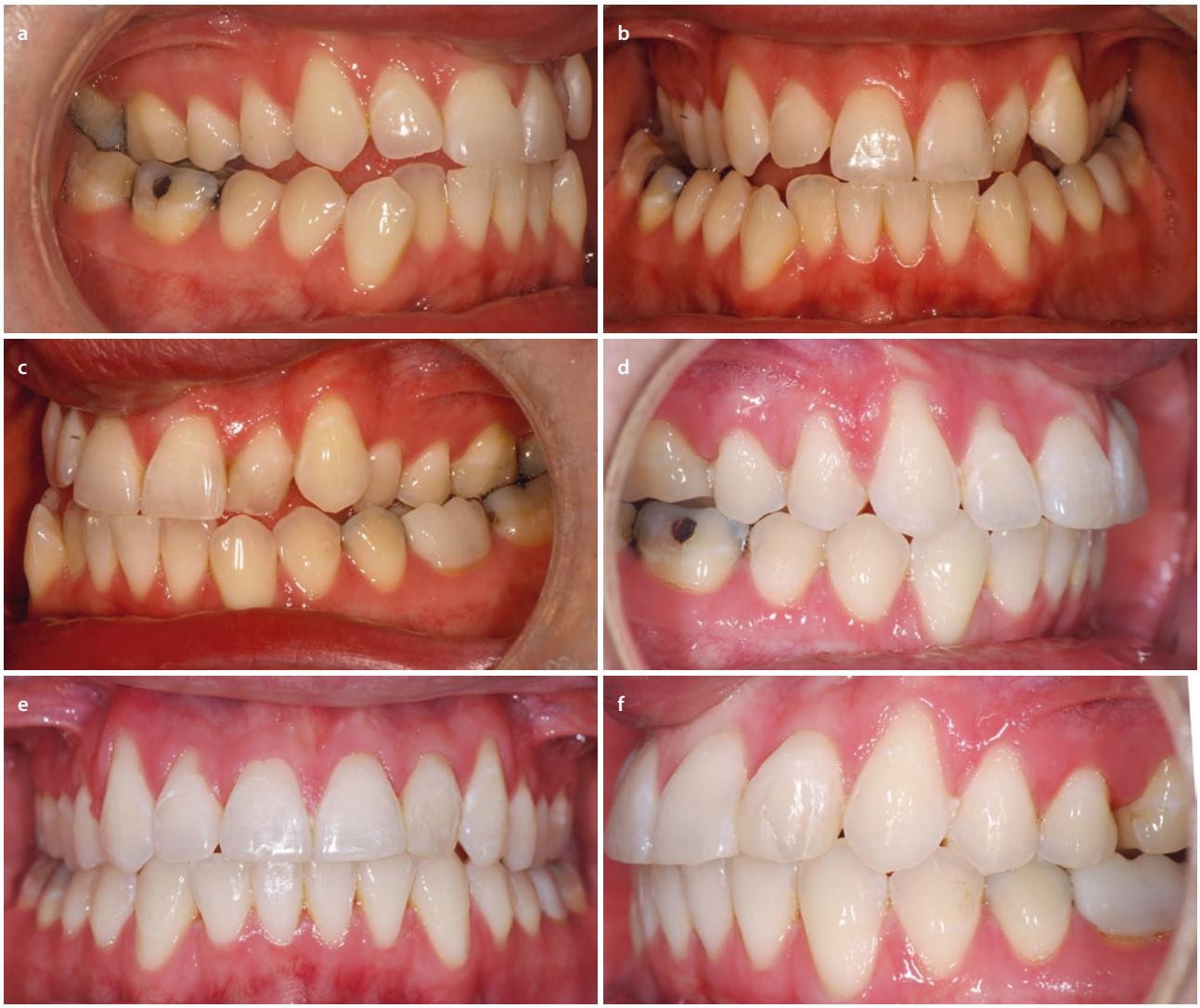
months after the end of the treatment, no significant changes in the AHI were observed. Pirelli et al. studied the long-term efficacy after a 12-year follow-up. All 23 patients were still normal as confirmed by PSG [211]. However, the long-term effect of maxillary width change in the early-treated (pre-pubertal) group was significantly increased about 3.0 mm, but not in the late-treated (pubertal and post-pubertal) group [157].

Applying a heavy force in a young patient may cause a dorsal hump or paranasal swelling [212]. At present, there is no specific guideline for how much expansion is optimal, as well as the ideal rate of expansion to improve OSA symptoms, especially for pre-pubertal patients.

Side effects of RME include alveolar bone loss, dehiscence, fenestration, and root resorption of anchoring teeth [213–215]. An attempt at RME in an adult patient would be futile since a midpalatal sutural opening cannot be achieved, likely causing deleterious periodontal sequelae. Therefore, surgical-assisted maxillary expansion (SARME) is required for adult patients [216, 217]. (See ◘ Fig. 10.10).

After SARME, distinct subjective improvements were reported in nasal breathing and associated with enlargement of the nasal valve toward normal values and an increase of nasal volume [218]. Recently, temporary skeletal anchorage devices have been incorporated to expansion appliances (bone-borne) to minimize the side effects of RME [213, 219–222].

In 2017, Bazargani et al. compared hybrid (tooth-bone borne) RME and traditional (tooth-borne) RME in a randomized controlled trial. The hybrid RME technique demonstrated significantly greater nasal airway flow and lower nasal resistance vs. traditional RME [223]. Another study also suggests that bone-



◘ **Fig. 10.10** Gingival recession caused by RME in an adult patient. **a**–**c** Bilateral posterior crossbite, **d**–**f** Post treatment pictures. Notice the gingival recession in the maxillary canines and first premolars

borne appliance my help to reduce OSA symptoms in adult patients [220]. However, as Algharbi et al. [224] suggested in their systemic review, tooth-borne RME should be used because there is no difference between tooth-borne and bone-borne appliances in adolescent patients.

10.3.1.5 Conclusion of RME Role in Sleep-Related Breathing Disorders

Langer and colleagues recommended RME be used for orthodontic purposes to correct crossbite, but warned that the benefits of RME on nasal function should not be generalized [199]. In 2009, Haralambidis et al. evaluated nasal cavity morphology post-RME using 3D CT. They concluded that RME should not be advocated solely to increase nasal cavity volume and nasal respiration unless a transverse maxillary deficiency is present [176].

It seems certain, however, that RME increases nasal airway volume and decreases nasal airway resistance. Nevertheless, patients must be informed of the side effects of RME and questioned to determine if their OSA problems arose from nasal constriction prior RME. When a patient with normal airway resistance receives RME, it is uncertain whether it will have a positive effect. Additionally, it should be noted that, in most cases, additional orthodontic treatment is needed when RME is completed. This is especially true for a patient who does not have transverse maxillary deficiencies.

In terms of SARME, one must be aware of the side effects following the treatment. SARME should be one component of a comprehensive treatment plan instead of single, independent, unilateral treatment modality because most patients require orthodontic treatment following SARME. Again, randomized controlled trials and other methodologically rigorous studies are needed to determine how much expansion is needed to minimize the side effects of expansion, to maximize the improvement of patient's breathing, and to determine the best timing of RME treatment. It is unethical to proceed with RME for patients who do not have constricted maxilla or posterior crossbite. While there is a body of research that demonstrates RME's positive effects, to date, there is no evidence that posterior crossbite and/or a constricted maxilla is more prevalent in OSA patients and vice versa.

10.3.2 Orthodontic Extraction and the Risk of OSA

Extraction of permanent teeth is performed for a variety of orthodontic reasons, the most common of which is to relieve crowding (tooth size-arch length discrepancy). Both maxillary and mandibular anterior teeth can be retracted to decrease procumbent anterior teeth by using premolar extraction spaces. Maxillary or mandibular premolar extraction spaces can be used to achieve normal overjet as a camouflage treatment in Class II or Class III malocclusion. There have been some claims that anterior teeth retraction followed by extraction can result in a tongue position change, which leads to more posterior position. This positional change has been reported to decrease oropharyngeal airway space and increase the risk of OSA [225–228].

If there is any airway space change after extraction, where is it and how much change will happen? How does this decrease in airway space affect OSA or increase risk of OSA?

In 2005, Kikuchi published a case report of decreased airway dimension after orthodontic extraction treatment in a girl with Class II malocclusion [225]. Two sisters very close in age (elder sister: 12 years, 11 months of age; younger sister: 11 years, 9 months) were concerned about maxillary protrusion and requested orthodontic treatment. The older sister was treated with extraction of five teeth (one was congenitally missing and one tooth root-resorption was noted), whereas the younger sister was treated with nonextraction and with a Herbst appliance. Baseline cephalograms for both sisters, when superimposed, showed few differences. A satisfactory result was achieved in both sisters after treatment (elder sister treatment duration was 3 years, 11 months; the younger sister was treated for 3 years, 2 months). However, upon cephalogram superimposition post-treatment, the elder sister's image revealed 7 mm less pharynx volume compared to the younger sibling, suggesting that orthodontic treatment, such as extraction, might impact airway size in developing adolescents.

Chen et al. [226] reported in 2012 that a decreased airway size resulted after orthodontic extractions with maximum anchorage in adult patients. They also found that the decreased airway size was correlated with the retraction amount of the lower incisors. Germec-Cakan et al. [227] used lateral cephalography to investigate changes in airway dimensions following extraction and reported that middle and inferior airway sizes narrowed in subjects treated with extraction and maximum anchorage. Wang et al. [228] studied 44 Class I bimaxillary protrusion adults with four premolar extractions, and upper airway narrowing was observed following retraction of incisors.

However, some report there are no changes in the airway dimensions after orthodontic extraction [229–231]. Maaitah et al. [229] evaluated 40 adult bimaxillary protrusion patients who required four first premolar extractions. Before and after cephalograms were examined and it was concluded that even with the significant reduction in tongue length and arch dimensions, the dimensions of the upper airway remain unchanged and the position of the hyoid bone not affected. They concluded that

reduction in arch dimensions resulting from extraction does not impact upper airway dimensions. Valiathan et al. [230] evaluated CBCT comparing four premolar extractions vs. a nonextraction group and likewise concluded that extraction of four premolars with retraction of incisors did not affect oropharyngeal airway volume. Stefanovic et al. [231] used 31 subjects with extraction of four first premolars and 31 matched control samples. Before and after CBCT were evaluated and no differences were observed in the pharyngeal airway between groups. Pliska et al. [232] analyzed 74 adult's CBCTs before and after orthodontic treatment. There was no evidence of differing effects on the nasopharynx, or the retropalatal and retroglossal regions of the oropharynx between extraction and nonextraction treatments.

It has not been scientifically proven that dental arch lengths are decreased during orthodontic treatment during development of the upper airway [233]. All prior studies used either cephalography or CBCT. It should be noted that airway size and shape in 2D radiographs can be extremely variable, depending on head posture and the breathing stage [234, 235]. In addition, minimum cross-sectional areas in 3D CBCT have not been found to be a reliable metric for airway analysis [236].

The pathophysiological mechanism of OSA is complex with many possible factors involved. Evidence is lacking to support the direct causal link of a decreased airway space and an increase in the risk of OSA. No prior orthodontic studies have evaluated the impact of extractions, a decreased airway space, and an association with OSA; such a correlation remains speculative. The 2015 systemic review by Hu et al. concluded that while the retractions of anterior teeth may decrease the upper airway dimensions, there is not yet a meaningful answer supported by data that decreased airway size can increase susceptibility of OSA and deleteriously impact sleep quality [237].

3D airway modeling after orthodontic treatment with premolar extraction and maximum anchorage in adults are mainly morphological changes with the anteroposterior dimensions compressed in airway cross-sections, rather than a decrease in size [238]. After orthodontic treatment with premolar extraction and maximum anchorage, the airway volume, height, and cross-sectional area were not significantly changed. Morphology of airway cross-sections was compressed at the anteroposterior dimension with unchanged area after orthodontic extraction treatment in the middle and inferior part of the upper airway. The effect of the morphological change on the respiratory function remains unknown. Whether such effect is stable is another important question, and Larsen et al. [239] set out to answer it. Their sample of 5584 patients was obtained from the electronic medical and dental health records of HealthPartners in Minnesota. Half of the subjects (n = 2792) had one missing premolar in each quadrant and the other half were not missing any premolars. Cases and controls were age-, gender-, and body mass index (BMI)-matched on a 1:1 basis. The endpoint was presence or absence of a diagnosis of OSA confirmed by a polysomnogram. OSA prevalence was not observed to be significantly different between groups; therefore, the absence of four premolars (a presumed indicator of past "extraction orthodontic treatment") was not supported as a significant factor in the cause of OSA.

10.3.3 Headgear and Risk of OSA

Cervical headgear is a widely used extra-oral orthodontic device for children with Class II occlusion. The most common finding concerning its effects on the nasomaxillary complex is the reduction of SNA value, either as a result of restricting the forward growth of the maxilla [240–245] or by the distal placement of the maxilla [246–248]. (See ◘ Fig. 10.11).

Godt et al. [249] reported that use of cervical headgear reduced anteroposterior dimensions at all pharyngeal airway levels during sleep. Hiyama et al. [250] evaluated 10 healthy adults and reported that cervical headgear reduced the sagittal dimension of the upper airway. Kirjavainen and Kirjavainen [251] studied the effects of cervical headgear on upper airway in chil-

◘ **Fig. 10.11** Cervical headgear

dren and reported that the treatment is associated with an increase in the retropalatal airway space. Julku et al. [252] evaluated related craniofacial structures and pharyngeal airway dimensions in children with a Class II occlusion treated with cervical headgear and randomized into early- and late-treatment groups, finding that neither early nor late cervical headgear treatment had any adverse effects on upper airway dimensions. Pirila-Parkkinen et al. [253] divided 30 children into three groups: those treated with headgear therapy (n = 10), an age-matched control group (n = 10), and confirmed OSA (n = 10). The headgear group had a slightly more posterior mandible position vs. the control group, and the headgear group children were found to have significantly more apnea/hypopnea periods during the time the appliance was in use. At present, there is no clear evidence that using cervical headgear will change airway dimensions and, therefore, cervical headgear for non-OSA children is likely safe in this regard. However, using cervical headgear for children with OSA or for those considered high-risk of OSA requires additional caution.

10

10.3.4 Protraction Headgear for OSA

Protraction headgear has been used for Class III malocclusion with maxillary deficiency. Maxillary skeletal protraction, forward movement of the maxillary dentition, counterclockwise rotation of the palatal plane, labial tipping of the maxillary incisors, increase anterior face height, clockwise rotation of the mandible and lingual tipping of the lower incisors have all been shown as treatment effects with growing children [254–256]. (See Fig. 10.12).

Midface distraction osteogenesis has been used to alleviate upper airway obstruction from midface hypoplasia associated with various craniofacial anomalies [257–259].

Nguyen et al. compared airway volumes and minimum cross-section area changes of Class III patients treated with bone-anchored maxillary protraction versus untreated Class III controls [256]. Subjects treated with bone-anchored maxillary protraction showed an increase in airway volume and oropharyngeal dimensions.

Sayinsu et al. evaluated 19 Class III patients with RME and protraction headgear. The nasopharyngeal airway dimensions were increased [260]. Several studies report the similar results [261–263]. Conversely, Baccetti et al. reported that no significant changes in the sagittal oropharyngeal and nasopharyngeal airway dimensions were induced by protraction headgear [264].

Pamporakis et al. [265] evaluated 22 patients treated with protraction headgear after RME and evaluated with CBCT. They concluded that RME/FM treatment did not at all result in a changed pharyngeal airway volume, but inhibited the normal expected increase of the volume of the pharynx when compared with a control group comprised of normal individuals. Mucedero et al. [266] reported that even with positive orthopedic effect for Class III malocclusion, protraction headgear does not produce a significant increase in airway dimensions. At present, it is difficult to draw any solid conclusion whether protraction headgear has any appreciable effects on airway dimensions. Although a few studies do indeed demonstrate increased airway dimensions, it is still unclear if these changes translate into any improvement for OSA.

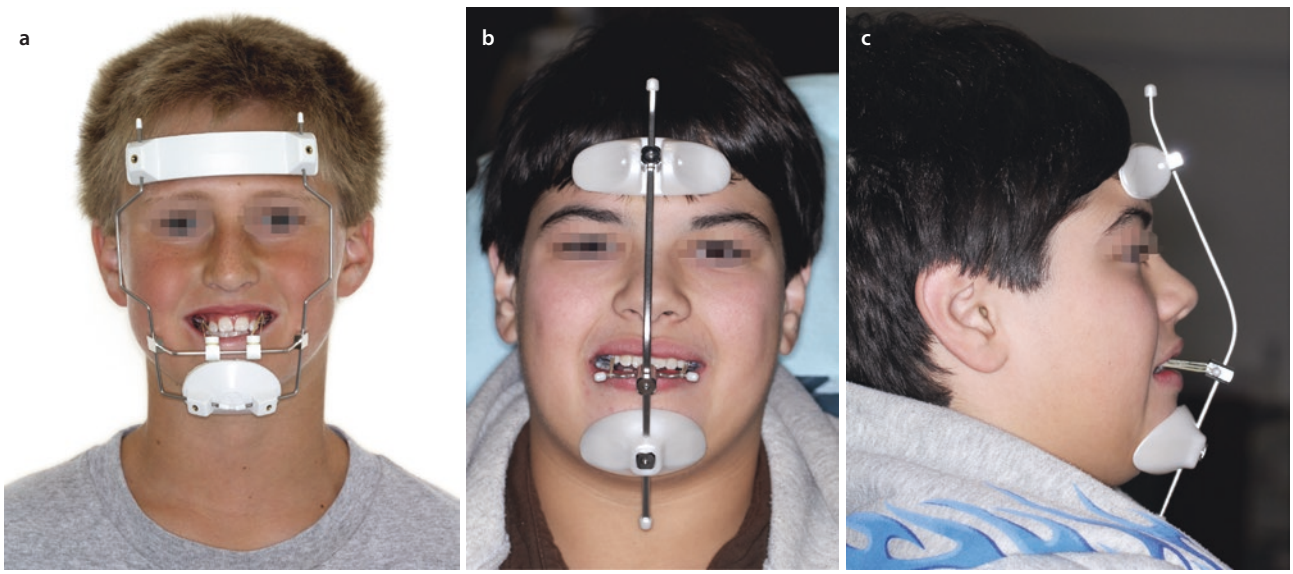


Fig. 10.12 Protraction headgear

Fig. 10.13 Chin cup

10.3.5 Chin Cup and OSA

Chin cup therapy has been used to control mandibular growth in patients with Class III malocclusion with prognathic mandible. This treatment's skeletal effects are primarily achieved by the restraining of mandibular forward growth with backward and downward rotation of mandibular plane [267–270]. (See Fig. 10.13).

Tuncer et al. [271] studied chin cup therapy to determine any adverse effects on the sagittal pharyngeal dimensions in Class III malocclusion patients by using cephalometric radiographs. Following chin cup treatment, no adverse effects were observed in pharyngeal airway dimension.

10.3.6 Functional Appliances Treatment for Class II Malocclusion and OSA

Functional appliances are a common treatment method for growing patients with retrognathic mandibles [272–275]. (See Fig. 10.14).

Several studies suggest functional appliances have a positive effect on upper airway dimensions [276–279]. Xiang et al. [276] report that functional appliance can enlarge the oropharyngeal airway dimension in children with skeletal Class II malocclusion. Maspero et al. [277] assessed the response and changes on pharyngeal airway to Class II functional appliance in Class II growing patients with OSA and showed increased pharyngeal airway dimensions. Ali et al. [279] showed that pharyngeal airway dimensions were increased after twin block appliance. Elfeky and Fayed [280] used twin block in Class II malocclusion patients and reported that the mean change of the oropharynx and nasopharynx in the twin block group was significantly higher than those in the control group. Ozbek et al. [281] also showed that pharyngeal airway dimensions increased significantly in the functional appliances group, especially those with sagittally smaller and more retrognathic maxillomandibular complexes and smaller airway dimensions. Jena et al. [282] reported that in comparison to the control group, the twin block groups demonstrated significantly higher mean changes in soft palate morphology and oropharynx depth.

Few studies show similar results with the twin block appliance. Oropharyngeal dimensions have been reported to be significantly increased [283, 284]. Temani et al. [278] reported that a Forsus-fixed functional appliance increased pharyngeal airway volume in skeletal Class II malocclusion adolescent patients with a retrognathic mandible. Bavbek et al. [285] also used a Forsus spring as a fixed functional appliance to evaluate airway dimensions and hyoid bone position in Class II malocclusion children. They reported that the fixed functional appliance group showed increased airway dimensions at soft palate and more forward positioning of the hyoid bone.

However, several studies showed there was no airway dimensional change [286–288]. One of these studies reported that dentoalveolar changes produced by the Forsus appliance did not result in any changes of the posterior airway [286]. Kinzinger et al. [287] found their pharyngeal airway space data dimensions to be unreliable, concluding that functional appliance treatment for the correction of Angle Class II malocclusion cannot be presumed to help prevent OSA.

Lin et al. [288] evaluated the pharyngeal airway dimension and the position of the hyoid bone after treatment with a functional, removable bionator. No changes in airway dimensions or changes in the vertical position of the hyoid bone were reported. Ulusoy et al. [289] evaluated the long-term effects of Class II functional appliances and found no significant difference in mean change of airway and skeletal parameters between control and functional appliances groups.

In their 2017 systematic review, Xiang et al. [276] concluded that evidence supported the notion that functional appliances can indeed enlarge upper airway dimensions in growing children with skeletal Class II malocclusion. When compared to the control group, oropharyngeal dimensions in treatment group subjects were significantly increased at the superior pharyngeal

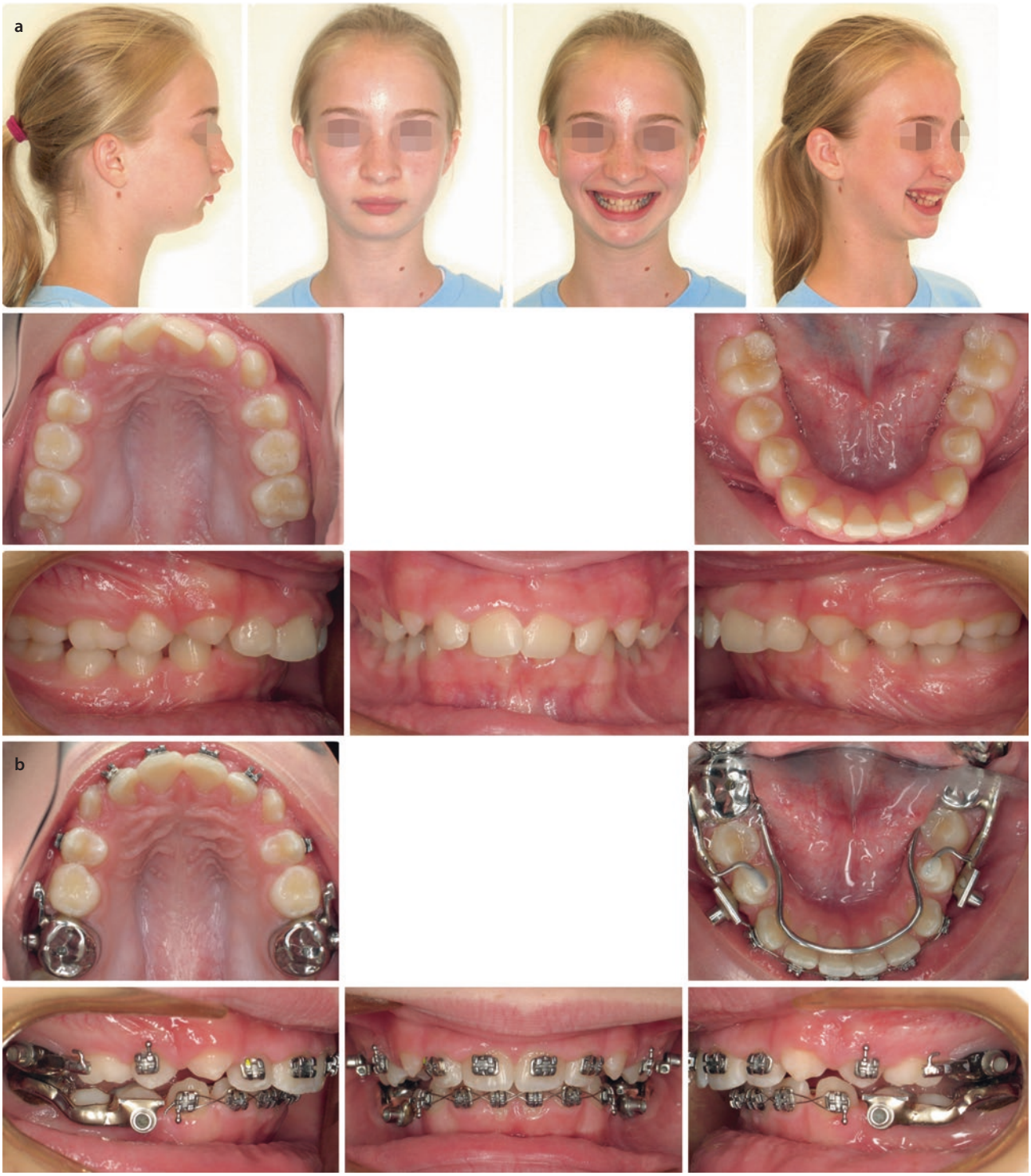


Fig. 10.14 Functional appliances. **a** Initial records, **b** Herbst appliance placed, **c** Herbst appliance removed, **d** Final records, **e** Initial cephalogram, f Cephalogram after Herbst appliance removed, **G** Final cephalogram

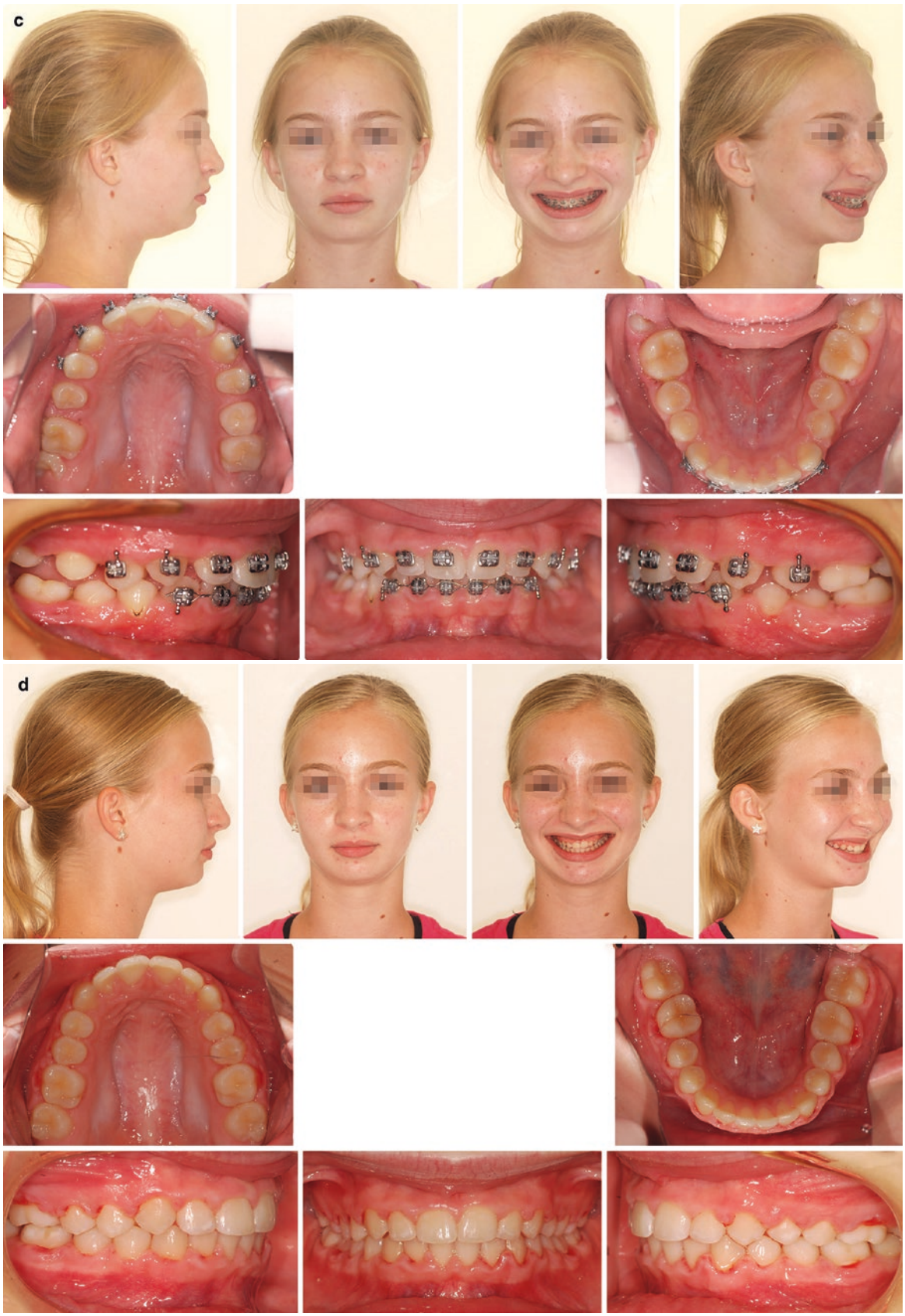


Fig. 10.14 (continued)

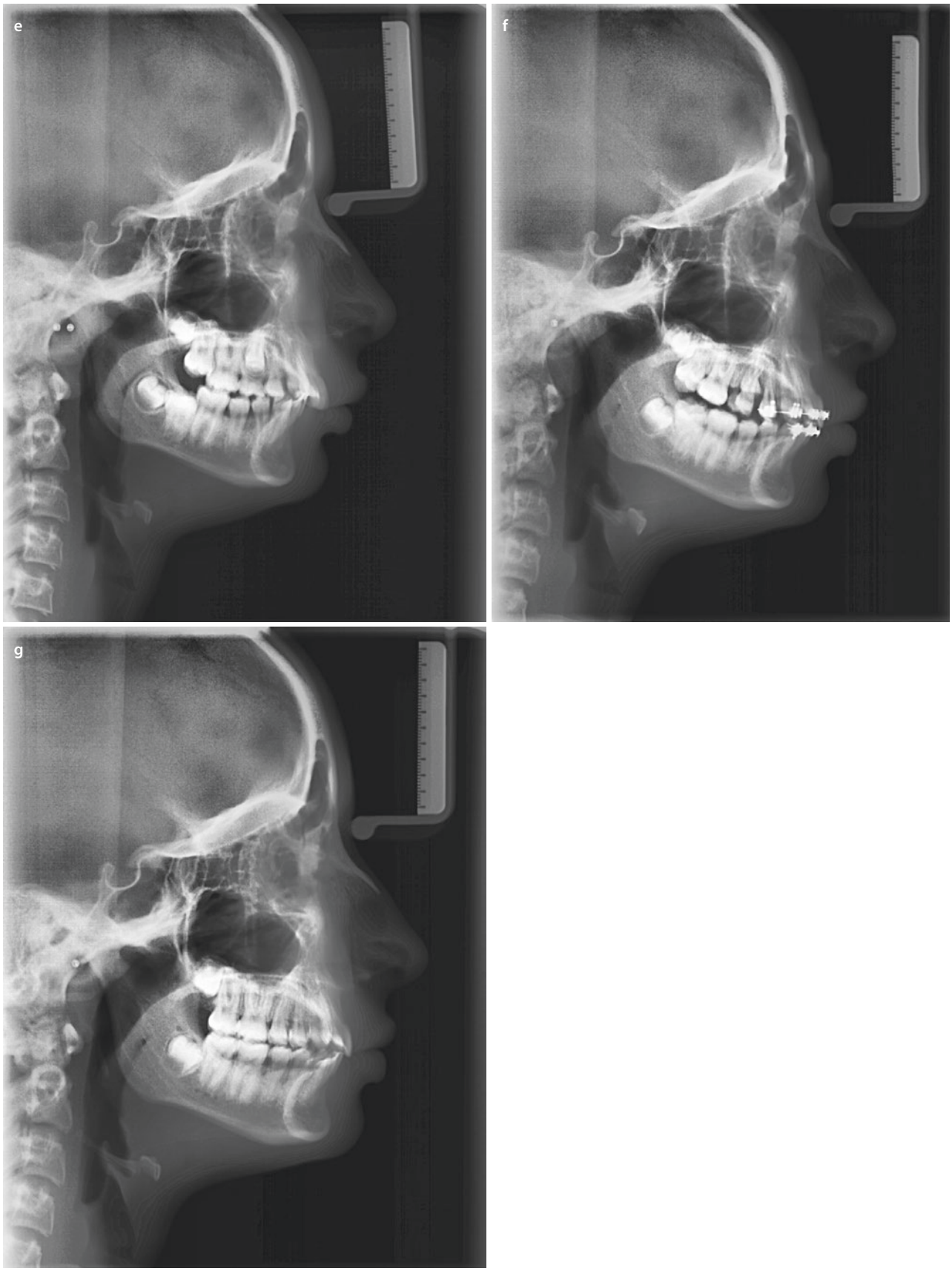


Fig. 10.14 (continued)

space (MD = 1.73 mm per year), middle pharyngeal space (MD = 1.68 mm per year), and inferior pharyngeal space (MD = 1.21 mm per year). No significant differences were found in nasopharyngeal and hypopharyngeal dimensions and the position of hyoid bone. Most studies focus only on airway dimensional changes before and after functional appliance treatment; very few studies addressed effects of functional appliances with children who were diagnosed with OSA. Schütz et al. [290] reported that a Herbst appliance coupled with RME resulted in improved OSA symptoms. Villa et al. [291] reported that children who used oral appliance showed decrease AHI.

Overall, even with the significantly increased airway dimensions following the use of functional appliances, data remain insufficient to support the use of functional appliances expressly for the treatment of children with OSA [292].

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