Management of Obstructive Sleep Apnea

An Evidence-Based, Multidisciplinary Textbook

Ki Beom Kim
Reza Movahed
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Principles and Fundamentals of OSA

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Classification of Sleep-Related Breathing Disorders

Katelyn Smith

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1

1.1 Introduction

The overarching term sleep-related breathing disorders (SRBDs) is utilized to collect the various conditions in which a person experiences an aberration of respiration, entirely or in part, during sleep. These disorders are divided into several categories and detailed in the International Classification of Sleep Disorders Third Edition (ICSD3). The primary classifications include obstructive sleep apnea disorders, central sleep apnea syndromes, sleep-related hypoventilation disorders, and sleep-related hypoxemia disorders [2]. Of note, The American Academy of Sleep Medicine Manual for the Scoring of Sleep and Associated Events describes the particulars of the specific respiratory events, which comprise these SRBDs, a portion of which will not be discussed here. Overall, the focus of this text will be on the broader categories of SRBDs.

1.2 Obstructive Sleep Apnea

1.2.1 Obstructive Sleep Apnea, Adult

The most prevalent of the sleep-related breathing disorders is obstructive sleep apnea (OSA) [9]. In OSA, there are partial and complete upper airway obstructions which occur in sleep. Obstructive hypopneas (Fig. 1.1) consist of an incomplete airflow reduction, whereas obstructive apneas (Fig. 1.2) are considered complete. Hypopneas are also associated with an oxyhemoglobin desaturation of at least 3% or 4% (depending of the definition used) and/or an arousal at the termination of the event. Additionally, respiratory effort-related arousals (RERAs) are episodes of airway obstruction in which there is flattening of the inspiratory flow signal and/or amplified respiratory effort, followed by an arousal, which do not fulfill the hypopnea or apnea definitions.

These three different obstructive events (apneas, hypopneas, and RERAs) all must last a minimum of 10 seconds in adults and co-occur with continued respiratory effort [4]. It is believed that these obstructive events share a fundamental pathophysiology, and all are thought to contribute to the symptoms and sequelae of OSA. Obstructions tend to be more severe during stage R sleep, particularly while in the supine position. Apneas, hypopneas, and RERAs are tallied and averaged per hour of sleep to arrive at the Respiratory Disturbance Index (RDI) [2]. With certain insurance companies and payers, RERAs are excluded, and the criteria for hypopneas are more stringent. In this case, the Apnea Hypopnea Index (AHI) is calculated, which excludes RERAs.

$$\text{RDI} = \frac{(\text{Apnea} + \text{Hypopnea} + \text{RERA}) \times 60}{\text{Total sleep time}}$$

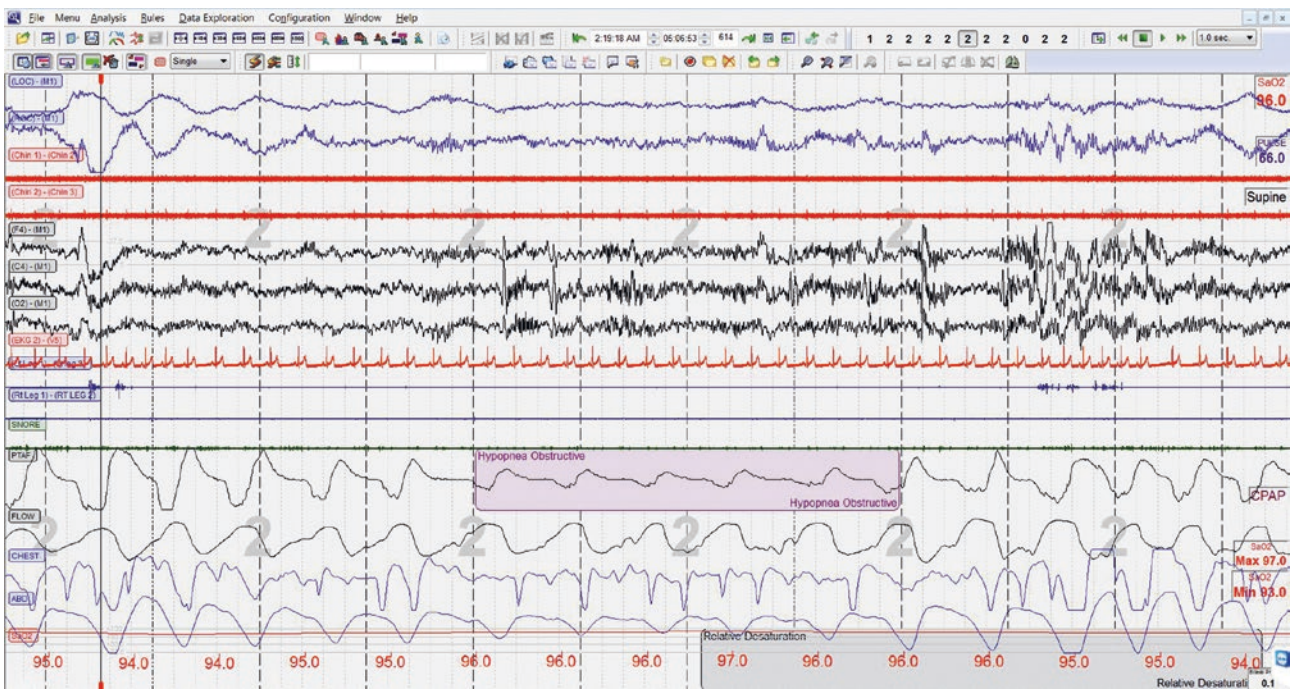

Fig. 1.1 This is a 60-second epoch showing an obstructive hypopnea meeting the criteria of at least a 30% drop in pressure signal from the baseline for at least 10 seconds with an associated 3% desaturation

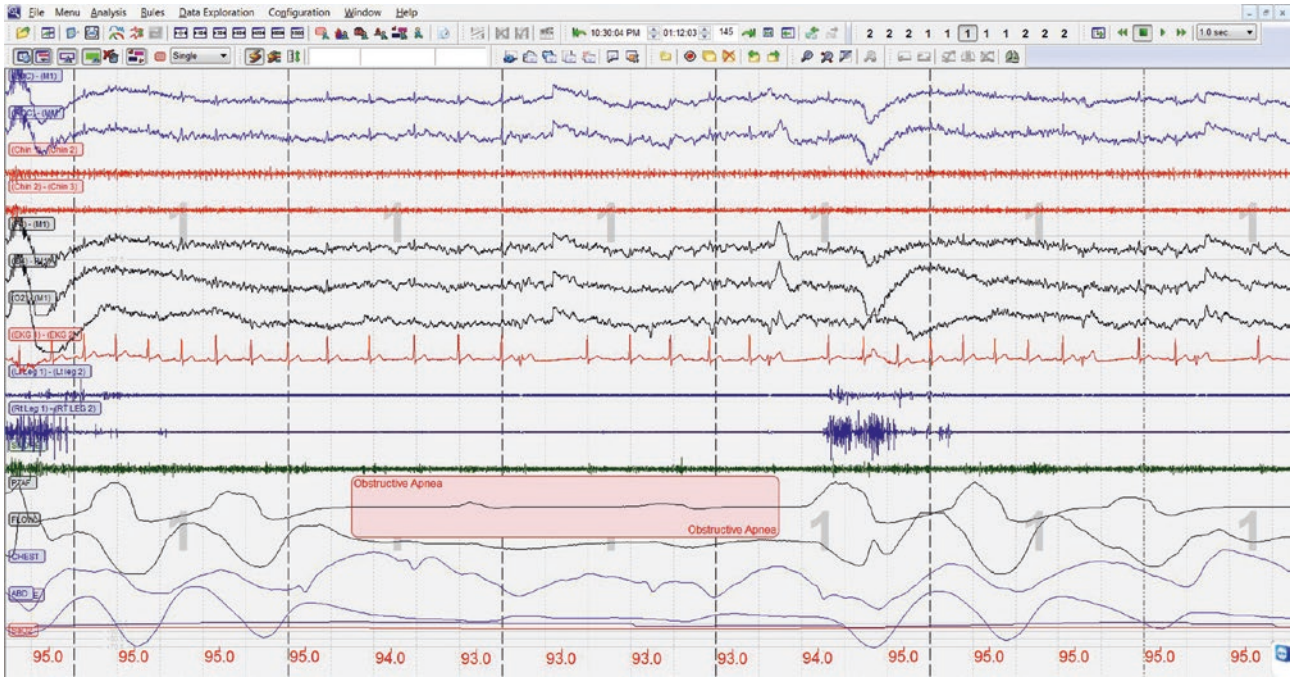

Fig. 1.2 This is a 60-second epoch showing an obstructive apnea meeting the criteria of at least a 90% drop in flow signal from the baseline for at least 10 seconds

$$\text{AHI} = \frac{(\text{Apnea} + \text{Hypopnea} + \text{RERA}) \times 60}{\text{Total sleep time}}$$

The RDI or AHI is then used to gage the severity of OSA. The severity classification for adults is as follows:

- Mild: RDI or AHI ≥ 5 and < 15
- Moderate: RDI or AHI ≥ 15 and < 30
- Severe: RDI or AHI ≥ 30 [9]

If the RDI or AHI is in the mild range (<15 events per hour), then there are other diagnostic criteria required in addition to polysomnographic findings to make the diagnosis of OSA. These criteria include one or more of the following:

1. Awakenings associated with gasping, choking, or breath holding
2. Witnessed repeated episodes of snoring or pauses in breathing
3. Comorbidities of mood disorder, cognitive dysfunction, hypertension, coronary artery disease, congestive heart failure, atrial fibrillation, stroke, or diabetes mellitus type 2
4. Complaints of fatigue, sleepiness, insomnia, or unrefreshing sleep

However, if the RDI or AHI reaches or exceeds 15, then the diagnosis is made even in the absence of additional signs, symptoms, and comorbidities [2].

Both in-lab polysomnography and home sleep apnea testing (HSAT) may be used to evaluate for OSA. While in-lab polysomnography is the firmly established gold-standard method of testing for sleep disorders, the HSAT may be adequate to evaluate for obstructive sleep apnea. There are important stipulations regarding which patients are appropriate for HSAT (discussed in ▶ Chap. 8).

Oxyhemoglobin desaturations may result from the obstructions and generally resolve after the obstructive event ends. However, if there are lengthy or particularly recurrent apneas and hypopneas or there is underlying pulmonology disease, these desaturations may persist. For example, it is not uncommon for OSA and COPD to co-occur, and this dual contribution from diverse pathologies can lead to more marked desaturations in sleep and hypercapnia in wake. Arousal may also result from obstructive events and are included in the diagnostic criteria of hypopneas and RERAs. The prevalence of obstructions may increase with the ingestion of alcohol and sedatives, as well as with weight gain [2].

1.2.2 Obstructive Sleep Apnea, Pediatric

There are several differences in the classification and diagnosis of Pediatric OSA and in the definition of obstructive events. Rather than having a 10-second

duration requirement, in children the duration of obstructive event must be at least the length of two breaths [4]. The use of HSAT has not been validated in children [14]. Diagnosis requires snoring, abnormal breathing (e.g., paradoxical), or daytime symptoms such as sleepiness or behavior issues.

Additionally, the child must have at least one obstructive event per hour of sleep or meet criteria for obstructive hypoventilation in addition to as associated feature (i.e., paradoxical breathing, snoring, or flattening of the inspiratory portion of the nasal pressure waveform). The ICSD3 describes this obstructive hypoventilation as spending at least 25% of the total sleep time with arterial carbon dioxide levels greater than 50 mm of Hg.

1.3 Central Sleep Apnea

Patients with central sleep apnea (CSA) often have multiple or even unknown contributing etiologies, which lead to the wide variety of disease entities which fall under the CSA classification. There are a few unifying themes within the fundamental elements. During the central event, there is near or complete cessation of airflow in tandem with an absence of respiratory effort (Fig. 1.3). Additionally, the central apneas tend to occur due to unstable central nervous system respiratory controller mechanisms [8].

The ICSD3 further subdivides CSA into the following categories for adults: Cheyne-Stokes breathing (CSB), CSA due to a medical condition without CSB, CSA due to high-altitude periodic breathing, CSA due to medication or substance, primary CSA, and treatment-emergent CSA. When considering the pediatric population, this list also includes primary CSA of prematurity and primary CSA of infancy.

To make the above adult diagnoses, clinical features must be present. For all but CSA due to high-altitude periodic breathing and the pediatric diagnoses, the average of central respiratory events must be at least 5 per hour, and the majority of the respiratory events must be central in nature. In general, the treatment options include treating the underlying conditions and PAP therapy [2].

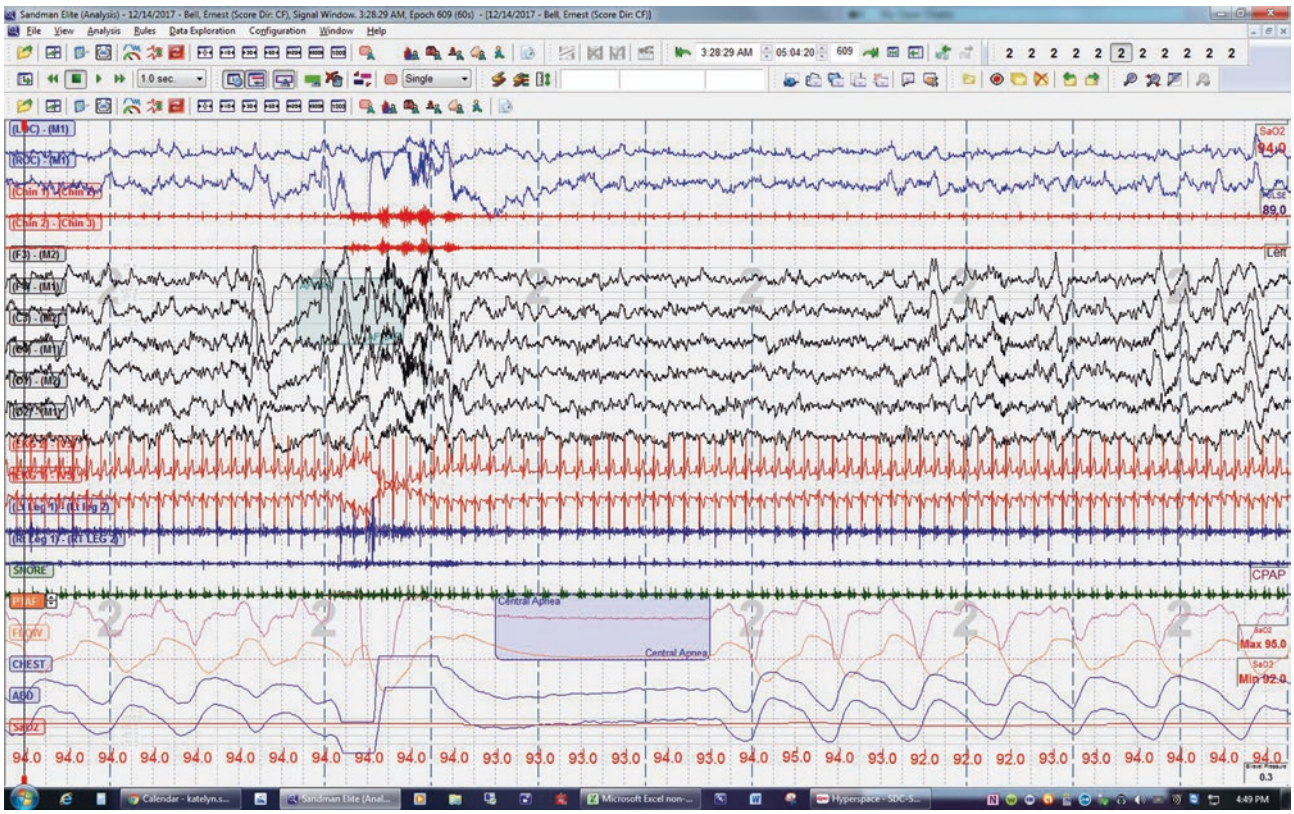

Fig. 1.3 This is a 60-second epoch showing a central apnea meeting the criteria of at least a 90% drop in the flow signal (orange waveform) for at least 10 seconds with a complete cessation of respiratory effort as displayed by the flattening of the chest and abdominal signals (blue waveform)

1.3.1 Central Sleep Apnea With Cheyne-Stokes Breathing

Central sleep apnea with Cheyne-Stokes breathing (CSA-CSB) is a subcategory of CSA in which periodic breathing is exhibited through a string of crescendo-decrescendo breathing episodes in between central apneas and/or hypopneas. CSA-CSB, as it is classified in the ICSD3, requires specific symptomatology (e.g., snoring, sleepiness, or witnessed apneas) and/or comorbidity including congestive heart failure, atrial flutter/fibrillation, or neurologic disorder [2, 16]. Additionally, the diagnosis requires that the disordered breathing is not better explained by another sleep disorder or medication or substance use. The polysomnogram should demonstrate at least five central breathing events per hour, the total number of which is >50% of the total apneic/hypopneic events along with the CBS pattern of ventilation. Heart failure and stroke may be underlying causes and should be evaluated for in a patient with a diagnosis of CSA-CSB [3, 12].

1.3.2 Central Sleep Apnea Due to a Medical Disorder Without Cheyne-Stokes Breathing

Central sleep apnea due to a medical disorder without Cheyne-Stokes breathing (CSA w/o CSB) lacks the CSB-patterned breathing but comprises all other forms of CSA thought to be caused by a medical diagnosis in adults. While these medical diagnoses may range from respiratory to cardiovascular to neurologic, the neurological causes cover the vast majority of etiologies. Brainstem lesions, stroke, and Chiari malformations are a few of the most common neurological causes [7]. The diagnostic criteria require symptomatology (i.e., snoring, sleepiness, awakening with dyspnea, difficulty sleeping, or witnessed apneas) if the patient is an adult and the absence of CSB on polysomnogram. The polysomnogram should display at least five central breathing events per hour, the total number of which is >50% of the total of apneic/hypopneic events. Additionally, the disordered breathing cannot be due to substance or medication use. Sleep-related hypoventilation may also be present but is not required [2].

1.3.3 Central Sleep Apnea Due to High-Altitude Periodic Breathing

Typically seen in altitudes of at least 2500 meters but also seen as low as 1500 meters, this is a classification of CSA wherein the body responds to a sudden increase in altitude with manifestation of symptoms and periodic breathing while asleep. The breathing pattern is comprised of hyperpnea alternating with central apnea in a cycle length between 12 and 40 seconds [2, 5].

1.3.4 Central Sleep Apnea Due to Medication or Substance

Central sleep apnea due to medication or substance is a secondary form of CSA caused by the use of a respiratory depressant substance or medication, most commonly an opioid. Morphine, oxycodone, fentanyl patches, narcotic infusions, and suboxone are possible culprits [2]. Opioids have multiple deleterious effects on breathing while asleep in that they suppress the respiratory drive at a central level, increase the likelihood of OSA by relaxing the patient's airway, and induce hypoventilation [10].

1.3.5 Primary Central Sleep Apnea

Primary central sleep apnea is rare and characterized by the lack of other differentiating features. To establish this diagnosis, there must not be Cheyne-Stokes breathing, daytime or nocturnal evidence of hypoventilation, or the existence of a causal medical condition or use of medication.

1.3.6 Treatment-Emergent Central Sleep Apnea

Treatment-emergent central sleep apnea (TE-CSA) and complex sleep apnea both describe the same clinical phenomenon; this occurs when a patient displays CSA while using a positive airway pressure (PAP) without a backup rate to treat established OSA. A high number of arousals persist on PAP treatment along with an AHI that is higher during NREM than REM. Patients on opioids are more likely to manifest TE-CSA than opioid naive patients [13]. While some consider TE-CSA merely a manifestation of OSA, the ICSD3 includes TE-CSA as a discrete form of CSA in its diagnostic criteria. In the available literature, TE-CSA is found in up to 20% of patients with OSA who have initiated PAP therapy. Fortunately, as treatment with PAP continues this percentage drops to around 2% [2].

1.4 Sleep-Related Hypoventilation Disorders

In the disorders of sleep-related hypoventilation (SRHV), the essential element is a sustained aberrantly elevated arterial partial pressure of carbon dioxide while asleep. There are two scenarios that would qualify an adult as having sleep-related hypoventilation, the first of which is arterial carbon dioxide (CO_2) levels of greater than 55 mmHg for at least 10 minutes of sleep. The second possible scenario is an increase of at least 10 mmHg in sleep when compared to supine wake if it exceeds 50 mmHg for at least 10 minutes. In children, sleep-related hypoventilation is defined as arterial carbon dioxide levels of greater than 50 mmgHg [2] for at least 25% of the total sleep time. Carbon dioxide may be monitored through end-tidal CO_2, transcutaneous PCO_2, or ABG [4].

The ICSD-3 distinguishes six separate subclassifications. The adult subtypes include obesity hypoventilation syndrome, idiopathic central alveolar hypoventilation, disorders due to another medical disorder, and disorders due to a medication or substance. In children, the subtypes include congenital central alveolar hypoventilation syndrome and late-onset central hypoventilation with hypothalamic dysfunction [2]. While all the SRHV disorders may have hypoventilation during wake, only obesity hypoventilation disorder requires it for diagnosis [15]. If hypoventilation is present in wake, it will likely worsen in sleep. Briefly, the foundation of SRHV disorders treatment is directed toward the particular underlying etiology and often hinges on positive airway pressure therapy.

1.5 Sleep-Related Hypoxemia Disorder

The defining characteristic of this classification is the disordered systemic hypoxemia which is not secondary to hypoventilation. Hypoxemia is diagnosed through PSG, HSAT, or continuous oximetry during sleep. The differentiation between sleep-related hypoventilation and sleep-related hypoxemia depends upon the ability to assess for nocturnal hypoventilation by monitoring CO_2. To qualify as sleep-related hypoxemia disorder, the arterial oxygen saturation needs to be 88% or less for at least 5 minutes in adults. In children, the arterial oxygen saturation must be 90% or less for at least 5 minutes. If the hypoxemia can be solely attributed to OSA or CSA, it excludes the diagnosis of sleep-related hypoxemia or hypoventilation. Conversely, while OSA and CSA may be present, they do not necessarily preclude the diagnosis of sleep-related hypoxia disorder provided they are not associated with the majority of the sleep time during which hypoxemia is present. Conditions that may be responsible for sleep-related hypoxemia include venous shunting, ventilation-perfusion mismatch, diffusion abnormalities, low atmospheric PO_2, and increased dead space [2, 6].

1.6 Mixed Disorders

Central apneas and hypopneas may occur along with the obstructive events. Generally, if the central events are few and there is a relative preponderance of obstructive events, then the individual is still considered to have OSA. However, there are those whose polysomnograms meet criteria for both obstructive and central sleep apnea; therefore, they are considered to have a mixed disorder [2].

1.7 Isolated Symptoms

1.7.1 Snoring

Essentially, snoring is a sound produced by respiration while sleeping, most often in the inspiratory phase. A designation of simple or primary snoring is given in the absence of apnea or hypoventilation and when there is no associated sleep disturbance or daytime sleepiness. While sporadic snoring is extremely common, persistent nocturnal snoring is less common and should be considered a potential symptom of OSA. Additionally, the ICSD recommends PSG or HSAT for all patients with cardiovascular disease who snore [2]. Adult men snore more commonly than any other affected group, and there is a strong correlation between snoring and obesity [11]. In children who snore, adenotonsillar hypertrophy is strongly implicated. Smoking, alcohol, opiates, and muscle relaxants can all increase the likelihood of snoring [2].

1.7.2 Catathrenia

Catathrenia is the term used to describe a vocalization in sleep during a prolonged expiration, typically in REM sleep. It is also referred to as sleep-related groaning. The clinical significance of catathrenia is debated in the literature, and currently the phenomenon is seen chiefly as a social problem rather than a medical concern [1, 2].

1.8 Summary

The sleep-related breathing disorders are a collection of several conditions, all of which include dysfunctional respiration during sleep. The primary classifications include obstructive sleep apnea disorders, central sleep

apnea syndromes, sleep-related hypoventilation disorders, and sleep-related hypoxemia disorders. These classifications are further broken down into subcategories. The initial treatment of sleep-related hypoxemia and many of the central sleep apnea disorders and sleep-related hypoventilation disorders requires treatment of the underlying conditions. One patient may have multiple SRBDs, and, in these cases, each disorder should be addressed.

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Pathophysiology of Obstructive Sleep Apnea (OSA)

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2

2.1 History and Introduction

Prior to 1978, investigations related to obstructive sleep apnea pathogenesis focused on obesity-hypoventilation syndrome (OHS). At the time, OHS was a disorder associated with many theories of pathogenesis, but there was little strong support for any of them. In the mid- to early 1960s, Gastaut et al. were the first to describe obstructive sleep apnea in these patients [16, 17], and over the ensuing years multiple reports appeared demonstrating the successful reversal of OHS after treatment of obstructive sleep apnea [50, 51, 66]. (Hereafter, OSA will be used to represent the entire spectrum of sleep-related obstructive events: apneas, hypopneas, and respiratory effort-related arousals.) A landmark study published in 1978 by Remmers et al., demonstrated that OSA events were accompanied by a closed upper airway and that negative pharyngeal pressure behind the tongue prevented forward tongue movement causing, in essence, asphyxia. This continued until an arousal-mediated recruitment of the airway dilator muscles opened the airway, with a surge in anterograde tongue activity associated with the arousal [52]. These findings changed the focus of physiology research to examine the pathogenesis of OSA with or without OHS: to attempt to understand why the pharynx is narrowed, how this affects the flow of air, circumstances of activation of pharyngeal muscles, and mechanism of arousals [72].

OSA is characterized by episodes of partial or complete collapse of the airway, with an associated decrease in oxyhemoglobin saturation and/or an arousal that causes the individual to reopen the airway, resume normal (or even hyperpneic) ventilation, and (in patients without OHS) return to eucapnia and baseline oxygenation. Understanding the mechanisms causing this collapse is key to preventing and treating OSA. Moreover, the cycle of airway obstruction and arousal results in poor sleep consolidation, often with loud or disruptive snoring and, in many individuals, excessive daytime sleepiness.

2.2 Airway Collapsibility and Pcrit (Fig. 2.1)

Our understanding of the pathophysiology of OSA advanced significantly with the application of a flow limitation theory known as the Starling resistor model – the physics of collapsible tubes surrounded by an environment characterized by varying pressures (represented conventionally as a box within which the collapsible segment resides) and connected proximally and distally to rigid tubes, both of which are also subject to varying pressures [12]. Flow limitation was used to explain the shape of the forced expiratory spirogram at low lung volumes [14] and the effect of mass and gravity on flow through the pulmonary vasculature in the three lung zones defined by West [47, 68]. With respect to obstructive sleep-disordered breathing events – apneas (OAs), hypopneas (OHs), respiratory effort-related arousals (RERAs), and primary snoring – the Starling model of the upper airway in combination with upper airway anatomy and unstable ventilatory control admirably explain most, but not all, aspects of OSA pathogenesis and treatment. When applied to OSA, the oropharynx is modeled as the collapsible tube surrounded by the complex environment of the remaining tissues of the neck. The proximal rigid tube consists of the nose and mouth, while the distal rigid tube is considered to be the supraglottis, glottis, and subglottis (collectively, the larynx) and the trachea. There are obvious pitfalls to considering the larynx to be a rigid tube given the presence of the epiglottis and vocal cords, but notwithstanding the potential variations from rigidity that these structures introduce, the model serves in most cases. Absent significant nasal obstruction or the application of positive airway pressure, the pressure within the proximal or "upstream" segment, plays little or no role, leaving the distal or "downstream" pressure and the resistance to collapse of the pharynx and the pressure exerted on the pharynx by the surrounding tissues (the pressure within the "box") as the major players in determining pharyngeal patency. The pressure exerted by the "box" surrounding the collapsible segment is generally a matter of anatomy, while a complex combination of upper airway muscle activity collectively acting as pharyngeal dilators determines the resistance of the pharynx to collapse. Completing the model, the pressure within the downstream segment also plays a major role. During inspiration, this pressure is negative relative to ambient pressure, pulling air into the lungs but also exerting a negative pressure on the collapsible pharyngeal segment. In a sleeping individual without any degree of obstructive sleep-disordered breathing (including snoring), the action of the upper airway dilator muscles is coordinated with inspiration so as to completely counteract the forces acting to collapse the pharynx (the downstream negative pressure and the pressure exerted by the surrounding tissues), and breathing is not impeded. Disruption of any aspect of this finely tuned system can, and does, result in the spectrum of abnormal breathing seen in obstructive sleep-disordered breathing.

As is the case with any attempt at modeling a physiologic function, the Starling model of the upper airway is not perfect. As has already been alluded to, the epiglottis or vocal cords may not in some cases function as rigid conduits during inspiration. There are, in fact, examples of OSA with obstruction at the epiglottal level [62] and disorders, such as multiple system atrophy, that

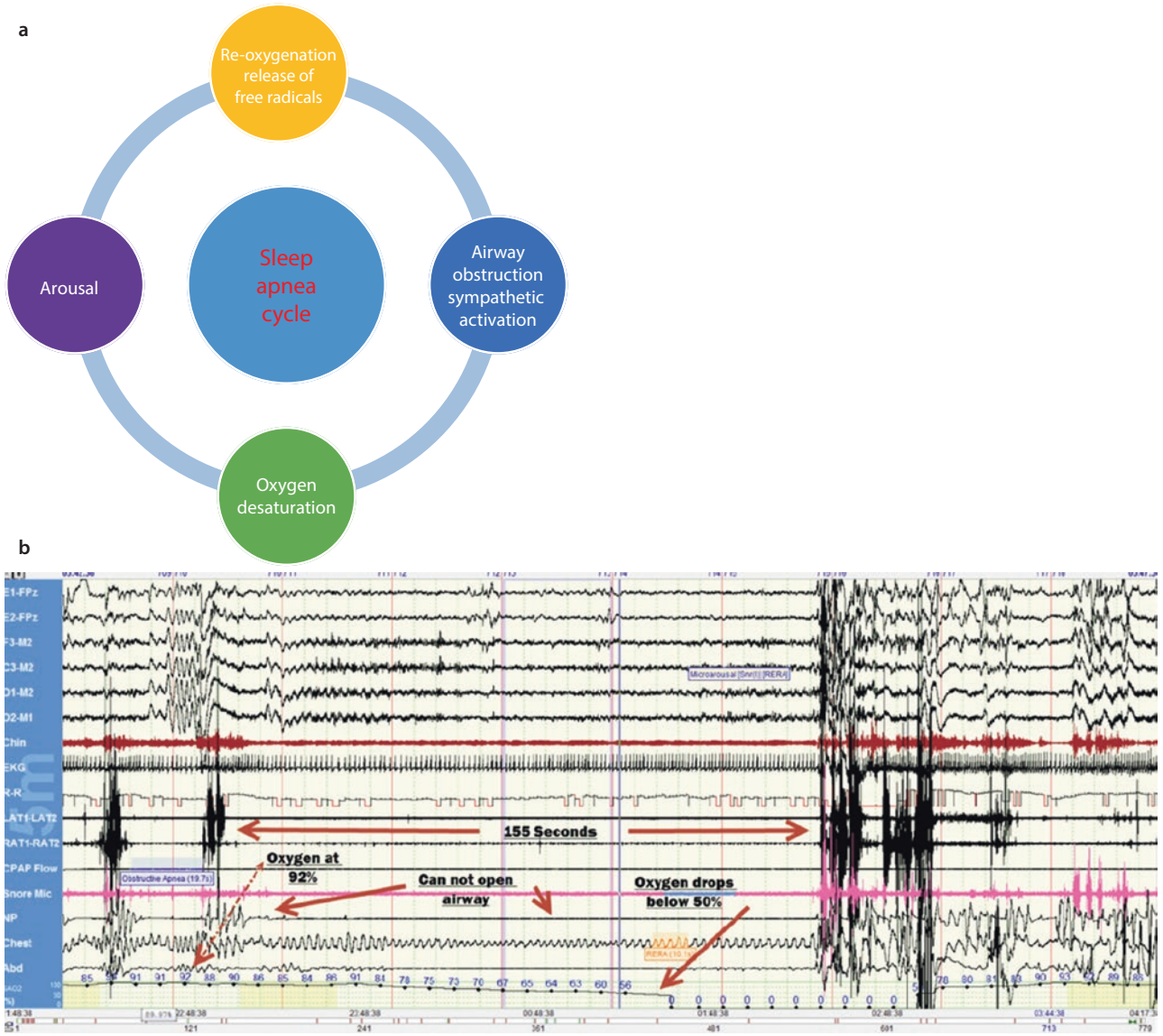

Fig. 2.1 **a** Sleep apnea typically pursues a cycle of obstruction, oxygen desaturation, arousal, and reoxygenation. When airway obstruction occurs, the body enters into the "fight or flight" response with activation of the sympathetic system resulting in increases of cortisol, blood pressure, and heart rate. Frequent arousals from sleep impair sleep consolidation and many patients will complain of excessive daytime sleepiness or fatigue. **b** A respiratory event must be at least 10 seconds long to be scored as an apnea or hypopnea. The duration of apneic events varies greatly among individuals. In the above example, the apneic event lasts 155 seconds and the oxygen saturation starts at 92% and drops below 50% following the apneic event. An arousal eventually occurs allowing the patient to breathe and re-oxygenate

result in OSA due to failure of the vocal cords to abduct during inspiration [29]. In addition, Owens and colleagues have demonstrated that, in some patients, negative effort dependence (increasingly negative downstream pressure) can produce an initial small peak in the expected flat inspiratory flow pattern predicted by strict adherence to the Starling model [43].

The collapsibility of the airway is characterized by the pharyngeal critical closing pressure (P_{crit}) [2, 24, 48]. Early experiments by Gleadhill et al. demonstrated that application of nasal negative and positive airway pressures could distinguish between asymptomatic snorers, patients with predominantly OHs, and patients with predominantly OAs. In this experiment, pressures of -6.5 ± -2.7 cm H_2O, -1.6 ± 1.4, and 2.5 ± 1.5 cm H_2O, respectively (mean ± SD, $p < 0.001$), correlated with the point at which inspiratory airflow completely ceased, thereby identifying the value of P_{crit} [19]. The same group had earlier shown that OSA could be induced in normal individuals by applying even greater degrees of negative nasal pressure [56]. It can be concluded, therefore, that whatever factors determine the value of P_{crit} are the fac-

tors that help determine whether an individual will enjoy normal breathing during sleep, snore, or exhibit varying severities of OSA with respect to the proportion of OHs and OAs. A variety of factors have been demonstrated to increase P_{crit} above and beyond anatomic upper airway narrowing and ventilatory instability/dilator muscle tone. These include craniofacial characteristics [54, 60], obesity [18], age [13], head position [67], mouth opening [38], and sleep fragmentation [57]. However, none of these factors alone appear to be key in the pathogenesis of OSA. As will be discussed below, additional considerations that are likely involved in OSA pathogenesis can be divided into those based on anatomy (including obesity) and/or those associated with instability of ventilatory control.

2.3 Instability of Ventilatory Control During Sleep

Control of breathing represents an example of a negative feedback control system, about which much knowledge has been developed by engineers and other physical scientists beginning with James Clerk Maxwell's analysis of instability in a common mechanical system then in common use [36]. The primary purpose of such a system in humans is to minimize deviations in blood gases from desired levels. The respiratory centers in the brainstem, along with inputs from other more cephalad CNS sites, alter the drive to the inspiratory muscles (e.g., the diaphragm) in proportion to, but opposite in direction from, changes in $PaCO_2$ and PaO_2 that differ from a value (the set points) that are consistent with optimal function of the organism [11]. The difference between the optimal values of these variables and the actual values at a given point in time is known as the "error signal" and is used by the controller as effected by the overall gain of the system to govern the magnitude of drive to the respiratory muscles. Systems theory dictates that the respiratory negative feedback control system will become unstable under certain conditions, hunting back and forth over a range of outputs and establishing periodic variations in ventilation, up to and including complete cessation (apnea). Important attributes of such a control system are described as the "gain" of the whole system or some of its parts:

1. Controller gain, defined as the response of the controller's output per unit change in $PaCO_2$ or PaO_2
2. Plant gain, the change in $PaCO_2$ or PaO_2 per unit change in ventilation
3. Loop gain, which represents the product of controller gain and plant gain

A control system becomes unstable (hunts back and forth above and below the set point, resulting in periodic breathing) if one or more of the following conditions exist:

1. Controller or plant gain is excessive or is nonlinear.
2. Controller gain changes recurrently.
3. There is excessive time delay between when a controlled parameter changes to when that information is received by the controller.
4. The set point periodically changes.
5. Underdamping. Damping is a mechanism that counteracts the tendency of a system to oscillate when perturbed. A physical example would be the dampening action of shock absorbers in an automobile suspension, which counteract the tendency of the automobile to keep bouncing after traversing a pothole by absorbing the energy transferred to the suspension.

One or more of these factors are known to be involved in the pathogenesis of central sleep apnea and Hunter-Cheyne-Stokes ventilation. However, the situation with respect to ventilatory control instability and the pathogenesis of OSA is considerably more complicated. Respiratory control is an example of a multiple input/multiple output system (MIMO). The importance of this concept is magnified in the case of OSA due to a control function not necessarily related to the CNS controller, that of local reflex control of upper airway dilator muscle tone, and one additional output from the controller, that to the upper airway dilator muscles. Consequently, the factors outlined above that are key with respect to central sleep apnea pathogenesis have only a contributory role with respect to OSA pathogenesis.

White and Younes have comprehensively (and exhaustively) reviewed the multitude of upper airway muscles responsible for upper airway tone and caliber, the details of which are too extensive to be presented here [69]. Suffice it to say that muscles or groups of muscles are responsible for tongue position and shape, palatal position and shape, hyoid bone position, and pharyngeal constriction. These muscles are responsible not only for the caliber of the upper airway but also can act to change the compliance of the pharynx and therefore affect P_{crit}, as explained by the Starling model referenced above. One aspect of dysfunctional ventilatory control concerns upper airway dilator muscle reflex response to occlusion. In patients with OSA, a substantial increase in genioglossus (GG) muscle activity during apneas and hypopneas usually fails to restore normal airflow. A study that compared GG muscle and

non-GG muscle (styloglossus, geniohyoid, sternohyoid, and sternomastoid) EMG activity in patients with OSA demonstrated that, during wakefulness, flow limitation triggered increases in GG and non-GG muscles to the same degree. During sleep, however, flow limitation affected the GG much more than the non-GG muscles: flow limitation increased the GG EMG more than two-fold the level observed during wakefulness and the non-GG EMG on average only about 2/3 the wakefulness level [42]. The explanation for this finding and that of the effects of transitioning from wakefulness to sleep on the response of many of the upper airway dilator muscles to flow limitation largely involves local reflexes that counteract negative pressure. As explained by the Starling model, flow limitation occurs when negative pressure fails to increase flow; the negative pressure developed during inspiration would normally cause reflex increase in upper airway dilator tone but is presumably blunted during sleep in some, but not necessarily all, patients with OSA. However, as elegantly outlined by Younes in a recent commentary, this cannot represent the only explanation for the inability of the upper airway to open fully during an obstructive event, and therefore some degree of heterogeneity exists in the phenotypes of OSA and future approaches to treatment [72].

In addition to reflex control, upper airway dilator muscles receive innervation that in part derives from the brainstem respiratory controller. For instance, indirect evidence (in dogs) strongly suggests that the neurons in the hypoglossal nucleus responsible for tongue protrusion by the GG muscle activate synchronously with inspiration and that this activation is not a local reflex but rather under central control [65, 69]. In fact, investigators have demonstrated that GG tone increases 50–100 msec prior to activation of the diaphragm and initiation of inspiratory airflow, eliminating the likelihood that the finding could be related to a local reflex. Moreover, GG tone increases in response to hypercapnia and hypoxia [45, 65, 69]. Significantly, GG tone initially decreases at sleep onset, and this effect may occur to a larger degree in patients with OSA, although the effect of sleep on upper airway muscle tone has been better demonstrated with respect to other muscles, e.g., the tensor palatine [39]. The effect of sleep on GG tone diminishes as sleep progresses [3]. It has been hypothesized that the return of GG tone during sleep in normal individuals is related to a delayed onset of local reflex reaction to negative pressure and the increase in $PaCO_2$ due to the upward shift of the $PaCO_2$ set point known to occur during sleep [69]. As outlined above, the failure of this reflex during sleep in patients with OSA represents yet another avenue for pathogenesis of this disorder. Finally, while this discussion has largely been focused on the GG, similar findings, although some quite scant, have been published concerning other muscles involved in upper airway dilator muscle activity [69].

Interestingly, it has long been known that (at least in cats) neural drive to the diaphragm and that to the upper airway dilator muscles may not respond synchronously and in parallel to certain stimuli. For instance, Haxhiu et al. demonstrated that GG and posterior cricoarytenoid muscle EMG activity did not increase in proportion to increases in $FICO_2$ compared to the diaphragmatic EMG until a measured threshold of $FICO_2$ was reached [22]. This may, in part, relate to the findings of Iber et al. demonstrating a possible mechanism for the appearance of mixed apneas [27].

Finally, it is necessary to address the role of arousal in destabilizing ventilatory control and playing a role in OSA pathogenesis. Arousal-terminating obstructive events have been linked to the degree of negative pharyngeal pressure in combination with the arousal threshold in any given patient [20]. Arousal (which may even be subcortical or so subtle as to be questionable as to whether a real arousal took place, and not evident on polysomnography) recruits the upper airway dilating muscles, elicits compensatory ventilation or hyperventilation, and is followed by a return to sleep. The alternation between sleep and arousal is, in and of itself, a manifestation of respiratory control instability. However, in addition to the arousal threshold, there are a multitude of factors that govern when arousal will occur, and most are independent of the factors that are known to destabilize a feedback control system (e.g., aberrations in circulation time, plant gain, or controller gain). As elucidated by Younes, obstructive event termination is determined by which is lower: the negative pressure sufficient to recruit the upper airway dilator muscles without an evident arousal (which may be mainly dependent on upper airway muscle reflex control or on brainstem controller output) or the amount of respiratory controller output sufficient to stimulate a detectable arousal accompanied by recruitment of the upper airway dilator muscles [72]. Adding complexity is the fact that lung-carotid circulation time results in communication of arterial blood gas values that were present just before event termination and these values may continue to worsen as far as the central controller is aware, even after upper airway patency is restored. Consequently, a degree of hyperventilation may ensue that is unnecessary, governed by loop gain, and further destabilizes ventilatory control.

2.4 Anatomical Factors (Fig. 2.2)

Several predisposing anatomical factors affect the P_{crit}, promoting upper airway obstruction during sleep that leads to OSA. These include obesity, a prominent uvula/soft palate, tonsillar hypertrophy, macroglossia, retrognathia, thickness of the lateral pharyngeal muscle, pharyngeal length, tongue base, and the parapharyngeal fat pad. In obese patients with OSA, the enlarged volume of the parapharyngeal fat pad results in a concentric type of retropalatal obstruction [18, 31]. In addition, obesity reduces total lung capacity and consequently has been shown to diminish "tracheal tug," a mechanism by which the upper airway is held open [63]. Pediatric patients are unique in that OSA may be caused primarily by adenotonsillar obstruction of the airway particularly in patients of primary school age wherein tonsillar size is large compared to the total size of the pharyngeal lumen.

Common risk factors for OSA include obesity, age, regional fat distribution, skin-fat fold thickness, male gender, and neck circumference (NC) of more than 41 cm for females and 43 cm for males. The predictive value of NC is highest in middle-aged patients with OSA. NC is more strongly predictive of OSA than waist circumference, waist-to-hip ratio, or BMI. The predictive value of neck circumference was significantly lower for younger and older patients with OSA [34]. Many obese adolescents do not develop OSA due to vigorous upper airway neuromuscular responses during sleep. Upper airway reflexes normally decline during adolescent development [26]. In overweight and obese children, body fat distribution as described by neck-to-abdominal-fat percentage (NAF% ratio) predicts OSA. A cross-sectional retrospective study at a tertiary children's hospital evaluated 30 children aged 6–18, 24 of whom had a BMI >99th percentile, and 10 of whom had an apnea hypopnea index (AHI) >5 which is considered moderate severity of OSA in children. NAF% ratio was an independent predictor of OSA severity among overweight and obese children except in those with extreme obesity (BMI >99th percentile) [21]. A study that compared tongue fat in 30 obese patients without OSA to 90 obese patients with an average AHI of 43 demonstrated that in those with sleep apnea, there is a significantly increased deposition of fat at the base of the tongue compared to controls [34]. Common sites of the airway that are prone to collapse and precipitate apneic episodes are the tip of the soft palate and the base of the tongue. In obese patients, increased fat deposition at these sites significantly increases the likelihood of severe OSA [8, 35]. Furthermore, lung volume reduction due to excessive central fat deposition may decrease longitudinal tracheal traction forces as noted above, not only reducing upper airway caliber but also affecting pharyngeal wall tension [30].

Additional contributions to abnormal upper airway anatomy have been described that most likely also play a role in OSA pathogenesis. These consist of upper airway tissue injury and upper airway edema. With respect to the former, multiple studies have demonstrated patterns of injury to airway mucosae as well as upper airway dilator muscles that are attributable to vibratory injury from snoring and, possibly, recurrent closure and opening of the airway. These include inflammatory changes with recruitment of leukocytes [46] and changes in the proportion of GG type I vs. types IIa and IIb muscle fibers, with somewhat different findings in patients with OSA vs. simple snoring [58]; pharyngeal wall edema of the lamina propria, mucous gland hypertrophy, and focal squamous metaplasia, as well as muscle fiber atrophy and infiltration of mucous glands [70]; upper airway muscle inflammation and denervation [6]; increased upper airway muscle edema as measured by magnetic resonance imaging [55]; upper airway sensory impairment [41]; and dysfunctional mechanical coupling of upper airway muscle, presumably as a consequence of injury and denervation [59].

The issue of upper airway edema most frequently is cited in the literature concerning OSA in heart failure and chronic kidney disease (CKD). Most often, it takes the form of progressive changes in the phenotype of sleep-disordered breathing as the night progresses in these patients. These patients are commonly fluid-overloaded, and, when they sleep in a recumbent position, extracellular fluid that has collected in the lower extremities makes its way cephalad, causing upper airway edema and transitioning what may be primarily a central sleep apnea phenotype to that of mixed central sleep apnea and OSA or worsening preexisting OSA [7, 15, 53]. This phenomenon has also been used to justify more intensive dialysis, including nocturnal home dialysis, in patients with end stage renal disease [4].

2.5 Gender, Genetics, and Pathogenesis (Fig. 2.3)

Significant differences exist between men and women in OSA. A study of 180 adult patients with OSA, 144 males and 36 females, demonstrated that an increase in severity of OSA in men was significantly correlated with body mass index (BMI), a higher accumulation of adipose tissue in the upper part of the body as measured by NC, and shoulder thickness of skin-fat folds, whereas in females severity was only correlated with BMI [71]. A study of 858 males and 174 females demonstrated that BMI, waist circumference, and overall body fat were sig-

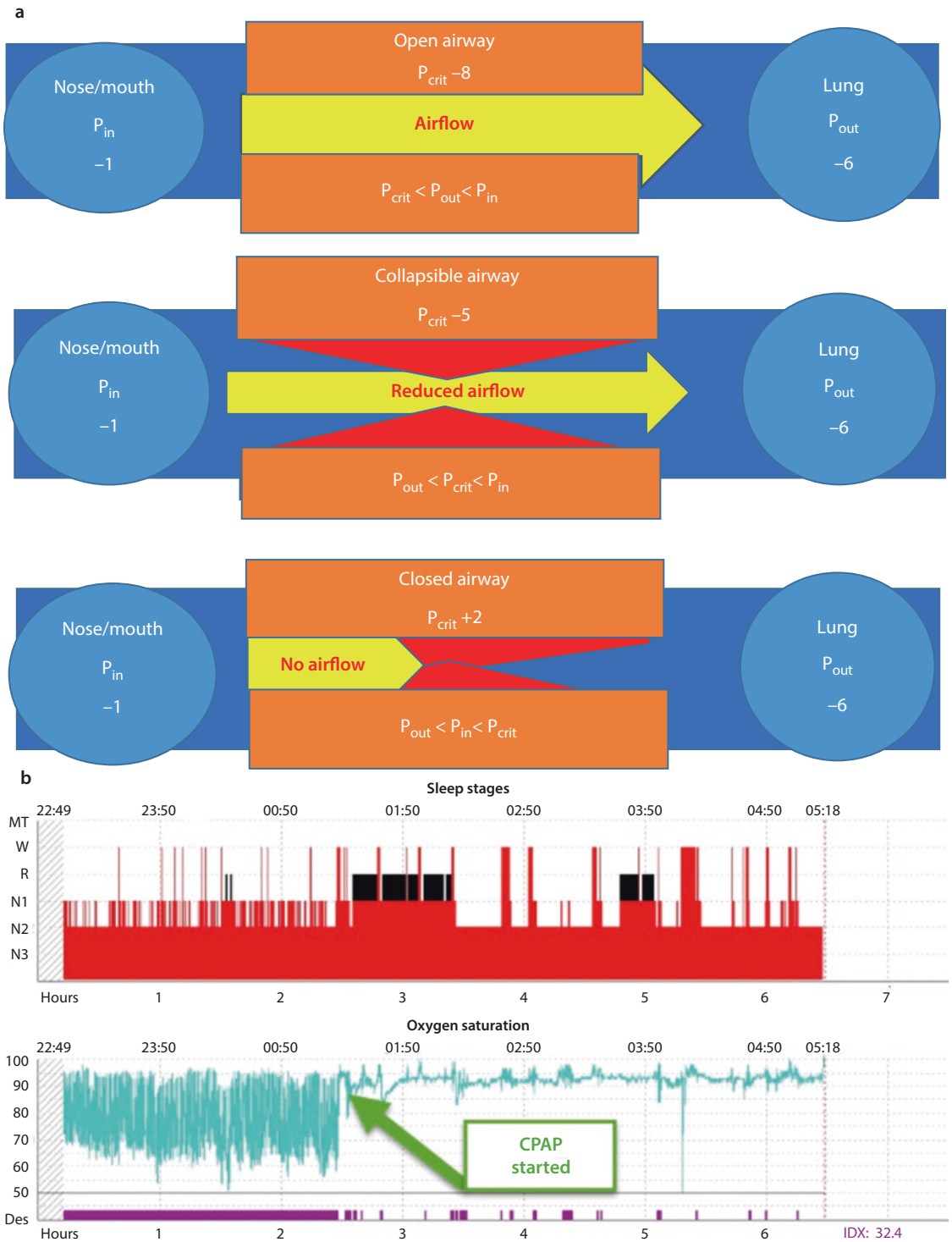

Fig. 2.2 **a** The above figure represents the flow of air and the P_{crit} values are arbitrary and used for representation only. The more negative the P_{crit} value of the pharynx, the stronger the ability of the pharynx to "pull away" from the center, thereby stabilizing the airway. The more positive the P_{crit} value of the pharynx in relation to the P_{in} and P_{out}, the more the pharynx tissue is "pushing" toward the center, thereby closing the airway

In the top example, the atmospheric pressure of P_{in} has a value of 1 cwp, the P_{crit} of the pharynx (open airway) has a value of −8 cwp, and the P_{out} has a value of negative −6 cwp. The pharynx has the most negative P_{crit} and can "pull away" from the center, does not collapse, is stable, and airflow is unimpeded

The middle example represents a collapsible airway, which can result in a hypopnea. The P_{in} is −1, P_{out} – 6, and the P_{crit} of the pharynx is −5, causing a partial collapse of the airway, restricting airflow. These patients often present with significant snoring

The bottom example represents a closed airway, which results in an apnea. The P_{in} is −1, P_{out} – 6, and the P_{crit} of the pharynx is +2. As the P_{crit} of the pharynx has the most positive value, the pharynx tissue collapses completely, causing obstruction

b An example of a highly collapsable airway and the immediate response to positive airway pressure that overcomes the Pcrit

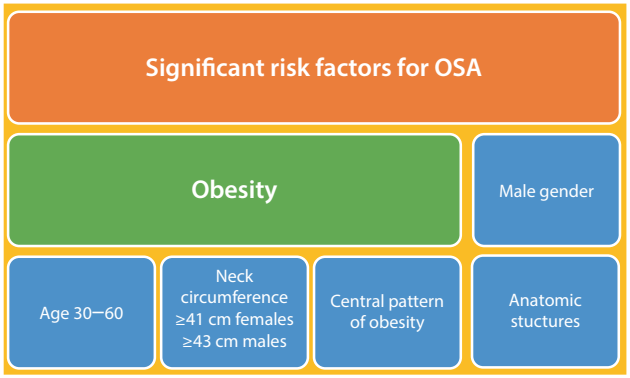

Fig. 2.3 Risk factors for obstructive sleep apnea

nificantly associated with severity of OSA in men; overall body fat was not associated with severity of OSA in women. Hip circumference and height-normalized neck circumference were associated with OSA severity in women [5, 37]. Males typically report more witnessed apneas and women report morning headache, fatigue, insomnia, mood disturbance, and enuresis. A study of 1370 male patients with OSA demonstrated that an increase in diastolic blood pressure was an independent variable associated with an elevated AHI [25].

OSA is heritable, and there are both direct genetic contributions to OSA susceptibility and indirect contributions via intermediate phenotypes such as obesity, craniofacial structure, neurological control of upper airway muscles, and circadian rhythm [40]. In recent years, much attention has been directed at defining heritable pathophysiologic mechanisms and finding genetic loci that contribute to development of OSA. A better understanding of the association of genetic markers could delineate diagnoses and treatment. A study of 751 participants of European ancestry utilizing single-nucleotide polymorphisms (SNPs) demonstrated several markers associated with obstructive sleep apnea [33]. A subsequent genome-wide association test was performed on 19,733 participants of African, Asian, European, and Hispanic/Latino American ancestry. RAI1 on chromosome 17 was identified as a possible quantitative trait locus for NREM AHI in men but not in women [10]. A meta-analysis of symptoms of sleep apnea in 1475 individuals of European descent identified a rare 3′-untranslated region of ERCC1 and ED3EAP genes on chromosome 19q13 confirming the association of symptoms of sleep apnea [64]. Both genes are expressed in tissues in the neck area such as the tongue, muscles, cartilage, and the trachea. A study of 86 severe OSA patients and 86 controls demonstrated higher proportion caveolin-1 polymorphisms in patients with severe OSA [1]. A subgroup analysis of 48 patients demonstrated an overexpression of the AMOT gene in patients with a high severity index for OSA [9]. Telomere shortening is linked to conditions that are highly prevalent in OSA such as hypertension, diabetes mellitus, and cardiovascular disease. However, a study comparing 106 OSA patients with 104 non-OSA patients demonstrated a J-shaped relationship between telomere length (TL) and OSA severity. The longest TL was found in those with moderate-to-severe OSA and was significantly longer than in the control group. The shortest TL occurred in mild OSA [49]. These findings indicate that telomere shortening is not a unidirectional process related to age and disease. Telomeres are similar to the plastic tips on shoelaces in that they keep the chromosome ends from "fraying." As a cell divides, telomeres normally get shorter, and at some point the cell can no longer divide. This shortening process is associated with aging and a higher risk of death. In cancer cells, telomeres are crucial for survival, and longer telomeres are key to the "immortality" of cancer cells. The association of longer telomeres and severe OSA is of unique interest due to do the familial perpetuation of OSA.

2.6 The Possible Role of Leptin

Leptin is a peptide hormone produced mainly in white adipose tissue and is present in the entire respiratory system. Leptin contributes to the regulation of energy homeostasis, inflammation, metabolism, and sympathetic nerve activity. Leptin and ghrelin levels are abnormally high in patients with OSA. Leptin was initially considered a hormone of satiety, thereby suppressing appetite. Ghrelin is considered a hormone of hunger, thereby promoting appetite. Recent studies in animal models demonstrate that leptin also has a role in regulating sleep architecture, upper airway patency, ventilator function, and hypercapnic ventilatory drive. Leptin may contribute to the regulation of breathing indirectly via changes in body temperature, acid-base balance, or mass of adipose tissue. However, obese individuals tend to develop a resistance to the protective effects of leptin through mechanisms that are not adequately delineated at this point. Leptin levels are higher in obese patients with OSA than obese and nonobese controls. In nonobese patients with OSA, leptin levels are often normal. Treatment of OSA decreases leptin levels independent of changes in BMI. Leptin may augment neural compensatory mechanisms in response to upper airway obstruction and minimize upper airway collapse [28, 32, 44, 61].

2.7 Summary

The fact that it has taken almost 4000 words to provide just a sampling of the evidence surrounding the various avenues by which OSA can arise should alert the reader to the fact that we actually don't precisely know which

factor or combination of factors are of greatest importance. Indeed, it is likely, as stated by Younes, that there are a variety of phenotypes of OSA and different mechanisms or combinations of mechanisms result in the different phenotypes [72]. Since it has not been possible to identify the pathogenesis underlying each phenotype (or even to delineate clearly the different phenotypes), OSA treatment has remained a "one-size-fits-all" proposition: positive airway pressure (PAP) treatment if tolerated; mandibular advancement prostheses for mild or mild to moderate OSA, or as a second-line treatment in those unable to tolerate PAP; and surgical approaches that continue to be the least attractive options with an uncertain role. Alternatively, hypoglossal nerve stimulation appears to be coming into vogue in carefully selected patients, with most optimal results associated with leaner patients and, paradoxically, those of more advanced age [23]. Whether this describes a particular phenotype, given the extensive data reviewed above, is unlikely. As with most reviews of any medical subject, we are forced to admit that additional research will be necessary in order to clarify OSA pathogenesis, particularly as to whether different mechanisms apply to different phenotypes.

Conflict of Interest Dr. Ralls has no conflicts to declare.

Dr. Cutchen has no conflicts to declare.

Dr. Brown has participated in advisory panels for Philips Respironics and has been an insurance claims reviewer for Considine and Associates, Inc. He co-edits the sleep and respiratory neurobiology section of Current Opinion in Pulmonary Medicine and wrote on CPAP treatment for obstructive sleep apnea in UpToDate and on obstructive sleep apnea in Clinical Decision Support: Pulmonary Medicine and Sleep Disorders. He is co-edited an issue of Sleep Medicine Clinics on positive airway pressure therapy. He serves on the Polysomnography Practice Advisory Committee of the New Mexico Medical Board and chairs the New Mexico Respiratory Care Advisory Board.

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Health Consequences of Obstructive Sleep Apnea

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Contents

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Core Message

- Obstructive sleep apnea (OSA) adversely affects virtually every organ system resulting in adverse health outcomes:
  - Neurocognitive consequences include impairments in daytime alertness, attention/vigilance, delayed long-term visual and verbal memory, visuospatial/constructional abilities, and executive function while neuropsychological ones include depression, somatic syndromes, anxiety, and attention deficit/hyperactivity disorder.
  - Cardiovascular consequences of OSA include CHF, systemic hypertension, ischemic heart disease, atrial fibrillation, ventricular arrhythmia, and stroke.
  - Respiratory consequences include poor symptom control in asthma, worse pulmonary function in chronic obstructive pulmonary disease (COPD), increased frequency of exacerbation in both asthma and COPD, increased prevalence and recurrence of pulmonary embolism, and a higher prevalence pulmonary hypertension.
  - Endocrine consequences include diabetes mellitus, metabolic syndrome, and sexual dysfunction in men and women.
  - Gastrointestinal consequences include gastroesophageal reflux disease and nonalcoholic fatty liver disease.
  - Obstetric consequences include pregnancy-related hypertensive disorders and gestational diabetes, as well as maternal cardiovascular, pulmonary, and surgical complications.
  - Perinatal consequences include low birth weight, preterm birth, NICU admission, and hyperbilirubinemia.
  - Perioperative consequences include postoperative ICU transfer, respiratory complications, cardiovascular events, and neurologic complications.
  - Accident-related consequences include motor vehicle crashes and work-related injuries.
  - Oncologic consequences include increased cancer incidence including breast and colorectal cancer.
  - Mortality-related consequences include higher death rates overall and from cardiovascular, noncardiovascular, and COPD-related causes.
- Nocturnal respiratory dysfunction (i.e., hypoxemia-reoxygenation and hypercapnia), poor sleep quality (i.e., increased arousals, poor sleep efficiency, and altered sleep architecture), and intrathoracic pressure variations, in addition to shared comorbid risk factors, result in oxidative stress, inflammation, sympathetic activation, endothelial dysfunction, neurohormonal changes, thrombophilia, and hemodynamic changes, which are the pathophysiologic mechanisms for these adverse clinical outcomes.

Obstructive sleep apnea (OSA) is associated with a growing number of adverse health outcomes (Fig. 3.1). This chapter will quantify the risks and describe the mechanisms behind the association between OSA and various adverse cardiovascular, cerebrovascular, respiratory, endocrine and metabolic, gastrointestinal, obstetric, perinatal, perioperative, accident-related, oncologic, and survival outcomes. This literature review is limited to OSA and includes neither the health consequences of other types of sleep-related breathing disorders (SRBD) such as snoring, central sleep apnea, or sleep-related hypoventilation or hypoxemia disorders nor the efficacy of various OSA therapies (e.g., CPAP) on these consequences. Neurocognitive (i.e., hypersomnolence, fatigue, impairments in attention/vigilance, delayed long-term visual and verbal memory, visuospatial/constructional abilities, and executive function) [13] and neuropsychological (e.g., depression, somatic syndromes, anxiety, and attention deficit/hyperactivity disorder) dysfunction due to OSA is discussed in detail in ▶ Chap. 7.

3.1 Cardiovascular Consequences

The Sleep Heart Health Study (SHHS), a cross-sectional, population-based epidemiologic study utilizing domiciliary polysomnography (PSG), described the association between OSA and cardiovascular disease in community-dwelling, middle-aged adults in the United States [92]. The SHHS revealed an apparent dose–response relationship between the severity of OSA based on the apnea–hypopnea index (AHI) or duration of nocturnal hypoxemia (SpO_2 <90%) and the prevalence of cardiovascular diseases, even after adjusting for known risk factors such as age, sex, body mass index (BMI), systemic hypertension, and high-density lipoprotein [100]. Since the publication of the SHHS study, several meta-analyses had corroborated the association between OSA and cardiovascular disease (Table 3.1).

3.1.1 Chronic Heart Failure

Of all the cardiovascular comorbidities, the SHHS reported chronic heart failure (CHF) had the strongest association with OSA [100]. The highest quartile of AHI severity (>11/hr) had the strongest relationship with heart failure. To date, there are no prospective cohort studies comparing the incidence of CHF in OSA patients with controls.

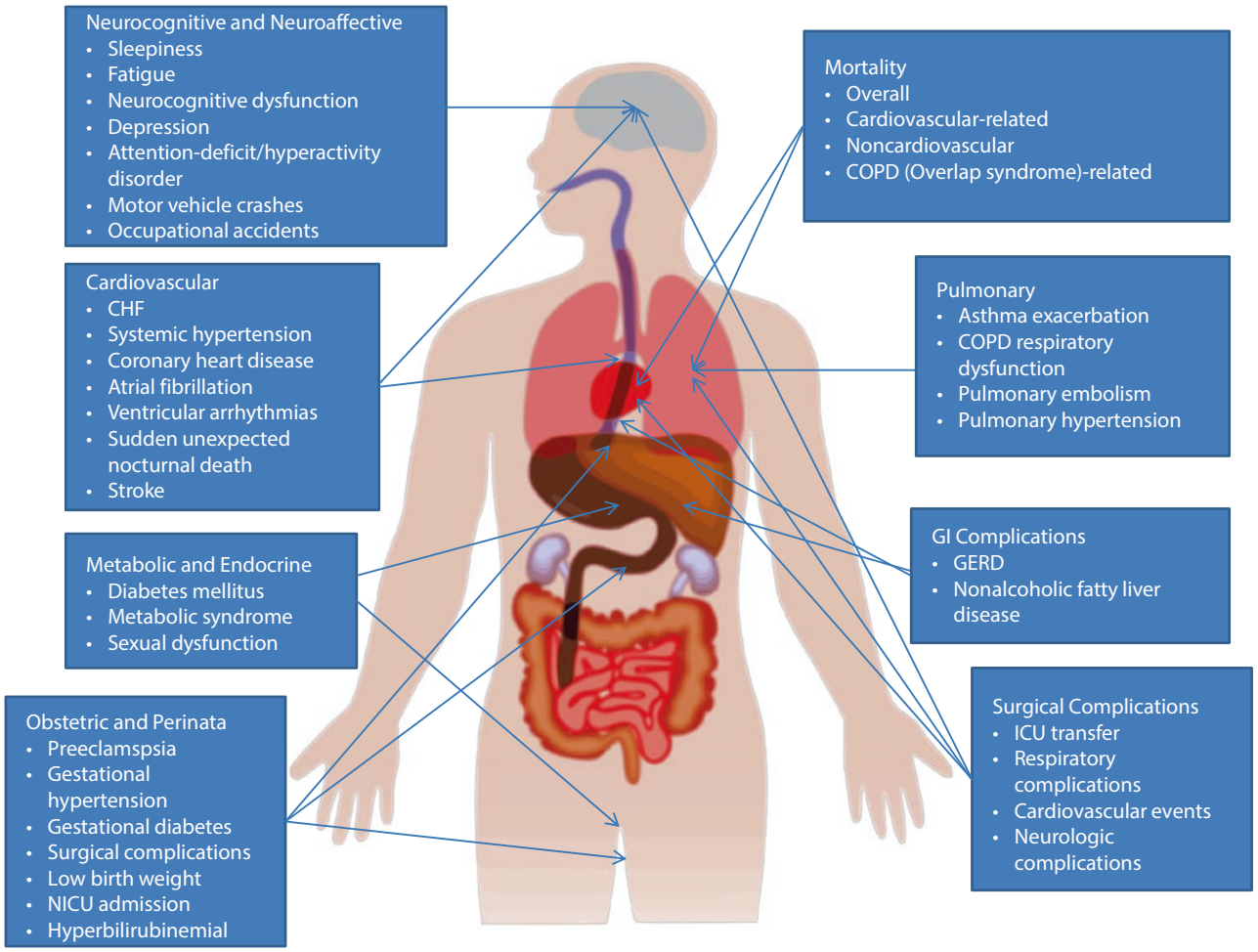

Fig. 3.1 Organ-based adverse health consequences of obstructive sleep apnea

3.1.2 Systemic Hypertension

The SHHS reported a higher prevalence of systemic hypertension in participants with OSA (AHI ≥5/hr) or nocturnal hypoxemia (SpO_2 <90% for ≥12% of the total sleep time) compared to controls [82]. In contrast, a prospective cohort analysis of SHHS data did not find an increased incidence of hypertension after controlling for BMI [83]. Nevertheless, a meta-analysis of six studies with 20,637 participants confirmed a statistically significantly increased incidence of systemic hypertension in OSA, regardless of severity [71]. A more recent meta-analysis reported increasing odds ratio of systemic hypertension with worsening severity of OSA [36].

OSA also has a strong association with treatment-refractory hypertension in patients with chronic kidney disease. The Sleep-SCORE study conducted unattended home PSG and monitored automated blood pressure (BP) in 88 end-stage renal disease (ESRD) patients not receiving dialysis, and demonstrated a significant association between the severity of sleep apnea and resistant hypertension (BP ≥140/90 mmHg on ≥3 BP medications) in those with ESRD on dialysis (but not in those without CKD or in those with CKD not on dialysis) [1]. A more recent meta-analysis by Hou et al. demonstrated a statistically significant association between OSA and resistant-hypertension [36].

3.1.3 Coronary Heart Disease

Based on the cross-sectional analysis of the SHHS, there appeared to be no increase in the prevalence of self-reported coronary heart disease (CHD) in OSA [100]. Subsequent meta-analyses reported conflicting results on the association between OSA and CHD. The first 2 meta-analyses of prospective studies by Loke and Dong, respectively, did not find an association between OSA and new-onset CHD [20, 64]. In contrast, one meta-analysis reported a doubling of the risk of a recurrent ischemic event [121] and nonfatal cardiovascular events in patients with OSA [23]. There was also an increased

Table 3.1 Strength of association between obstructive sleep apnea and cardiovascular outcomes

Cardiovascular outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Congestive heart failure	aOR = 2.38 (1.22, 4.62) overall aOR = 1.19 (0.56, 2.53) for AHI = 1.3–4.3/hr aOR = 1.96 (0.99, 3.90) for AHI = 4.4 to <10.9/hr aOR = 2.20 (1.11, 4.37) for AHI ≥11/hr.	[100]	Cross-sectional
Systemic hypertension	OR = 1.37 (1.03, 1.83) comparing highest (AHI ≥30/hr) vs. lowest (AHI <1.5/hr) categories OR = 1.41 (1.29, 1.89) comparing highest (≥12%) vs. lowest (0.05%) categories of percentage of sleep time below 90% oxygen saturation aOR = 1.51 (0.93–2.47) for AHI >30/hr OR = 1.26 (1.17, 1.35) for mild OSA OR = 1.50 (1.27, 1.76) for moderate OSA OR = 1.47 (1.33, 1.64) for severe OSA OR = 1.18 (1.09, 1.27) mild OSA OR = 1.32 (0.86, 1.20) moderate OSA OR = 1.56 (1.29, 1.83) severe OSA	[36, 71, 82, 83]	Cross-sectional Prospective cohort Meta-analysis Systematic review and meta-analysis
Resistant hypertension	aOR 3.5 (0.8, 15.4) in non-CKD aOR = 1.2, (0.4, 3.7) in nondialysis CKD aOR = 7.1, (2.2, 23.2) in ESRD on dialysis OR = 2.84 (1.7, 3.98) in all OSA patients	[1, 36]	Prospective cohort Systematic review and meta-analysis
Coronary heart disease	aOR = 1.27 (0.99, 1.62) OR = 1.56 (0.83, 2.91) OR = 1.92 (1.06, 3.4) in 5 male-predominant studies RR = 1.37 (0.95–1.98) RR = 2.06 (1.13, 3.77) for recurrent ischemic heart disease	[20, 64, 100, 121]	Cross-sectional Meta-analysis Meta-analysis Meta-analysis
Cardiovascular disease	RR = 2.48 (1.98, 3.10) RR = 1.79 (1.47, 2.18) for severe OSA	[20, 115]	Meta-analysis Meta-analysis
Nonfatal cardiovascular events	OR = 2.46 (1.80, 3.36)	[23]	Meta-analysis
Cardiovascular events after percutaneous coronary intervention	RR = 1.59 (1.22, 2.06)	[128]	Meta-analysis
Subclinical cardiovascular disease	aOR range = 1.036–2.21 for coronary artery calcium	[2]	Systematic review
Nocturnal atrial and ventricular arrhythmias	Prevalence ratio = 1.04 (1.01, 1.07)	[17]	Prospective cohort study
Prevalent atrial fibrillation	aOR = 4.02 (1.03, 15.74) OR = 2.15 (1.19, 3.89) in older men in the highest RDI quartile	[69, 70]	Cross-sectional Cross-sectional
Incident atrial fibrillation	HR = 2.18 (1.34, 3.54)	[27]	Retrospective cohort
Atrial fibrillation recurrence after catheter ablation	RR = 1.25 (1.08, 1.45) OR = 1.70 (1.40, 2.06)	[18, 79]	Meta-analysis Meta-analysis
Atrial fibrillation post-coronary artery bypass grafting	OR = 2.38 (1.57, 3.62)	[90]	Systematic review and meta-analysis
Nonsustained ventricular tachycardia	OR = 3.40 (1.03, 11.20) aOR = 1.07 (1.02, 1.12) in hypertrophic obstructive cardiomyopathy patients	[69, 113]	Cross-sectional Cohort study

(continued)

Table 3.1 (continued)

Cardiovascular outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Complex ventricular ectopy	OR = 1.74 (1.11, 2.74)	[69]	Cross-sectional
Ventricular arrhythmias	OR = 5.6 (2.0, 15.6) in patients with cardioverter-defibrillator aOR = 1.02 (0.98, 1.07) in a national inpatient sample	[96, 126]	Prospective cohort Cross-sectional study
Stroke	aOR = 1.42 (1.13, 1.78) OR = 2.24, (1.57, 3.19) RR = 2.02 (1.40, 2.90) RR = 2.15 (1.42, 3.24) for severe OSA OR = 1.94, (1.29, 2.92) RR = 2.15 (1.42, 3.24) in severe OSA aHR = 1.94 (1.31, 2.89)	[20, 58, 64, 100, 115, 118, 121]	Cross-sectional Meta-analysis Meta-analysis Meta-analysis Meta-analysis Meta-analysis Meta-analysis

Abbreviations: *aOR* adjusted odds ratio, *OR* odds ratio, *AHI* apnea–hypopnea index, *CKD* chronic kidney disease, *ESRD* end-stage renal disease, *RR* relative risk or risk ratio, *HR* hazard ratio, *aHR* adjusted hazard ratio

incidence of acute coronary events after percutaneous coronary intervention in OSA patients [128]. Noninvasive studies investigating subclinical cardiovascular disease described an increased occurrence of atherosclerosis (i.e., coronary artery calcification, carotid intima thickness, brachial artery flow-mediated dilatation, and pulse wave velocity) in OSA subjects [2].

3.1.4 Arrhythmias

Arrhythmias are perceived to occur more commonly in patients with OSA. A population study in Brazil reported an increased occurrence of nocturnal atrial and ventricular arrhythmias on polysomnography [17]. According to a 5-year retrospective cohort study, the risk of incident atrial fibrillation (AF) was doubled in OSA subjects, particularly those younger than 65 years, even after controlling for cardiovascular risk factors [27]. Nocturnal hypoxemia was found to be a significant predictor of new-onset AF. The risk of AF after coronary artery bypass grafting was also significantly increased in OSA [90]. In addition, recurrence of AF after catheter ablation therapy appeared to also be higher with OSA [18, 79].

A systematic review of 22 studies by Raghuram et al. reported an elevated risk of ventricular ectopy and arrhythmias [93]. OSA was associated with a higher prevalence of nonsustained ventricular tachycardia (NSVT) [69]. The severity of AHI correlated with the prevalence of NSVT in patients with hypertrophic obstructive cardiomyopathy [113]. On the other hand, although a cross-sectional analysis of a national inpatient sample showed a higher prevalence of ventricular arrhythmias in hospitalized OSA patients, no significant association was determined after adjusting for cardiovascular risk factors [96].

3.1.5 Cerebrovascular Disease

There is an increased prevalence of SRBD in patients diagnosed with cerebrovascular disease (CVD), with estimates ranging from 71 to 72% for an AHI >5/hr. and 20–30% [98] for AHI >20/hr. [42, 98]. The predominant type of SRBD was OSA, with only 7% having primarily central apnea [42]. Factors associated with SRBD in stroke were male gender, recurrent strokes, and an idiopathic etiology, but not event type (ischemic vs. hemorrhage), timing after stroke, or type of monitoring [42]. The cross-sectional analysis of the SHHS data also reported a strong association between stroke and OSA [100]. Conversely, four subsequent meta-analyses had confirmed the higher incidence of stroke in OSA patients. Li and colleagues reported a doubling of the risk of incident fatal and nonfatal strokes in patients with OSA [58]. Loke et al. corroborated this association but reported that most studies primarily enrolled men [64]. Xie and colleagues confirmed that OSA patients with a history of CVD or CHD had a significantly higher risk of stroke [121]. A meta-analysis of prospective cohort studies involving three million participants corroborated the increased incidence of cerebrovascular disease, even after controlling for

known risk factors [118]. The risk of stroke appeared to be related to the severity of OSA, that is, a higher stroke risk in moderate-to-severe OSA but not in mild OSA [115].

3.2 Respiratory Consequences

3.2.1 Asthma

Asthmatic patients are more than twice as likely to have OSA, especially with higher BMI [51, 59] (Table 3.2). OSA may aggravate asthma control and increase asthma exacerbation frequency [110, 116].

3.2.2 Chronic Obstructive Pulmonary Disease

The prevalence of OSA in patients with chronic obstructive pulmonary disease (COPD) ranges anywhere from 10 to 66% depending on the population sample [22]. A prospective cohort study demonstrated that comorbid OSA in COPD patients was associated with a significantly higher frequency of hospitalization due to severe exacerbation [67]. The overlap of OSA and COPD is associated with worse diurnal and nocturnal lung function (i.e., hypoxemia, hypercapnia, and 6-minute walk distance) and polysomnographic findings [i.e., worse AHI and oxygen desaturation index (ODI), nocturnal hypoxemia, sleep efficiency, arousal index, and sleep architecture].

3.2.3 Pulmonary Embolism

Patients diagnosed with venous thromboembolism (VTE) have a significantly higher prevalence of OSA. A nested case–control study found patients, especially women, with VTE had more than double the odds of having OSA even after adjusting for thrombophilic risk factors [5]. More than half of acute pulmonary embolism (PE) survivors had OSA [8, 52]. OSA may well be considered a thrombophilic condition. A case–control study of 209 patients found a higher prevalence of PE in patients with OSA [3]. The same investigators followed 120 PE patients who had stopped their anticoagulation for 5 to 8 years and demonstrated a 20-fold higher incidence of recurrent PE [4]. A population-based, retrospective cohort study reported that OSA patients were more likely to suffer from VTE (deep venous thrombosis and PE) [88]. A retrospective cohort study at the Mayo Clinic revealed heightened risks of occurrence, as well as recurrence of PE in OSA patients [97]. Moreover, high-risk PE is more likely to occur in those with moderate-to-severe OSA [8, 52]. A diagnosis of OSA may worsen pulmonary artery thrombus load and disease severity in

Table 3.2 Strength of association between obstructive sleep apnea and pulmonary outcomes

Pulmonary outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Asthma	OR = 1.92 (1.34, 2.76) OR = 3.73 (2.90, 4.57)	[51, 59]	Meta-analysis Meta-analysis
Asthma exacerbation	aOR = 1.322 (1.148, 1.523) with AHI aOR = 3.4 (1.2, 10.4)	[110, 116]	Case–control Retrospective cohort
COPD exacerbation requiring hospitalization	RR = 1.70 (1.21, 2.38)	[67]	Prospective cohort
Deep venous thrombosis	HR = 3.50 (1.83, 6.69)	[88]	Longitudinal, nationwide, population-based cohort
Pulmonary embolism	aOR = 3.7 (1.3, 10.5) HR = 3.97 (1.85, 8.51) aOR = 1.44 (1.07, 1.90)	[3, 88, 97]	Prospective cohort Longitudinal, population-based cohort Retrospective cohort
Recurrent pulmonary embolism	aHR = 20.73 (1.71, 251.28) aOR = 2.21 (1.05, 4.68)	[4, 97]	Prospective cohort Retrospective cohort

Abbreviations: *aOR* adjusted odds ratio, *AHI* apnea–hypopnea index, *COPD* chronic obstructive pulmonary disease, *RR* relative risk or risk ratio, *HR* hazard ratio, *aHR* adjusted hazard ratio

acute PE [31]. However, the transient increase in central venous pressure after an acute PE does not seem to affect OSA severity once patients are clinically stable to undergo PSG [7]. The severity of OSA based on the AHI and time spent with SpO_2 <90% were independent predictors of recurrent PE risk. The proposed mechanisms for this increased VTE risk include the heightened blood viscosity, clotting factors, tissue factor, platelet activity, and whole blood coagulability, as well as the attenuated fibrinolysis in OSA [60].

3.2.4 Pulmonary Hypertension

The prevalence of SRBD tends to be much higher in patients with pulmonary hypertension (PH). One study found a 71% SRBD prevalence in patients with pulmonary arterial hypertension, with 56% having OSA [72]. Conversely, there is a disproportionately higher prevalence of PH in OSA patients, with estimates ranging from 17 to 67% [47, 95]. A study employing right heart catheterization (RHC) in 220 consecutive OSA calculated a PH prevalence of 17% [15]. PH occurrence in this RHC study was attributed to the comorbid obstructive ventilatory abnormality with associated hypoxemia and hypercapnia rather than the severity of OSA. In contrast, an echocardiographic study determined that a nadir SpO_2 <70% rather than the AHI was a good predictor of PH [117]. A meta-analysis of studies employing echocardiography demonstrated a higher prevalence of RV dilatation, hypertrophy, and dysfunction in OSA [68].

3.3 Endocrine and Metabolic Consequences

3.3.1 Diabetes Mellitus

The SHHS and the Atherosclerosis Risk in Communities Study corroborated a significantly increased 13-year incidence of DM in patients with severe OSA patients [77] (Table 3.3). A meta-analysis of six prospective cohort studies confirmed this link between DM in severe OSA [114]. Diabetic microvasculopathy appeared to be aggravated by OSA. A meta-analysis of longitudinal and cross-sectional studies determined a 73% greater risk of diabetic nephropathy with OSA [57]. A subsequent meta-analysis by the same investigators also found a higher occurrence of diabetic retinopathy and maculopathy, which correlated with the duration of nocturnal hypoxemia [56].

3.3.2 Metabolic Syndrome

The metabolic syndrome is defined by the cluster of high blood pressure, diabetes, hypercholesterolemia, and abdominal obesity. Two meta-analyses estimated a two-to-threefold increased risk of metabolic syndrome in OSA [91, 122]. Nadeem et al. singled out the AHI as a significant independent predictor of hypercholesterolemia and hypertriglyceridemia [75]. OSA is also associated with elevated leptin levels, nocturnal hypoxemia, impaired glucose tolerance, and increased C-reactive protein level independent of BMI [6, 43].

Table 3.3 Strength of association between obstructive sleep apnea and endocrine and metabolic outcomes

Metabolic disease outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Diabetes mellitus type 2	RR = 1.22 (0.91, 1.63) for mild OSA RR = 1.63 (1.09, 2.45) for moderate-to-severe OSA HR = 1.71 (1.08, 2.71)	[77, 114]	Meta-analysis Prospective cohort
Diabetic kidney disease	OR = 1.73 (1.13, 2.64)	[57]	Meta-analysis
Diabetic retinopathy	OR = 0.91(0.87–0.95) with minimum oxygen saturation	[56]	Meta-analysis
Metabolic syndrome	OR = 2.87 (2.41, 3.42) OR = 2.56 (1.98, 3.31) aOR = 1.97 (1.34, 2.88)	[91, 122]	Meta-analysis of cross-sectional studies Meta-analysis of case-control studies Meta-analysis
Erectile dysfunction	RR = 1.82 (1.12, 2.97) OR = 2.22 (1.41, 5.55)	[45, 62]	Meta-analysis Systematic review and meta-analysis
Female sexual dysfunction	RR = 2.00 (1.29, 3.08)	[62]	Meta-analysis

Abbreviations: *RR* relative risk or risk ratio, *HR* hazard ratio, *OR* odds ratio, *aOR* adjusted odds ratio

3.3.3 Sexual Dysfunction

Meta-analyses on the association of OSA and sexual function calculated a doubling of the risk of erectile dysfunction in men and sexual dysfunction in women, respectively [45, 62]. A systematic review by Steinke and colleagues determined that in addition to altered hormone levels, the duration of nocturnal hypoxemia significantly predicted sexual dysfunction in women while BMI and inflammatory markers were significant predictors in men [109].

3.4 Gastrointestinal Consequences

3.4.1 Gastroesophageal Reflux Disease

OSA patents are at a 1.75- to twofold higher risk of gastroesophageal reflux disease (GERD) [32, 119] and a threefold higher risk of nocturnal GERD [125] (Table 3.4). Nocturnal GERD correlated with the severity of OSA [106]. You et al.'s endoscopic-based investigation observed a higher occurrence of nonerosive, but not in erosive, esophagitis in OSA [125]. OSA also appeared to be associated with Barrett's esophagitis, with the risk related to the AHI severity [33, 55].

Conversely, GERD symptoms can aggravate sleep-disordered breathing, resulting in higher AHI, longer maximum apnea duration, lower minimum oxygen saturation, higher ODI, and poorer sleep efficiency [48]. Gastroesophageal reflux events usually occur after spontaneous awakenings and arousals rather than after disordered-breathing events [39, 124]. Utilizing high-resolution esophageal manometry and 24-hr. esophageal pH-impedance monitoring, Shepherd et al. identified obesity to be the mediator of reflux events in OSA [105].

3.4.2 Nonalcoholic Fatty Liver Disease

The presence of OSA approximately doubled the likelihood of a histological, chemical, or radiographic diagnosis of nonalcoholic steatohepatosis, steatohepatitis, and hepatic fibrosis [74, 107]. Elevations in ALT, but not AST, levels significantly correlated with the AHI [41].

Table 3.4 Strength of association between obstructive sleep apnea and gastrointestinal outcomes

Gastrointestinal disease outcome	Strength of association, point estimate (95% confidence interval)	References	Study design
GERD	aOR = 2.13 (1.17, 3.88) OR = 1.75 (1.18, 2.59)	[32, 119]	Cross-sectional population-level analysis Meta-analysis
Non-erosive gastroesophageal reflux Erosive gastroesophageal reflux	aOR = 1.82 (1.15, 2.90) aOR = 0.93 (0.56, 1.55)	[125]	Cross-sectional
Nocturnal GERD	aOR = 2.97 (1.19, 7.84) aOR = 1.84(1.28, 2.63) for moderate OSA aOR = 2.39 (1.71, 3.33) for severe OSA	[106, 125]	Cross-sectional Cross-sectional
Barrett's esophagitis	aOR = 1.2 (1.0, 1.3) per 10-unit increase in AHI OR = 3.26 (1.72, 6.85) per 10-unit increase in AHI	[33, 55]	Cross-sectional Retrospective cohort
Nonalcoholic fatty liver disease: Fatty liver Inflammation Fibrosis	OR = 2.556 (1.184, 5.515 OR = 1.800 (0.905, 3.579) OR = 2.586 (1.289, 5.189)	[107]	Meta-analysis
Nonalcoholic fatty liver disease: Histology Radiology Elevated AST or ALT NASH, any stage Fibrosis Advanced fibrosis	OR = 2.01 (1.36, 2.97) OR = 2.34 (1.71, 3.18) OR = 2.53 (1.93, 3.31) OR = 2.37(1.59, 3.51) OR = 2.16 (1.45, 3.20) OR = 2.30 (1.21, 4.38).	[74]	Meta-analysis

Abbreviations: *GERD* gastroesophageal reflux disease, *aOR* adjusted odds ratio, *OR* odds ratio, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *NASH* nonalcoholic steatohepatosis

The predisposition for nonalcoholic fatty liver disease in OSA patients is expected given the shared risk factors (e.g., obesity) and comorbid conditions (e.g., DM and metabolic syndrome).

3.5 Obstetric Outcomes

3.5.1 Pregnancy-Related Hypertensive Disorders

Pregnancy-related hypertensive disorders (i.e., preeclampsia, gestational hypertension, and eclampsia) occurred more frequently in pregnant women with OSA [63]. Several studies had corroborated a 2- to threefold increased risk of preeclampsia in gravid women with OSA [10, 19, 38, 63, 123] (Table 3.5). Virtually all of the studies found a significant association between OSA and gestational hypertension. A national cohort study observed a threefold increased incidence of eclampsia in pregnant women with OSA [10].

3.5.2 Gestational Diabetes

The odds of developing gestational diabetes were 1.5–4.7 times greater in pregnant women with OSA [10, 19, 37]. Reutrakul et al. identified arousal index and ODI as significant independent predictors of impaired glucose tolerance in pregnancy with OSA [94]. Pathophysiologic mechanisms behind gestational diabetes in OSA patients include maternal sleep disruption, intermittent hypoxemia, oxidative stress, inflammation, catecholaminergic activation, peripheral vasoconstriction, and endothelial dysfunction.

3.5.3 Maternal Cardiovascular and Pulmonary Complications

A nationwide cohort study of 1,577,632 gravidas in the United States recorded a significantly higher occurrence of adverse cardiovascular events such as pulmonary edema, CHF, and cardiomyopathy in pregnant women with OSA [10]. Although there was a fivefold increase in the odds of PE or pulmonary infarction in pregnant women with OSA, the difference was not statistically significant [10]. The incidence of peripartal stroke was also not increased [10].

3.5.4 Maternal Surgical Complications

Although a small prospective cohort study found no difference in the need for caesarean delivery when using the Berlin Questionnaire to screen for OSA [50], subsequent prospective studies [65, 108] and a meta-analyses of cohort studies [123] observed significantly higher rates of both elective and emergent caesarean delivery in pregnant women with OSA.

Two studies on wound complications after delivery showed conflicting results, with a prospective cohort study [65] showing no increase while a large retrospective national cohort study reporting a significant increase [10]. The same national cohort study reported higher rates of maternal hysterectomy and ICU admission and a longer length of stay but no difference in blood transfusion requirement [10].

3.6 Perinatal Outcomes

3.6.1 Impaired Fetal Growth

Studies on the effect of OSA on fetal growth have conflicting results. A prospective cohort study of 26 high- and 15 low-OSA risk pregnant women did not find a significant association between OSA and fetal growth after adjusting for BMI [25] (Table 3.6). In contrast, a case–control study and a meta-analysis of 24 studies found a significant association between maternal OSA and impaired fetal growth [19, 49]. However, a national cohort study of more than 1.5 million gravidas did not corroborate the association between maternal OSA and fetal growth [10].

3.6.2 Preterm Birth

An earlier prospective cohort study of 175 obese pregnant women found no association between OSA and preterm birth in neonates of women with OSA [65]. In contrast, three subsequent meta-analyses reported a significant doubling of the risk of preterm birth in neonates of pregnant women with OSA [12, 19, 123].

3.6.3 Small for Gestational Age/Low Birthweight

Although a prospective cohort study of Korean pregnant women reported no association between suspected maternal OSA (based on the Berlin

Table 3.5 Strength of association between obstructive sleep apnea and obstetric outcomes

Obstetric outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Preeclampsia-eclampsia	OR = 2.72 (1.33, 5.57)	[38]	Prospective cohort study
Preeclampsia	OR = 2.19 (1.71, 2.80) RR = 1.96 (1.34, 2.86) aOR = 2.22 (1.94, 2.54)	[10, 19, 123]	Systematic review and quantitative analysis Meta-analysis Retrospective national cohort
Gestational hypertension	OR = 2.38 (1.63, 3.47) RR = 1.40 (0.62, 3.19) aOR = 1.67 (1.42, 1.97)	[10, 19, 123]	Systematic review and quantitative analysis Meta-analysis Retrospective national cohort
Eclampsia	aOR = 2.95 (1.08, 8.02)	[10]	Retrospective national cohort
Gestational diabetes	OR = 1.78 (1.29, 2.46) aOR = 1.52 (1.34, 1.72) OR = 4.71 (1.05, 21.04)	[10, 19, 37]	Systematic review and quantitative analysis Retrospective national cohort Case–control
Pulmonary edema	aOR = 5.06 (2.29, 11.1)	[10]	Retrospective national cohort
Congestive heart failure	aOR = 3.63 (2.33, 5.66)	[10]	Retrospective national cohort
Cardiomyopathy	aOR = 3.59 (2.31, 5.58)	[10]	Retrospective national cohort
Pulmonary embolism and infarction	aOR = 5.25 (0.64, 42.9)	[10]	Retrospective national cohort
Stroke	aOR = 3.12 (0.41, 23.9)	[10]	Retrospective national cohort
Assisted vaginal delivery	OR = 1.88 (1.10, 3.21)	[12]	Meta-analysis
Cesarean delivery	aOR = 1.53 (0.79, 2.96) for BMI <30 aOR = 3.48 (0.90, 13.37) for BMI ≥30 aOR = 3.04 (1.14–8.1) RR = 1.87 (1.52, 2.29) aOR = 1.60 (1.06, 2.40) OR = 1.81 (1.55, 2.11) OR = 1.38 (1.09, 1.76) For elective OR = 2.52 (1.20, 5.29) For emergency	[12, 50, 65, 108, 123]	Prospective cohort Prospective cohort Meta-analysis Retrospective cohort Meta-analysis
Wound complications	aOR = 3.44 (0.7–16.93) aOR = 1.77 (1.24, 2.54)	[10, 65]	Prospective cohort Retrospective national cohort
Hysterectomy	aOR = 2.26 (1.29, 3.98)	[10]	Retrospective national cohort
Transfusion	aOR = 0.81 (0.11, 5.85)	[10]	Retrospective national cohort
Length of stay	aOR = 1.18 (1.05, 1.32)	[10]	Retrospective national cohort
Maternal ICU admission	aOR = 2.74 (2.36, 3.18)	[10]	Retrospective national cohort

Abbreviations: *OR* odds ratio, *RR* relative risk or risk ratio, *aOR* adjusted odds ratio, *BMI* body mass index, *ICU* intensive care unit

Table 3.6 Strength of association between obstructive sleep apnea and perinatal outcomes

Perinatal outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Impaired fetal growth	aOR = 5.3 (0.93, 30.34) OR = 1.44 (1.22, 1.71) aOR = 1.05 (0.84, 1.31) aOR = 3.9 (1.2, 12.6)	[10, 19, 25, 49]	Prospective observational Systematic review and quantitative analysis Retrospective national cohort Case-control
Preterm birth:	aOR = 0.63 (0.18, 2.24) for <37 weeks aOR = 0.94 (0.10, 8.92) for <32 weeks OR = 1.98 (1.59, 2.48) RR = 1.90 (1.24, 2.91) OR = 1.86 (1.50, 2.31)	[12, 19, 65, 123]	Prospective cohort Systematic review and quantitative analysis Meta-analysis Meta-analysis
Small for gestational age <tenth percentile	OR = 2.56 (0.56, 11.68) for BMI <30 OR = 0.83 (0.04, 19.4) for BMI ≥30	[50]	Prospective cohort
Low birth weight	OR = 1.75 (1.33, 2.32) OR = 1.67 (1.00, 2.78)	[12, 19]	Systematic review and quantitative analysis Meta-analysis
Stillbirth Stillbirth or perinatal death	aOR = 1.17 (0.79, 1.73) aOR = 2.02 (1.25, 3.28)	[9, 12]	Retrospective national cohort Meta-analysis
APGAR score <7	OR = 2.14 (1.24, 3.71)	[12]	Meta-analysis
NICU admission Neonatal nursery admission	aOR = 3.39 (1.23, 9.32) OR = 2.43 (1.61, 3.68) RR = 2.65 (1.68, 3.76) OR = 1.90 (1.32, 2.61)	[12, 19, 65, 123]	Prospective cohort Systematic review and quantitative analysis Meta-analysis Meta-analysis
Hyperbilirubinemia	aOR = 3.63 (1.35–9.76)	[65]	Prospective cohort
Respiratory morbidity	aOR = 1.56 (0.5–4.59)	[65]	Prospective cohort

Abbreviations: *aOR* adjusted odds ratio, *OR* odds ratio, *BMI* body mass index, *NICU* neonatal intensive care unit, *RR* relative risk or risk ratio

Questionnaire) and infants born small for gestational age [50], two meta-analyses observed a 67–75% increase in likelihood of low birthweight neonates in mothers with OSA [12, 19, 36].

3.6.4 Stillbirth

Although a large national cohort study observed no association between stillbirth and maternal OSA [10], a recent meta-analysis reported a significant doubling of the risk of stillbirth [12].

3.6.5 NICU Admission

Multiple studies (1 prospective cohort study and 3 quantitative/meta analyses) were unanimous in corroborating a two- to threefold significant increase in the risk of neonatal ICU/nursery admission in newborns of mothers with OSA [12, 19, 65, 123]. The likelihood of hyperbilirubinemia, but not respiratory morbidity, was higher in newborns of women with OSA. [65].

3.7 Perioperative Outcomes

Several meta-analyses as well as a retrospective nationwide cohort analysis corroborated the adverse health effects of OSA on most perioperative outcomes such as ICU transfer, respiratory complications (i.e., postoperative hypoxemia, acute respiratory failure, emergent intubation, and need for CPAP or noninvasive ventilation), major adverse cardiac or cerebrovascular events, AF, and neurologic complications [26, 34, 44, 73, 76, 90] (Table 3.7). However, OSA was not associated with a longer hospital stay [76]. A qualitative systematic review by the Society of Anesthesia and Sleep Medicine Task

Table 3.7 Strength of association between obstructive sleep apnea and perioperative outcomes

Perioperative outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Perioperative complications	OR = 3.93 (1.85, 7.77)	[76]	Meta-analysis
ICU transfer	OR = 2.81 (1.46, 5.43) OR = 2.97 (1.90, 4.64) OR = 2.46 (1.29, 4.68)	[26, 34, 44]	Meta-analysis Meta-analysis Meta-analysis
Postoperative hypoxemia	OR = 2.27 (1.20, 4.26) OR = 3.06 (2.35, 3.97)	[26, 44]	Meta-analysis Meta-analysis
Respiratory complications	OR = 2.77 (1.73, 4.43)	[26]	Meta-analysis
Acute respiratory failure	OR = 2.43 (1.34, 4.39) OR = 2.42 (1.53, 3.84)	[34, 44]	Meta-analysis
Postoperative tracheal intubation and mechanical ventilation	OR = 2.67 (1.0, 6.89)	[76]	Meta-analysis
MACCE	OR = 2.4 (1.38, 4.2)	[76]	Meta-analysis
Cardiac events	OR = 2.07 (1.23, 3.50) OR = 1.63, (1.16, 2.29) OR = 1.76 (1.16, 2.67)	[26, 34, 44]	Meta-analysis Meta-analysis Meta-analysis
New postoperative atrial fibrillation	OR = 1.94 (1.13, 3.33)	[76]	Meta-analysis
Atrial fibrillation after CABG	aOR = 2.38 (1.57, 3.62)	[90]	Meta-analysis
Hospital length of stay	Mean difference = +2.01 (0.77, 3.24) days	[76]	Meta-analysis
Neurologic complications	OR = 2.65, (1.43, 4.92)	[26]	Meta-analysis

Abbreviations: *OR* odds ratio, *ICU* intensive care unit, *MACCE* Major adverse cardiac or cerebrovascular events, *aOR* adjusted odds ratio, *CABG* coronary artery bypass graft

Force on Preoperative Preparation of Patients with Sleep-Disordered Breathing in 2016 conveyed a greater risk of pulmonary and combined complications [84].

3.8 Accident-Related Consequences

OSA doubled the odds of motor vehicle crashes [111], occupational accidents [29], and occupational injury [35] (Table 3.8). Hirsch Allen et al. reported a higher frequency of occupational injuries due to reduced vigilance in workers with OSA, but this association became statistically insignificant after adjusting for confounders [35]. A meta-analysis listed BMI, AHI, nocturnal hypoxemia, and daytime sleepiness as predictors for motor vehicle crashes [111]. Another meta-analysis singled out occupational driving as a high-risk activity in workers with OSA [29].

3.9 Cancer-Related Outcomes

There is growing evidence to suggest a higher incidence of cancer in OSA patients. Two meta-analyses on the association between cancer incidence and OSA had contradictory results [86, 127] (Table 3.9). More recent studies identified a higher cancer incidence in younger adults (<45 years) [11] and Veterans with OSA [40]. There were early reports of increased incidence of particular malignancies, including breast cancer [16] and colorectal cancer [54] in OSA. On the other hand, a meta-analysis by Zhang et al. did not find an association between OSA and cancer mortality [127]. Further studies are required to validate and elucidate the mechanisms behind the purported increased cancer incidence in OSA.

Table 3.8 Strength of association between obstructive sleep apnea and accident-related outcomes

Type of accident	Strength of association, point estimate (95% confidence interval)	References	Study design
Motor vehicle crashes	OR = 2.427 (1.205, 4.890)	[111]	Systematic review
Occupational accidents	OR = 2.18 (1.53, 3.10)	[29]	Meta-analysis
Occupational injury	aOR = 1.76 (0.86, 3.59)	[35]	Prospective cohort

Abbreviations: *OR* odds ratio, *aOR* adjusted odds ratio

Table 3.9 Strength of association between obstructive sleep apnea and cancer outcomes

Cancer outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Cancer incidence	aRR = 1.40 (1.01, 1.95) aHR = 0.91 (0.74, 1.13) for mild OSA aHR = 1.07 (0.86,1.33) for moderate OSA aHR = 1.03 (0.85, 1.26) for severe OSA aHR = 3.7 (1.12, 12.45) for OSA patients <45 years old aHR = 1.97 (1.94, 2.00)	[11, 40, 86, 127]	Meta-analysis Meta-analysis Prospective cohort Retrospective matched cohort
Breast cancer incidence	RR = 1.20 (1.04, 2.71) RR = 1.72 (1.10, 2.71)	[16]	Retrospective cohort study
Colorectal cancer incidence	aHR = 1.80 (1.28, 2.52) aOR = 3.03 (1.44, 6.34)	[54]	Nationwide population-based cohort
Cancer mortality	aHR = 0.79 (0.46, 1.34) for mild OSA aHR = 1.92 (0.63, 5.88) for moderate OSA aHR = 2.09 (0.45, 9.81) for severe OSA	[127]	Meta-analysis

Abbreviations: *aRR* adjusted relative risk, *aHR* adjusted hazard ratio, *RR* relative risk, *aOR* adjusted odds ratio

3.10 Survival Outcomes

3.10.1 Overall Mortality

Several meta-analyses were unanimous in corroborating that OSA increased overall mortality, particularly in those with severe disease [23, 24, 30, 87, 115, 120, 121] (Table 3.10). A meta-analysis on death and disability in sleep apnea confirmed a significant increase not only in cardiovascular but also non-cardiovascular deaths [23]. Nocturnal respiratory dysfunction (i.e., hypoxemia-reoxygenation and hypercapnia), poor sleep quality (i.e., increased arousals, poor sleep efficiency, decreased Stages N3 and REM), and intrathoracic pressure variations, in addition to shared comorbid risk factors (e.g., BMI and metabolic syndrome), promote oxidative stress, inflammation, sympathetic activation, endothelial dysfunction, neurohormonal changes, thrombophilia, and hemodynamic changes, which are the known pathophysiologic mechanisms of the adverse systemic outcomes in OSA [101] (Fig. 3.2).

3.10.2 Cardiovascular Death

Although Wang and colleagues' meta-analysis did not find an increased incidence in fatal and nonfatal CHD events [115], other meta-analyses reported significant association between severe OSA and cardiovascular death [23, 24, 30, 64]. A meta-analysis on CHF mortality reported an increased mortality only in those with central sleep apnea but not OSA [78]. A retrospective cohort study reported a higher incidence of sudden cardiac death (SCD) in patients with OSA with advanced age (>60 years), moderate-to-severe disease (AHI >20/hr.), and nocturnal hypoxemia (mean <93% and minimum <78% [28]. The severity of OSA appears to worsen QT prolongation in patients with congenital long QT syndrome, thereby raising the risk of SCD in this condition [102].

3.10.3 Chronic Obstructive Pulmonary Disease Mortality

A cross-sectional analysis of the National Health and Nutrition Examination Survey data found mortality to be similar in COPD patients with or without OSA [46]. In contrast, two prospective cohort studies reported significantly higher mortality in the overlap syndrome, particularly in those COPD patients with severe OSA [67]. The primary cause of death was COPD exacerbation.

3.10.4 Perioperative Mortality

A nationwide cohort study observed a counterintuitive reduction in postoperative mortality in OSA patients

Table 3.10 Strength of association between obstructive sleep apnea and survival outcomes

Survival outcomes	Strength of association, point estimate (95% confidence interval)	References	Study design
Death from all causes	HR = 1.19 (1.00, 1.41) for moderate OSA HR = 1.90 (1.29, 2.81) for severe OSA RR = 1.92 (1.38, 2.69) for severe OSA RR = 1.66 (1.19, 2.31) OR = 1.61 (1.43,1.81) RR = 1.59 (1.33, 1.89) for all-cause mortality HR = 1.262 (1.093, 1.431) HR = 0.945 (0.810, 1.081) for mild OSA HR = 1.178 (0.978, 1.378) for moderate OSA HR = 1.601 (1.298, 1.902) for severe OSA HR = 1.19 (0.86, 1.65) for mild OSA HR = 1.28 (0.96, 1.69) for moderate OSA HR = 2.13 (1.68, 2.68) for severe OSA RR = 1.54 (1.21, 1.97)	[23, 24, 30, 87, 115, 120, 121]	Meta-analysis Meta-analysis Meta-analysis Meta-analysis Meta-analysis Meta-analysis Meta-analysis
Cardiovascular death	OR = 2.09 (1.20, 3.65) HR = 1.40 (0.77, 2.53) for moderate OSA HR = 2.65 (1.82, 3.85) for severe OSA OR = 2.52 (1.80, 3.52) HR = 1.24 (0.53, 2.55) for mild OSA HR = 2.05 (0.57, 5.47) for moderate OSA HR = 2.73 (1.68, 2.68) for severe OSA	[23, 24, 30, 64]	Meta-analysis Meta-analysis Meta-analysis Meta-analysis
CHF mortality	RR = 1.09 (0.83, 1.42)	[78]	Meta-analysis
Non-cardiovascular death	OR = 1.68 (1.08, 2.61)	[23]	Meta-analysis
Sudden cardiac death	HR = 1.60 (1.14, 2.24) for AHI >20/hr	[28]	Retrospective cohort
COPD mortality	RR = 1.79 (1.16, 2.77) aHR = 1.5 (0.28, 2.80) HR = 2.01 (1.55, 2.62) for severe OSA	[21, 46, 67]	Prospective cohort Cross-sectional Prospective cohort
Postoperative mortality Orthopedic Abdominal Cardiovascular	OR = 0.65 (0.45,0.95) OR = 0.38 (0.22–0.65) OR = 0.54 (0.40–0.73)	[73]	Retrospective cohort analysis of a nationwide inpatient sample

Abbreviations: *HR* hazard ratio, *RR* relative risk or risk ratio, *OR* odds ratio, *OSA* obstructive sleep apnea, *CHF* congestive heart failure, *COPD* chronic obstructive pulmonary disease, *aHR* adjusted hazard ratio

who underwent orthopedic, abdominal, or cardiovascular surgery [73]. One proposed mechanism for this postoperative mortality reduction in OSA patients is the obesity paradox, i.e., the lower mortality observed in overweight or obese patients with CHF [61, 85, 103], acute coronary syndrome [81, 112], cardiovascular interventions [14, 61, 66], AF [89], pneumonia [80], lung cancer [104], and DM [53] (► Box 3.1). Another proposed mechanism is ischemic preconditioning, in which the intermittent hypoxemia due to OSA confers a wide array of protective end-organ effects (► Box 3.2). An observational cohort study provided some evidence for this concept of ischemic preconditioning based on lower troponin T levels and, as a corollary, less myocardial damage, in OSA patients suffering from acute myocardial infarction [99] (► Box 3.3).

Box 3.1 Obesity Paradox

Definition

The obesity paradox is the counterintuitive reduction in mortality observed in overweight or obese patients with CHF [61, 85, 103], acute coronary syndrome [81, 112], cardiovascular interventions [14, 61, 66], atrial fibrillation [89], pneumonia [80], lung cancer [104], and diabetes mellitus [53].

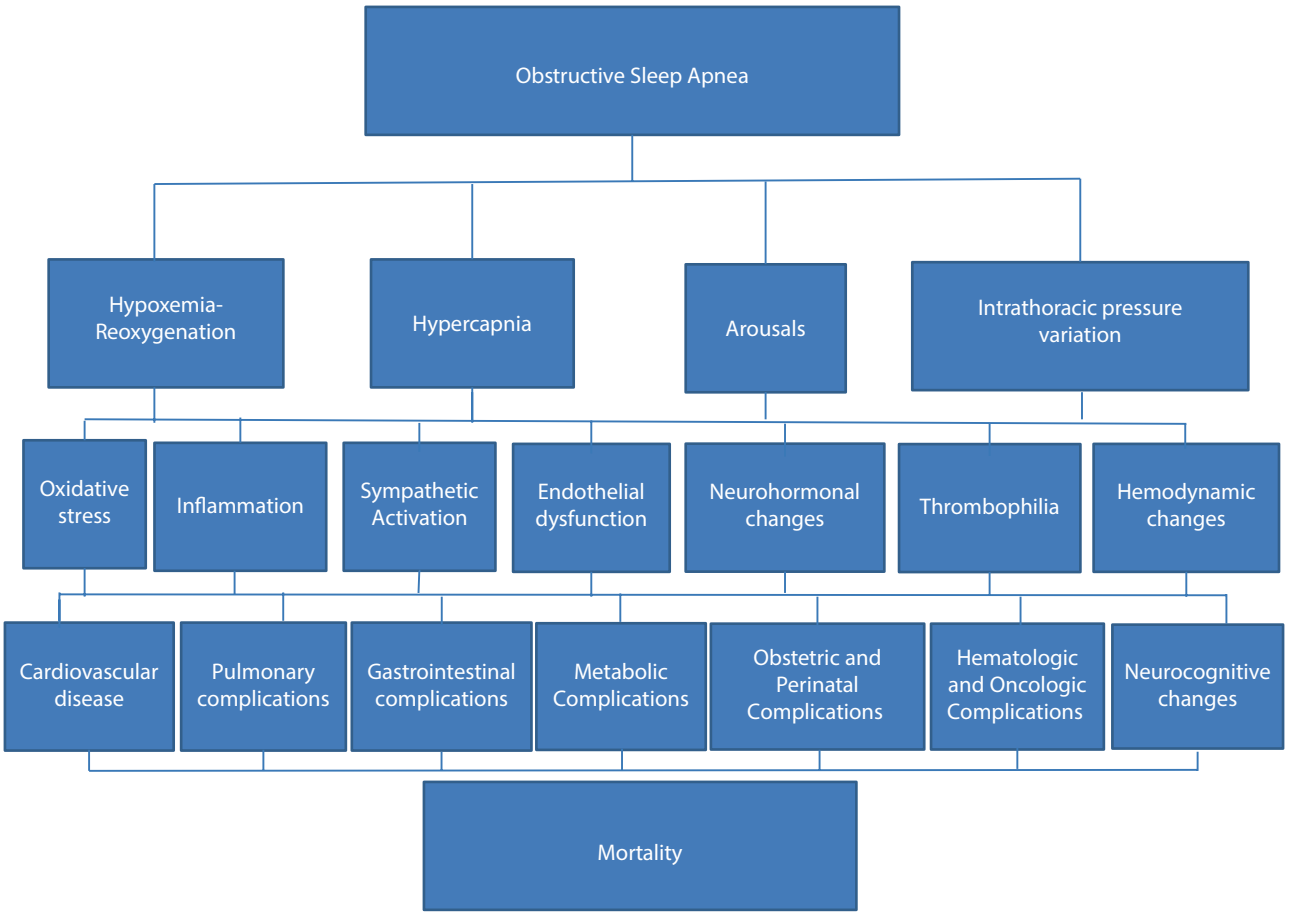

Fig. 3.2 Pathophysiology of the systemic adverse health consequences of obstructive sleep apnea

Box 3.2 Ischemic Preconditioning

Definition

Ischemic preconditioning refers to the purported wide array of protective end-organ effects conferred by intermittent hypoxemia due to OSA (e.g., lower troponin T levels) and, as a corollary, less myocardial damage, in OSA patients suffering from acute myocardial infarction [99].

Box 3.3 Questionnaire

Questions

1. Which of the following survival outcome measures is not adversely affected by obstructive sleep apnea?
   (a) Overall survival
   (b) Cardiovascular death
   (c) Non-cardiovascular death
   (d) Postoperative mortality
   (e) COPD-related mortality
2. Which of the following surgical outcomes is not adversely affected by obstructive sleep apnea?
   (a) Respiratory complications (e.g., hypoxemia, respiratory failure, need for intubation, and mechanical ventilation)
   (b) Cardiovascular complications (e.g., major cardiac events, atrial fibrillation)
   (c) Neurological complications (e.g., stroke)
   (d) ICU transfer
   (e) Length of stay

Answers

1. d. Postoperative mortality
2. e. Length of stay

Conclusion

The published literature on the adverse health consequences of OSA presents convincing evidence that OSA virtually affects every organ system, resulting in poor neurocognitive (i.e., hypersomnolence, fatigue, attention/vigilance, delayed long-term visual and verbal memory, visuospatial/constructional abilities, and executive function) and neuropsychological (e.g., depression, somatic syndromes, anxiety, and attention deficit/hyperactivity disorder), cardiovascular (i.e., CHF, systemic hypertension, ischemic heart disease, AF, ventricular arrhythmia, and stroke), respiratory (i.e., asthma and COPD exacerbation, pulmonary embolism, and pulmonary hypertension), endocrine (i.e., DM, metabolic syndrome, and sexual dysfunction), gastrointestinal (i.e., GERD and NAFLD), obstetric (i.e., pregnancy-related hypertensive disorders, gestational diabetes, maternal cardiovascular, pulmonary, and surgical complications), perinatal (i.e., low birth weight, preterm delivery, NICU admission, and hyperbilirubinemia), surgical (i.e., postoperative ICU transfer, respiratory complications, cardiovascular events, and neurologic complications), accident-related (i.e., motor vehicle crashes and work-related injuries), oncologic (i.e., cancer incidence), and survival (i.e., cardiovascular, non-cardiovascular, and COPD, and overall mortality) outcomes. Impairments in nocturnal respiratory function and sleep quality, in addition to comorbid conditions, result in oxidative stress, inflammation, sympathetic activation, endothelial dysfunction, neurohormonal changes, thrombophilia, and hemodynamic changes that lead to increased morbidity and mortality in patients with the OSA. On the other hand, perioperative mortality risk appears to be lower with OSA, purportedly due to the obesity paradox and ischemic preconditioning. Further research will help identify yet undiscovered adverse health effects of OSA, elucidate their pathophysiologic mechanisms, and propose preventive and therapeutic approaches to ameliorating these poor outcomes.

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Neurocognitive and Neuropsychological Effects of OSA

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Contents

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4.1 Patient Case

S.B. is a 57-year-old woman who presented to the general neurology clinic for evaluation of memory loss and overall cognitive decline. She first noticed the problem about 6 months prior to presentation. The rate of decline increased 3 months prior to presentation. She was working as a procedural nurse but transitioned to teaching due to the number of errors she was making. She reported that she was forgetting the names of friends, having trouble concentrating, and making paraphasic errors in conversations. She reported that her mood was depressed and irritable with no history of any psychiatric illness. When asked, she endorsed morning headaches, daytime fatigue, and difficulty maintaining sleep. An MRI of her brain identified a pituitary incidentaloma; pituitary hormone levels were checked and were normal. The brain parenchyma was normal. She was referred for home sleep apnea testing, which revealed an AHI of 26/h. CPAP was initiated as the only intervention between her first and second clinic visits. She returned to the clinic a month after starting CPAP and reported that her attention and cognition had normalized, her depression and irritability had resolved, and she was able to resume her usual job.

4.2 Introduction

The syndrome of obstructive sleep apnea (OSA) has been observed for hundreds of years, pre-dating the classic depiction in Dickens' *The Pickwick Papers* published in 1837 [1], but it was not until the late-1970s through mid-1980s that the full spectrum of consequences of OSA started to be recognized. While reports from the eighteenth and nineteenth centuries depict those suffering from OSA as obese, sleepy snorers, it is now known that the condition is far more dangerous than just excessive sleepiness. Sometimes lost among the cardiovascular and cerebrovascular consequences of untreated OSA are the neuropsychological effects and the cognitive impairment associated with this condition. This chapter explores the various mechanisms by which OSA could exert its deleterious effects on cognition and reviews the domains of cognitive impairment associated with untreated OSA.

4.3 History

Some of the earliest research on the cognitive dysfunction of OSA was performed by Guilleminault et al. [2] Fifteen of their 25 subjects (all male) reported intellectual deterioration, including poor attention and concentration. This study relied on self-report to determine the symptoms associated with upper airway obstruction. Beyond corroboration of intellectual function by speaking to the subjects' wives, no formal neurocognitive testing was performed.

Yesavage et al. [3] were among the first to formally study the cognitive consequences of OSA. They administered a neuropsychological battery to 41 non-demented, elderly men followed by nocturnal polysomnography. Five of the 11 test measures showed statistically significant impairments in men with sleep-disordered breathing compared to those without. The other six measures all trended the same way without reaching significance. The conclusion was that OSA has broad-ranging, negative cognitive consequences, but the mechanism remained unknown. The authors suggested that either daytime sleepiness or hypoxia-induced cortical insult were responsible for this impairment. However, hypoxia was deemed more likely due to impairments on both timed and untimed measures, with untimed measures being less susceptible to the effects of sleepiness. In addition, it was already known by that time that even mild hypoxia secondary to pulmonary disease led to neuropsychological impairments [4].

Around the same time, Kales et al. looked at 50 patients (7 women) with severe OSA who had been referred for tracheostomy [5]. Ninety-two percent of these patients showed deficits in "thinking, perception, or memory." These early studies laid the groundwork that established the extent of neurocognitive impairment in OSA but did not establish a mechanism.

4.4 Mechanisms of Action

4.4.1 Oxygen

OSA is associated with a wide range of physiological changes. One of the most prominent effects of OSA is recurrent periods of oxygen desaturation. Both the presence of low oxygen and the cycles of desaturation have been studied to determine their roles on cognition.

Findley et al. [6] studied 26 subjects with OSA who underwent neuropsychological testing followed by nocturnal polysomnography. Those with OSA were divided into groups with and without hypoxemia. Hypoxemia was determined using the median SaO2 and the number of desaturations of greater than 4% per hour of sleep. On four of the 8 tests, those who had OSA and hypoxemia performed significantly worse. The degree of hypoxemia also correlated to the degree of impairment. Although those with hypoxemia during sleep were also noted to have lower awake baseline SaO2, they were not compared to patients with low baseline SaO2 from pulmonary disease who did not have OSA. The hypoxemic group also had more sleep fragmentation and less slow-wave sleep, but these factors did not correlate to impairment.

To investigate the relative contributions of sleepiness and hypoxia to the cognitive deficits observed in OSA, Greenberg et al. studied 38 subjects (14 with OSA, 10 with other causes of hypersomnia, and 14 healthy controls) [7]. Only subjects with normal awake SaO2 levels were included. Age-related cognitive changes were controlled for by excluding subjects older than 55. Hypoxemia was determined using the lowest recorded desaturation and the total time not breathing. The subjects with OSA but not those with hypersomnia showed deficits relative to the healthy controls. They also identified hypoxemia severity as a significant correlate to some of the deficits observed.

Later, Roehrs et al. compared 25 male subjects with OSA to 24 men with COPD. [8] Both groups were hypoxic overnight, but the OSA group demonstrated greater sleep disruption and greater daytime sleepiness. Neuropsychological performance was equally impaired on most tests, but the OSA group performed worse on a test of sustained attention, presumably related to the increased sleepiness. The COPD group performed worse on a test of motor skills. The similarity of the OSA group to the COPD group suggested a larger role for hypoxia than sleep disruption as the primary driver of cognitive dysfunction.

This finding was further supported in an animal model by Gozal et al. [9]. To eliminate sleep disruption as a confounder, rats were subjected to 14 days of an intermittently hypoxic environment during their sleep periods. Sleep patterns normalized to these conditions after just 1 day. Behaviorally, the rats performed worse in a water maze test than did controls. This effect lasted for at least 2 weeks after completion of the episodic hypoxia exposure. The rats were then examined to identify the effect episodic hypoxia had on the brain, leading to the discovery of a variety of abnormalities. For example, the hippocampal CA1 cellular arrangement was disrupted with enhanced c-fos expression in all 8 rats in the experimental condition. Single-stranded DNA showed a marked increase in CA1 and the neocortex indicating enhanced apoptosis. Behavioral changes in the rats were observed even 14 days after returning to normoxic conditions.

Recurrent hypoxemia overnight, a consequence of OSA, has been shown to generate reactive oxygen species (ROS) that are responsible for oxidative stress [10]. OSA also appears to induce ROS through increased release of superoxide dismutase from neutrophils [11]. Oxidative stress is associated with both Alzheimer's disease and vascular dementia [12]. There are a variety of hypotheses as to how oxidative stress affects cognition, including causing vasoconstriction with reduced cerebral blood flow, upregulation of beta-amyloid production (the protein found in Alzheimer's disease plaques), and enhanced atherosclerosis through the oxidation of low-density lipoproteins (LDL).

Extensive evidence has been collected that demonstrates the role of hypoxia in the development of cognitive impairment due to OSA. Hypoxemia might be the most direct route of cognitive impairment through neurotoxic and oxidative stress pathways, but it is only one factor affecting the cognition of patients with OSA. There are likely numerous indirect contributors as well.

4.4.2 Vascular

Vascular risk factors including hypertension and hyperlipidemia have been shown to increase the risk of cognitive impairment [13, 14], and OSA is a significant contributor to the development of these conditions. The neurobiological changes that lead from vascular risk factors to cognitive impairment are not well defined. One hypothesis is that the microvascular damage from these factors accelerates capillary changes affecting brain perfusion at the microvascular level. [15] In turn, white matter disease disrupts cortical connectivity. Hypertension, in particular, has been associated with stroke, cerebral atrophy, reduced cerebral blood flow, and cellular dysfunction, each of which can impact cognition [14]. Lavie et al. showed that among adults aged 20–85 years, systolic and diastolic blood pressure was linearly related to the severity of OSA, independent of age, sex, and BMI [16]. Nieto et al. found a similar effect controlling for ethnicity as well [17]. In their study, the odds ratio of having hypertension was 2.27 when comparing the highest and lowest apnea–hypopnea indices (>30/hour to <1.5/hour). Thus OSA, by raising blood pressure, could lead to significant cognitive deficits.

HDL cholesterol could also play a role in cognitive impairment through multiple mechanisms [14]. High HDL is associated with reduced cardiovascular disease and lower plaque burden. It is involved in reverse cholesterol transport and has antioxidant properties, among other benefits [18]. HDL could, therefore, improve brain health by preventing the reduction in blood flow and damage from free radicals. Additionally, Wolf et al. showed that HDL levels and risk of dementia were inversely correlated in 86 elderly male and female subjects (relative risk 1.9 for the lowest quartile of HDL) [19]. They also found that low HDL (but not LDL or total cholesterol) was correlated with hippocampal volume loss.

Intriguingly, it is specifically HDL that is affected by OSA, rather than LDL, total cholesterol, or triglycerides [20]. Tan et al. demonstrated that OSA contributed to HDL dysfunction by measuring the ability of HDL to inhibit LDL oxidation in 128 subjects with sleep apnea compared to 82 controls [21]. Subjects with OSA had higher levels of oxidized LDL, and this was most closely tied to AHI. It appears OSA's effect on HDL could be another mechanism promoting cognitive decline.

4.4.3 Endocrine

The risk of insulin resistance is increased in patients with OSA [22]. Ip et al. studied 270 (197 male) subjects referred for polysomnography who did not have diabetes [23]. Those found to have OSA showed significantly higher levels of fasting serum insulin. Although obesity also predicted both OSA and insulin levels, the presence of OSA was found to be an independent predictor of insulin. Subsequent work by Peled et al. confirmed a correlation between the severity of OSA and the degree of insulin resistance [24]. It is important to note that not all researchers have come to the same conclusion about insulin resistance in OSA [25], but this appears to be due to the definition of insulin resistance that was used.

How OSA causes insulin resistance is not entirely clear, but Iiyori et al. have proposed the key factor is intermittent hypoxemia [26]. In a mouse model, they were able to demonstrate insulin resistance in otherwise lean, healthy mice by exposing them to intermittently hypoxic conditions. However, Polotsky et al. found that insulin resistance only developed in leptin-deficient, obese mice exposed to intermittent hypoxia, but not in healthy mouse controls [27]. Their conclusion was that disruption of the satiety hormone, leptin, was the key to developing insulin resistance in OSA.

Insulin has been shown to increase the release of intracellular beta-amyloid and accelerate trafficking to the plasma membrane [28]; thus, insulin resistance could lead to an accumulation of beta-amyloid in neurons with potentially toxic consequences. Insulin also appears to play a critical role in memory that could be impaired in the setting of insulin resistance. Insulin receptors are found in high numbers in the hippocampus [29], and treating patients with cognitive impairment with intranasal insulin showed positive effects on cognition, specifically verbal memory and attention [30]. These mechanisms could partly explain the cognitive impairment seen in OSA.

Hyperglycemia from insulin resistance also contributes to microvascular disease and adds advanced glycation end products and oxidative stress to the list of OSA-induced factors that contribute to cognitive decline [31]. Hyperglycemia also appears to contribute to beta-amyloid aggregation [32]. In a mouse model, elevated quantities of amyloid without hyperglycemia showed no aggregation, indicating a pathogenic role for hyperglycemia in amyloid deposition, which could be a direct downstream consequence of OSA.

Other endocrine abnormalities also contribute to the cognitive dysfunction seen in untreated OSA. Obesity, a common comorbid condition with OSA, is associated with hypercortisolism [33], which in turn can affect the hippocampus and impair learning and memory [34]. However, it is not clear if hypercortisolism is a direct effect of OSA or not [35]. Leptin, a hormone commonly implicated in regulating feeding behavior, appears to play a role in memory, as well. Leptin receptor-deficient mice showed longer swim times in the Morris water maze test and impaired long-term potentiation and long-term depression in the CA1 region of the hippocampus [36]. Serum leptin is elevated in OSA [37], which is thought to be a result of leptin resistance. Thus leptin, cortisol, and insulin and their corresponding effects on glucose metabolism create the milieu that supports cognitive impairment.

Low testosterone has been associated with both OSA and cognitive impairment. As part of the Baltimore Longitudinal Study of Aging, Moffat et al. followed 407 men for an average of 10 years and found that lower testosterone levels predicted lower scores on memory and visuospatial/visuomotor tasks as well as a faster rate of decline [38]. Subsequently, Beer et al. showed that men on androgen-depleting therapy for prostate cancer performed significantly worse on tests of memory than age-matched healthy controls [39]. Similarly, OSA is also thought to lower testosterone levels. In a small study of 30 obese men (15 with OSA and 15 without OSA matched on anthropomorphic measures), the presence of OSA was correlated to lower total and free testosterone [40]. This study also found the severity of hypoxia correlated with lower testosterone. Other small studies have supported these findings [41, 42].

Testosterone appears to affect cognition via androgen receptors in the brain [43]. Androgen receptors are found only in certain brain regions, but these include regions critical for learning, including the hippocampus [44]. Leranth et al. have studied the role of gonadal hormones on monkey hippocampi and discovered low CA1 spine synapse density in female monkeys who underwent oophorectomy [45], followed by an even lower density in male monkeys after orchiectomy [46]. These findings support the role of sex hormones in memory.

There is less known about the effect of OSA on female sex hormones. There is evidence that women with OSA have lower levels of estradiol and progesterone, even after accounting for age and menstrual status [47]. However, it is not clear that low female sex hormones are a consequence of OSA rather than a cause. While testosterone therapy is suspected (though not established) to worsen OSA [48], estrogen and progesterone therapy have been shown to reduce OSA [49]. Furthermore, in women with regular menstrual cycles, the rate of OSA was lowest during the follicular phase when estrogen was highest, suggesting female hormone fluctuations might drive OSA rather than vice versa [50]. While low female sex hormones might affect cognition, it cannot be concluded that OSA is a contributing factor.

4.4.4 Inflammation

In addition to hormones, inflammatory cytokines have been shown to contribute to cognitive impairment [51]. In a study of 3031 subjects with a mean age of 74 years, IL-6, TNF-alpha, and CRP were measured at baseline and over 2 years of follow-up. Subjects in the highest tertile of IL-6 and CRP demonstrated the highest likelihood of cognitive decline (odds ratios of 1.34 and 1.31, respectively). TNF-alpha did not predict decline (odds ratio 1.1).

Ample evidence supports elevations of pro-inflammatory cytokines in OSA [52]. Vgontzas et al. compared patients with sleep apnea to patients with other sleep disorders and normal controls and found elevations in TNF-alpha and IL-6 in patients with sleep apnea. [53] A similar effect was found for CRP [54, 55].

Additionally, the intracellular and vascular cell adhesion molecules sICAM-1 and sVCAM-1 were shown to be elevated in subjects with OSA, suggesting a link between OSA and vascular disease via these inflammatory mediators. Although sICAM-1 and sVCAM-1 were not shown to be associated directly with dementia in The Rotterdam Study [56], others have shown a link between these markers and cognitive decline [57] or impaired psychomotor function due to silent brain infarction and white matter lesions on MRI [58].

Evidence suggests that it is once again intermittent hypoxia that induces these inflammatory changes. Lam et al. demonstrated increased amounts of IL-1beta, IL-1r1, IL-6, gp130, TNF-alpha, and TNFr1 in the carotid bodies of rats exposed to intermittently hypoxic conditions [59]. Likewise, using HeLa cells, intermittent hypoxia led to elevations in NFkB, a pro-inflammatory transcription factor [52]. He et al. studied both cells and rats under various degrees of intermittent and sustained hypoxia [60]. They found that NF-kB was significantly correlated with the severity of intermittent hypoxia. Intermittent hypoxia was more inflammatory than sustained hypoxia or normoxia. Levels of TNF-alpha, IL-6, and leptin were highest in the most severe intermittent hypoxic group.

4.5 Neuroimaging Studies and Cognition

OSA causes many physiological changes that together lead to neurocognitive impairment. Some of these changes are visible with neuroimaging. Current literature on neuroimaging findings in OSA patients is compelling and provides additional understanding of the relationship between brain pathology, morphology, and functioning. Recent literature has spanned several methodologies for analyzing the structural, functional, and metabolic compositions of gray and white. Numerous studies have also explored quantitative electroencephalography (EEG) with event-related potential paradigms in OSA. While a comprehensive review of neuroimaging in OSA is beyond the scope of this chapter, a few studies are mentioned here as they include cognitive outcomes and analysis of neuroimaging correlates to neuropsychological outcomes (see Ferini-Strami et al. 2013 for a more comprehensive review of neuroimaging in OSA) [61]. For example, Canessa et al. conducted baseline neurocognitive assessment and quantitative MRI (voxel-based morphometry or VBM) in treatment-naïve OSA patients and controls, with follow-up measurement after 3 months of CPAP treatment [62]. At baseline, individuals with OSA demonstrated significantly worse neurocognitive functioning. In addition, this study demonstrated that prior to treatment, OSA patients showed focal volume reductions to left hippocampus gray-matter (GM), right superior frontal gyrus, and the left posterior parietal cortex. Following 3 months of CPAP treatment, the OSA group demonstrated significant change to brain regions, with increases in both frontal and hippocampal regions.

Torelli et al. also examined cognition and structural brain volume via VBM among individuals with untreated moderate to severe OSA and matched controls [63]. Neurocognitive assessment revealed significant group differences in a broad range of cognitive domains, with OSA patients performing worse on all accounts. In addition, VBM analysis demonstrated that OSA patients had significantly lower total gray matter volume, right hippocampus volume, caudate volume bilaterally, and left thalamus volume. As expected, this study also identified multiple associations between regional brain volumes and performance on cognitive testing.

Joo et al. analyzed cognition and structural brain volume (cortical thickness) in multiple regions of interest among individuals with OSA (untreated) relative to healthy controls [64]. This study identified multiple brain regions linked to cortical thinning in OSA, including the dorsolateral prefrontal cortex, the ventromedial prefrontal cortex, precentral/postcentral gyri bilaterally, the anterior cingulate, left insula, lateral temporal regions, right supramarginal gyrus, right precuneus, left inferior parietal cortex, bilateral uncas, left parahippocampal gyrus, and the right fusiform gyrus. Visual memory recall was correlated with right parahippocampal and right uncal cortical thickness. These authors suggested that these cortical volume changes are likely related to the cognitive changes often observed in OSA patients, particularly aspects of attention and executive functioning.

In addition to changes in hippocampal and entorhinal cortex volume and associated memory changes, additional research has also identified diminished mam-

millary body volume among individuals with mild to severe untreated OSA, and authors suggest that mammillary body volume may be important to the relationship between OSA and memory functioning [65].

Additional research has also specifically examined white matter integrity with diffusion tensor imaging (DTI) [66]. One particularly interesting longitudinal study examined patients with severe OSA pre-treatment and then again at 3 and 12 months post-treatment. Prior to treatment, OSA patients demonstrated reduced cognitive functioning in most of the tested areas, especially executive functioning. In addition, the OSA group also demonstrated reduced white matter integrity in multiple brain regions, including both left and right hemisphere regions. Importantly, after only 3 months of CPAP treatment, fewer WM changes were identified and by 12 months, there was a near-complete reversal of WM changes, and cognitive functioning -- particularly executive functioning -- also improved to some degree.

Neuroimaging studies support the notion that structural brain changes relate to OSA and likely contribute to the neuropsychological changes that take place in these patients. The next section discusses the numerous neuropsychological domains that are affected by OSA. It is likely that the variety of effects that have been observed is related to the mechanisms discussed above. The relative contributions of cerebrovascular disease, hormonal changes, hypoxemia, and inflammation all contributing to structural changes in the brain could account for the wide spectrum of neuropsychological impairments associated with OSA.

4.6 Neuropsychological Functioning in OSA

OSA has broad ramifications for neuropsychological function. Extensive research has shown both objective evidence of attenuated functioning [67] as well as frequent subjective cognitive complaints among individuals with OSA [68]. The following sections review what is known about the effects of OSA on a broad spectrum of cognitive domains, including language, attention and executive function, visual-spatial, memory, and affective functioning.

4.6.1 Attention & Executive Function

With extensive literature on the impact of OSA on executive functioning, the evidence is largely conclusive that this is a particularly vulnerable cognitive domain, with one comprehensive review suggesting that 60% of published works identify impaired attention/vigilance, and executive skills in OSA patients [69]. While there are several sub-domains of executive functioning, the general domain is thought to be functionally related to frontal and subcortical regions of the brain. As such, a discussion on executive functioning will include both broad executive tasks and attention, processing speed, and vigilance.

A large 15-year longitudinal study examined multiple aspects of neurocognitive performance among individuals with normal sleep versus individuals stratified for OSA severity [70]. While this study failed to identify neurocognitive differences between groups in several domains, it did find that those with severe OSA demonstrated reduced processing speed on a measure of visual search and attention.

In a sample of untreated moderate to severe OSA patients, authors demonstrated that OSA patients display attenuated attentional control and diminished reaction times compared to controls [71]. In a similar sample of untreated OSA patients versus matched controls, OSA patients displayed significantly worse decision-making abilities [72]. Arli et al. also indicated that OSA patients demonstrate reduced working memory, processing speed, and inhibitory control relative to individuals with nonhypoxic simple-snoring [73].

In addition to these studies comparing OSA to controls, additional literature has examined the impact of OSA on cognition with additional common comorbidities. For instance, in a study examining OSA impact on cognition, Bajaj et al. examined controls versus three patient groups – those with OSA, with OSA and cirrhosis, and those with cirrhosis only [74]. While this study had small sample sizes, it had the advantage of measuring cognition on treatment-naïve OSA patients and again post-treatment. None of the cirrhosis patients had hepatic encephalopathy or ascites. This study specifically focused on measures of processing speed, attention, and executive functioning with a specific aim at delineating whether there was an interaction between OSA and cirrhosis. Psychomotor speed was worse in groups with cirrhosis compared to those with OSA alone. However, groups with OSA demonstrated attenuated executive functioning compared to groups without OSA, including performance of inhibition, multitasking, and complex coding. In addition, performance was even worse among patients with OSA and cirrhosis. After treatment with CPAP (average of 2.5 months of treatment), OSA patient groups demonstrated improved executive functioning.

Next, given that OSA is common in stroke patients, a case–control study by Aaronson et al. examined a broad range of neurocognitive functional abilities in a group of post-stroke patients, and measured group differences between individuals who had OSA and those who did not [75]. While this study did not include a healthy control group, it is interesting to examine the relative differences in neurocognitive functioning of post-stroke patients while examining the impact of OSA. This study documented that post-stroke patients with OSA demonstrated attenuated executive functioning on measures

of mental flexibility and problem-solving ($d = 0.42$) and sustained and selective attention ($d = 0.48$) relative to post-stroke patients without comorbid OSA. These values indicate a moderate-sized effect.

A comprehensive meta-analysis of a broad range of neurocognitive functions in OSA relative to controls identified medium to large effect sizes for executive function ($d = 0.73$) and vigilance ($d = 1.40$) [76]. A subsequent meta-analysis demonstrated that OSA resulted in small to medium effect sizes for processing speed, concept formation, broad executive functioning, attention, working memory, and verbal reasoning [77].

Olaithe and Bucks used meta-analysis techniques to examine a broad range of executive functioning measures in OSA patients before and after CPAP treatment [78]. As expected, this study found that patients with OSA pre-treatment displayed moderate to strong effect sizes, with worse functioning in all aspects of executive function, but with improved executive functioning post-treatment. Similarly, Kylstra et al. also found that attention deficits in OSA have a slight improvement with CPAP treatment [79]. On the other hand, additional research postulated that executive deficits common in moderate to severe OSA may be relatively resistant to improvement with CPAP treatment [80].

While there are mixed results from the studies above, it is important to note significant methodological weakness in many OSA-cognitive studies, including small samples, poor treatment control, and inadequate control groups. Kushida et al. attempted to overcome these methodological shortcomings in a 6-month, double-blind, randomized, sham-controlled, multicenter trial study [81]. Though there was mild attrition, the study had a larger sample than most other studies, with at least 400 subjects in each group. Cognitive outcomes were also stratified based on OSA severity. Overall, the study demonstrated that the sham treatment group displayed significantly reduced complex working memory 2 months into the study relative to those with active treatment. However, there were no significant differences between groups at 4 and 6 months.

In summary, several individual studies, reviews, and meta-analyses suggest that OSA is frequently related to attenuated executive functioning, attention, and vigilance. Additionally, there is evidence that CPAP treatment leads to at least partial improvement in executive task performance over time.

4.6.2 Learning and Memory

The literature on learning and memory among OSA patients is variable, with some studies suggesting little to no difference in memory among OSA patients relative to controls, and other pointing to a strong difference.

For example, Aaronson et al. examined the impact of OSA on memory in two groups, including post-stroke patients without OSA and post-stroke patients with comorbid OSA [75]. The researchers examined verbal memory functioning with a common list learning task. When participants presented with aphasia, a nonverbal task was substituted, which included a test of location learning. There were no significant differences on these memory tests in the post-stroke patients with and without OSA. Similarly, Lutsey et al. used a 15-year longitudinal design to examine whether the severity of OSA was predictive of neurocognitive status, including delayed verbal memory [70]. This study failed to identify any differences in verbal memory between individuals with normal sleep status and those with OSA of any severity. While this study had some methodological shortcomings, it also suggested there was no change in delayed verbal memory for OSA patients over a 15-year study period.

The findings above are at odds with several other investigations, including a 2010 study by Twigg et al., which demonstrated impaired immediate and delayed contextual verbal memory in OSA relative to controls [82]. In another comparative population-based study, researchers examined delayed verbal memory performance among individuals with high versus low OSA risk [83]. This study demonstrated that those with a high-risk level of OSA displayed marginally lower verbal memory functioning relative to those with low risk. As noted above, however, this study assessed OSA risk via questionnaire and not laboratory testing, which is a methodological weakness for group classification.

It is clear that several years of research have resulted in disparate findings. Overall, however, with one comprehensive review of cognitive functioning and OSA suggesting that 60% of research articles identify memory impairments in OSA patients, it becomes important to systematically analyze findings through meta-analytic techniques [69]. One meta-analytic review of memory functioning in OSA versus controls demonstrated small but significant effect sizes for short-term verbal learning and small to medium effect sizes for long-term verbal memory recall ($d = 0.27$ to $.52$). Effect sizes for visual learning and recall were negligible and non-significant [76]. In a more recent and more methodologically rigorous meta-analysis, the authors demonstrated that OSA resulted in small to medium effect sizes for both verbal and non-verbal memory, although the study did not differentiate immediate learning from long-term recall. [77]

In addition, the pattern of memory deficits is also important to consider when evaluating neuropsychological outcomes in OSA. That is, a breakdown in memory is often thought to involve frontal systems, medial temporal systems, or thalamic systems, and each of these systems typically presents with a unique pattern on testing. Few studies have examined the specific memory characteristics

in OSA. However, an early study by Naëgelé et al. suggested that memory weaknesses in OSA patients appear to be related to poor acquisition and poor retrieval, but with relatively preserved recognition discrimination [84]. This suggests at least some executive influence on memory in OSA, and that storage (i.e., medial temporal lobe function) may be less influential than executive functioning in understanding memory functioning in OSA. This executive pattern of memory impairment is supported by additional research that demonstrated that OSA patients display poor immediate recall (i.e., encoding) across repeated learning trials on a verbal memory task, and reduced semantic clustering of a word list, but that recall and recognition remain intact [85]. Again, these findings support the premise that the impact of OSA on memory is at least partially related to executive functioning.

In summary, variable findings in memory outcomes in OSA patients may be a factor of the heterogeneity of patient populations and inconsistent testing parameters between studies. However, there is compelling cumulative evidence of attenuated learning and recall in OSA patients, with at least some impact from executive aspects of the tasks, including diminished acquisition, poor learning strategies, and poor retrieval, but better performance with cueing.

4.6.3 Language[1]

Variable findings are reported in the research literature regarding the effect of OSA on verbal fluency. For example, Aaronson et al. compared the semantic verbal fluency performance of post-stroke patients with and without OSA [75]. When examining performance on semantic verbal fluency tests, there were no significant statistical differences between post-stroke patients with and without OSA.

Addison-Brown et al. used population-based data to examine high versus low OSA risk on semantic and phonemic verbal fluency performance [83]. Here, semantic fluency was significantly worse for those with high OSA risk while OSA risk failed to predict phonemic verbal fluency performance. Note that this study had clear methodological weaknesses relative to other studies of OSA and cognitive functioning. First, the OSA risk was measured via questionnaire assessing snoring, daytime sleepiness, and an index of hypertension/BMI. In addition, only 11.6% of the sample reported a previous diagnosis of OSA and only 10% reported receiving treatment. While the authors suggest that the questionnaire utilized is predictive of respiratory distress, this is qualitatively different than a medically confirmed diagnosis of OSA.

Lutsey et al. used a 15-year longitudinal design to examine whether the severity of OSA was predictive of neurocognitive status [70]. This study failed to identify any differences in verbal fluency between individuals with normal sleep status and those with OSA of any severity. The study also noted that there was no change in verbal fluency over the study period among those with OSA. One limitation of this study, however, is that it only included one measurement time point of OSA and failed to control for the treatment status of individuals with OSA. Overall, there appear to be inconsistent findings when it comes to verbal fluency in OSA patients.

Next, non-fluency aspects of language have been examined in meta-analyses, again with disparate conclusions. One meta-analysis examining lexical and receptive/expressive fundamentals found no significant difference in verbal and language functioning between groups [76]. However, in a more recent and more methodologically rigorous meta-analysis, the authors demonstrated that OSA resulted in small but significant effect size in the domain of language/verbal functioning, with OSA patients performing worse than controls [77]. It is noteworthy that the effect size was a composite of three studies with measures of language including a test of confrontational visual naming, vocabulary knowledge, and fund of information knowledge. As such, this finding may be characteristically different than those examining verbal fluency.

In summary, there appear to be disparate findings in the literature in terms of verbal fluency performance. Two separate meta-analyses on non-fluency aspects of language functioning also appear at odds, although a more rigorous study demonstrated small but significant effects. That is, early meta-analyses suggested little to no impact from OSA on verbal/language functioning, while more recent meta-analysis suggested small but significant differences in areas of vocabulary and visual confrontational naming. Overall, findings of a language change in OSA are less compelling than those associated with attention and executive functioning.

1 It is important to recognize that there are measures of language that some researchers and clinicians often categorize as being executive in nature. Specifically, while verbal fluency tasks require engagement of language regions of the brain and are often markedly impaired in patient groups with language disorders, these tasks also engaged areas of the frontal lobes, particularly phonemic fluency. In fact, some researchers place verbal fluency tasks solely in a language functioning category, while others classify these tests as measures of executive functioning given the frontal lobe involvement. For simplicity, this chapter will classify verbal fluency tasks as language-based measures. Other measures of language might include confrontational naming tasks, vocabulary knowledge, fund of information knowledge, comprehension tasks, or reading and writing tasks. While all these measured areas can be grouped in language, it is also noteworthy that certain verbal tasks are considered "stay functions" in neuropsychology, meaning they are relatively stable in the face of neurological disease or injury. Vocabulary knowledge, for example, is often considered a stay function.

4.6.4 Visual-Spatial

The above noted case–control study of a stroke patient with OSA by Aaronson et al. also examined visual perception [75]. Patients were asked to complete two visual perception tasks examining their ability to search and identify target items in an array of distractor items. Compared to individuals with no history of OSA, those with OSA demonstrated significantly reduced visual perception abilities ($d = 0.35$).

Two large meta-analyses provided consistent results on the impact of OSA on visual-spatial and constructional abilities. First, a meta-analysis consisting of a broad range of visual perceptual, graphomotor reproduction, and construction tasks demonstrated moderate effect size between groups, with OSA patients performing worse [76]. This is supported by a later meta-analysis with additional literature, which demonstrated similar moderate effects [77].

In summary, there is compelling evidence that OSA is associated with at least some degree of visual-spatial deficits relative to their healthy counterparts. In clinical practice, however, a neuropsychologist would be tasked with delineating whether reduced visual-spatial performance on an isolated task was due to gross deficits in visual perception or simply from diminished executive functioning. In other words, poor planning, reduced organization, of impulsivity may result in attenuated task performance independent of intact visual perception. The meta-analyses noted above failed to discuss such difference, but they are important distinctions in clinical settings and from a functional neuroanatomy standpoint.

4.6.5 Affective Functioning

Understanding the relationship between OSA and mood is important in clinical care. Specifically, studies have demonstrated that OSA is associated with an overall increase in affective disorders, reduced quality of life, increased daytime sleepiness and fatigue, and exhaustion. Depression is also associated with decreased treatment compliance in OSA patients [86, 87]. The relationship between OSA and these factors is possibly confounded by the presence of common comorbidities among OSA patients, including higher rates of diabetes, obesity, cardiovascular disease, and reduced cognitive functioning [87]. As such, while depression is common in OSA, the entire clinical presentation of any given patient must be considered in understanding the nature of depression in this patient group.

Multiple studies have examined the rate of depression among OSA patients in community and in clinical populations [88]. A large population-based study (n = 19,980) with five European countries included a sample of 857 individuals with OSA. Among this group, 17% were identified as having depression [89]. In another large sample made up of US veterans [90], 21.8% of those with OSA were identified as also having depression. In addition, 16.7% were identified as having anxiety disorders, and 11.9% had PTSD. These figures were significantly higher than the veterans without OSA. Although this study compared veterans with and without OSA, a selection bias may account for a slightly higher rate in this group given that veterans may already have higher rates of affective disorders. Thus, 21% may reflect the upper limit of depression prevalence in community samples. Higher rates of depression are reported in clinical samples. For example, Acker et al. recruited a large clinic sample of 447 prospective patients referred to a sleep center [91]. Of these, 322 had an AHI >9, and the overall rate of depression in this group was 21.5%. Note that this is similar to the 21% found in veterans with OSA. However, one clinic-based study suggested that 41% of a Dutch OSA sample met criteria for at least mild depression [92].

Multiple cross-sectional studies have examined depression occurrence in OSA. Canessa et al. demonstrated that treatment-naïve OSA patients showed significantly higher depression scores on a self-report mood measure at baseline compared to healthy controls, and that mood significantly improved after 3 months of CPAP treatment [79]. These findings are similar to another study of untreated OSA patients versus controls, in which those with baseline untreated OSA showed relatively higher levels of depression and anxiety [69]. Jackson et al. likewise demonstrated that OSA patients report higher levels of depression relative to their non-OSA counterparts, but also suggested that the association between OSA and depression is mediated by sleepiness and fatigue [93].

Given the cross-sectional nature of these studies, the identified relationship between OSA and depression is simply correlational, and the directionality of this relationship is unclear. A few longitudinal studies have examined OSA and depression in an attempt to establish a causal association. One study followed a large cohort of working-age adults, with a broad range of AHI frequencies, and measured depression every 4 years. The results of their analysis demonstrated that as sleep-disordered breathing increases as measured by AHI, the adjusted odds of developing depression also increased [94].

An additional study examined the impact of OSA on depression occurrence over 1 year. At follow-up, the incidence of depression was twice as common among the OSA patients relative to those without OSA, with a Cox proportional hazards model suggesting a 2.18 times increased risk of developing depression in OSA patients by 1 year, with a slightly higher risk among women [95].

In summary, there is a clear association between OSA and depression, with some studies demonstrating a causal relationship. Clinical samples appear to have higher reported rates of depression relative to community samples. In addition, research suggests there are moderating variables in this association, such as the presence of significant comorbidities. Fatigue and daytime sleepiness are known to play a role in the degree of depression in OSA patients, and clinicians must practice caution in differentiating depression symptoms from common OSA complaints, given the high degree of symptom overlap.

4.7 Summary

OSA is a condition with a myriad of downstream effects, including attention and executive dysfunction, learning and memory impairment, visual-spatial dysfunction, depression, and mild language impairment as well. These effects are likely due to OSA causing any combination of the following: cyclic oxygen desaturation, free radical formation, hypertension, dyslipidemia, insulin resistance, low testosterone, leptin resistance, and elevations in inflammatory cytokines. These changes cause substantial real-world consequences for patients, including job loss, major depressive disorder, or even Alzheimer's disease [96]. Attention must be paid to these oft-overlooked consequences of OSA to ensure appropriate treatment is provided.

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Diagnostic Considerations in Metabolic Disease Associated with Obstructive Sleep Apnea

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Contents

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Core Message

- Recognition and diagnosis of metabolic diseases associated with sleep apnea is important to avoid adverse outcomes of treatment.
- In perioperative management of OSA, associated metabolic diseases not only increase risk for cardiovascular events, but also increase risk for perioperative bleeding, infection, and slow wound healing.
- To aid recognition and diagnosis of metabolic disease, we recommend use of recent diagnostic criteria for relevant metabolic diseases associated with sleep apnea, which include obesity, hypertension, diabetes, and liver disease.
- Recognition and treatment of metabolic disease is handled most efficiently by a multidisciplinary sleep disorders center that makes optimal use of diagnostic and therapeutic protocols in the context of electronic health records.

5.1 Introduction

While accurate diagnosis of obstructive sleep apnea (OSA) remains the core objective of this section on diagnostic considerations, one does not want to miss potentially dangerous metabolic disease associated with sleep apnea. Recognition and diagnosis of metabolic disease is important to avoid adverse outcomes of treatment, especially if treatment includes physiological stress of general anesthesia and surgical procedures.

Metabolic control requires cross-talk among various organs and signaling pathways. The relationship between OSA and metabolic disease represents a complex interplay among organs and tissues (see Fig. 5.1).

In this chapter, we consider current recommendations for diagnosis of major metabolic diseases associated with OSA, to include obesity, hypertension, diabetes mellitus, and fatty liver disease. Presence and severity of these closely related diseases will influence decisions on exercise and perioperative treatment through increased risks for cardiovascular events, coagulopathy, infection, and slow wound healing.

Given the practical nature of this book, we will not undertake exhaustive review of diagnostic criteria generated by multiple organizations, but instead focus on the most recent definitions of metabolic disease that seem appropriate to practitioners of sleep medicine. To avoid redundancy, we will not cover metabolic syndrome, which is not a disease, but a syndrome that variably includes all the metabolic diseases covered in this chapter (*v.i.*).

This chapter also avoids entry into the debate, now fueled by thousands of publications, over effectiveness of treatment of sleep apnea to improve in metabolic consequences of sleep apnea, other than to simply note that improvements in associated metabolic disease reflect

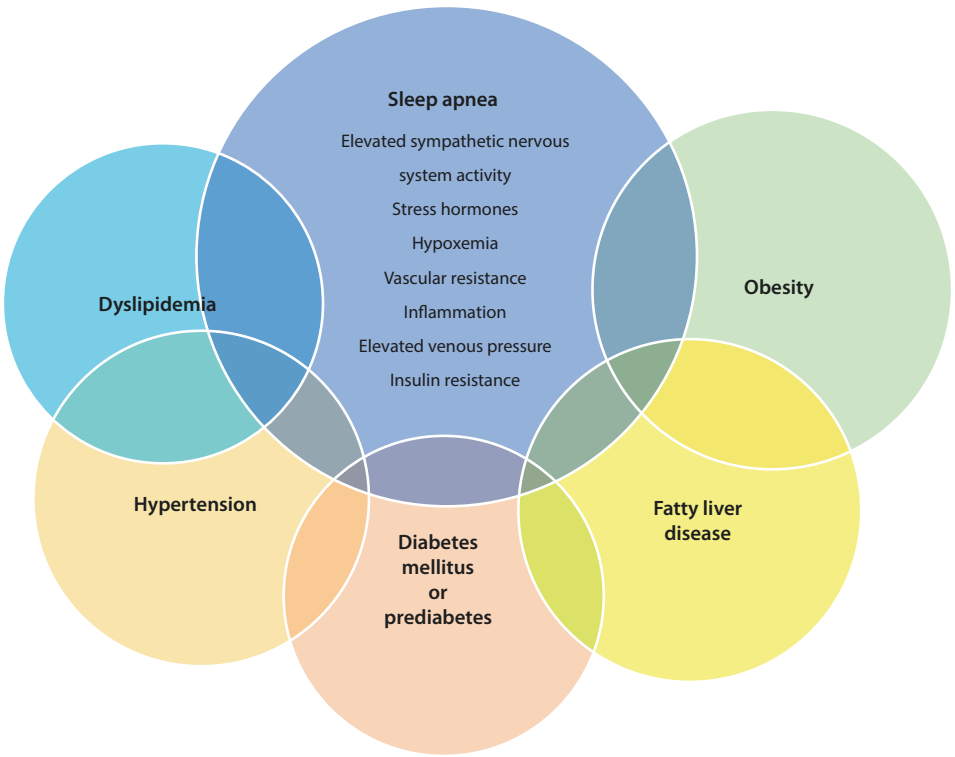

Fig. 5.1 Relationships among obstructive sleep apnea, obesity, hypertension, diabetes mellitus, steatohepatosis, and dyslipidemia

sufficient effectiveness of treatment over sufficient time. Treatment of sleep apnea is covered in parts III and IV of this book.

This chapter underscores recommendations for a multidisciplinary team in diagnosis and treatment of patients with OSA. Potential consequences of associated metabolic diseases will affect development and implementation of the treatment plan and the team should include individuals familiar with diagnosis and treatment of those diseases.

5.2 Obesity

Obesity, a disease of epidemic proportions in the United States and other industrialized countries, carries a close relationship to sleep apnea and other metabolic disease. Obesity can cause or exacerbate sleep apnea. In a reinforcing cycle, disruption of sleep due to sleep apnea (or any cause) can lead to weight gain and obesity [1–3]. Not surprisingly, treatment of obesity can lead to improvement in sleep apnea [4–6] and conversely, treatment of sleep apnea associates with early improvement in intra-abdominal fat mass, if not the total amount of adipose tissue [7, 8]. Although continuous positive airway pressure (CPAP) therapy can initially be associated with a disconcerting increase in mass, perhaps due to vascular relaxation and blood volume expansion, therapy seems important to subsequent success at weight loss [9]. As with other metabolic disease associated with sleep apnea, we should recognize not only the high prevalence of obesity in patients with sleep apnea, but also the high prevalence of sleep apnea in patients with obesity.

Obesity is now generally recognized as a disease [10], although some definitions of obesity include a somewhat circular definition that obesity must cause a second disease process, such as arthritis, diabetes mellitus, or sleep apnea. This requirement for comorbidity often makes the diagnosis of obesity somewhat redundant to other metabolic disease, but we believe diagnosis of obesity is important to care of patients with sleep apnea, as obesity and severity of obesity can be used as a marker of risk for hypoventilation and general anesthesia. Further, it is often used by third-party payers to justify payment for diagnostic and therapeutic procedures.

Patients with body mass index (BMI) of 30 or more are generally considered obese and patients with BMI of greater than 40 kg/m^2 are considered morbidly obese. Although this might be all that is needed for current requirements by third-party payers, a more physiological definition that includes markers of central adiposity and comorbidity is needed to refine diagnosis and therefore potential therapies.

The American Association of Clinical Endocrinology has dedicated significant resources to better define obesity in patients who warrant therapy. Recently published guidelines include both an anthropometric component and a clinical (associated disease) component to better identify patients who will benefit from therapy [11]. In this definition, the anthropometric component of the diagnosis of obesity is generally provided by BMI >30 kg/m^2 with evidence for obesity provided by a waist measurement of ≥102 cm (40 inches) for men in the United States or ≥88 cm (35 inches) for women, or BMI >35. I note that among untrained personnel, measurement of waist circumference can be problematic, as most patients with central obesity do not technically have a waist when defined as a narrowing between chest and hips. In this context, I recommend no measurement of waist circumference except in athletes in whom a diagnosis of obesity is inappropriate and a waist or narrowing is easily identified.

Tip

Measurement of waist circumference in patients without an identifiable waist or narrowing between chest and hips is problematic, and should be avoided. Absence of an identifiable waist should be sufficient evidence for adiposity.

See Table 5.1 for a working definition of obesity and staging of obesity based upon these guidelines.

Table 5.1 Diagnosis of obesity with adiposity based on AACE definitions [11]

Diagnostic categories	No obesity	Stage 0	Stage 1	Stage 2
Risk stratification		None	Mild to moderate	Severe
Anthropometric diagnosis	BMI < 25 kg/m^2	BMI = 25–29.9 kg/m^2 ≡ overweight BMI ≥ 30 kg/m^2 ≡ obesity Evidence for excess adiposity		
Clinical diagnosis (list of complications)		No complications	One or more mild–moderate complications	One severe complication or requires significant weight loss to treat

Table 5.2 Diagnosis of obesity

Anthropometric component	Clinical component
BMI ≥ 25 kg/m^2 or (BMI ≥ 23 kg/m^2 in certain ethnicities) AND excess adiposity	Obstructive sleep apnea
	Gastroesophageal reflux
	Asthma/restrictive airway disease
	Hypoventilation
	Hypertension
	Prediabetes
	Diabetes type 2
	Fatty liver disease
	Dyslipidemia
	Cardiovascular disease
	Polycystic ovarian syndrome
	Female infertility
	Male hypogonadism
	Osteoarthritis
	Depression/anxiety

The clinical component needed for staging of obesity consists of identification of an associated metabolic or mechanical problem as listed in Table 5.2.

Once the diagnosis of obesity is made, it should be graded by severity and included in the problem list to guide decisions as regards pulmonary function tests and tests for hypoventilation at rest or during sleep. I note that by this definition, all patients with BMI >35 kg/m^2 with severe sleep apnea have stage 2 obesity, and no further evaluation is required to add this diagnosis to the list of related problems that will need to be addressed in treatment of sleep apnea.

5.3 Hypertension

Hypertension is a consequence of multiple metabolic and subsequent hormonal and nervous system changes that occur with obstructive sleep apnea. Recognition of hypertension during evaluation of the patient with sleep apnea becomes important during therapy to reduce risks of cardiovascular events, which can be further increased by some medications for obesity, general anesthesia, and perioperative stress. Accurate diagnosis and staging of hypertension allow specific treatment plans to reduce these risks.

A strong relationship between hypertension and sleep apnea has long been recognized and formally reported since the early 1970s [12, 13]. As with other metabolic disease associated with sleep apnea, we need to recognize not only the high prevalence of hypertension in patients with sleep apnea [14–16], but also the high prevalence of sleep apnea in patients with hypertension [17, 18].

In the course of preparation of this chapter, the American Heart Association released an updated clinical practice guideline blood pressure [19]. Unlike the 2014 guidelines from the Joint National Commission, which dropped mention of sleep apnea or any other sleep disorder in relation to management of hypertension, these guidelines have reinstated recommendations for evaluation and treatment of sleep apnea, with the caveat that current data studies have demonstrated mixed results and that treatment of sleep apnea might not reduce cardiovascular disease risk.

As with all metabolic disease, early recognition and treatment can prevent progression and irreversible complications. Analogous to overweight and prediabetes, recommendations for hypertension have decreased the blood pressure at which treatment is recommended. The current categorization is seen in Table 5.3.

The diagnosis is based on accurate measurement of blood pressure in the office using first and fifth Korotkoff sounds to establish systolic and diastolic blood pressure, and/or use of an automated, validated, and calibrated home device.

Tip

It is our opinion that 24-hour blood pressure monitoring should be considered for all patients with obstructive sleep apnea, but especially for those with variably normal, elevated, or stage I hypertension or those with unexplained left ventricular hypertrophy.

For all patients with elevated blood pressure or hypertension, screening for secondary hypertension such as obstructive sleep apnea and nonpharmacological intervention is recommended. In addition to obstructive sleep apnea, consideration should also be given to measurement of potassium and screening for renal vascular disease, aldosteronism, coarctation of the aorta, or endocrine disease. If the diagnostic team does not include an endocrinologist or other specialist in secondary hypertension, then consideration should be given to referral.

Medications that cause hypertension are commonly used in patients with obstructive sleep apnea; these

Table 5.3 AHA definition of normal blood pressure, elevated blood pressure and Stages 1, 2, and 3 hypertension (*c.f.* [19])

Diastolic BP mm Hg	Systolic BP mm Hg					
		<120	120–129	130–139	140–159	>160
>100		Stage 2	Stage 2	Stage 2	Stage 2	Stage 2
90–99		Stage 2	Stage 2	Stage 2	Stage 2	Stage 2
80–89		Stage 1	Stage 1	Stage 1	Stage 2	Stage 2
<80		Normal	Elevated	Stage 1	Stage 2	Stage 2

Pharmacological treatment with cardiovascular disease (CVD) or estimated 10-year risk for CVD >10%

Pharmacological treatment with estimated 10-year risk for cardiovascular disease <10%

include not only self-medication with alcohol, caffeine, nonsteroidal anti-inflammatory medication, or nonprescription stimulants such as methamphetamine, but also prescribed medications that include amphetamines for treatment of attention deficit disorder, pseudoephedrine as a decongestant, and antipsychotics or antidepressants including high-dose serotonin noradrenaline reuptake inhibitor (SNRIs).

Evaluation of hypertension consists not only of good history and physical, but also basic blood testing to include glucose, metabolic panel, lipid profile, creatinine, electrolytes, thyroid-stimulating hormone (TSH), urinalysis, electrocardiogram, and perhaps echocardiogram.

Pharmacological treatment of hypertension is generally considered reasonable when the patient meets a combination of elevated mean blood pressure and a measure of cardiovascular risk; see Table 5.3.

We note that recent guidelines for diagnosis and treatment of hypertension include recommendation for care by multidisciplinary team members to include nephrologists, endocrinologists, cardiologists, pharmacists, etc. In the computer era, the sleep disorders center can easily institute electronic health record-driven testing and treatment protocols for hypertension and metabolic disease in collaboration with these specialists. This is important as treatment of one metabolic disease often leads to improvement in others. For example, treatment of hypertension improves blood flow and thereby insulin resistance, and it is generally recognized that any therapy for blood pressure (outside of thiazide diuretics and beta-adrenergic receptor antagonists, which decrease glucose-stimulated insulin release) will improve insulin action and decrease progression of prediabetes to diabetes.

5.4 Diabetes Mellitus

The relationship among insulin resistance, diabetes mellitus, and obstructive sleep apnea is well known. Prevalence of diabetes among patients with sleep apnea is proportional to severity of sleep apnea and has been most recently reported between 16% in a European group of 6442 patients with OSA [20] and 30% in a multiethnic group of 745 consecutive patients with OSA, at an urban medical center in the United States [21]. Prevalence of sleep apnea in patients with diabetes has been less well studied, and has been limited to home apnea testing, a less specific test at low apnea–hypopnea index (AHI). Results range from 36% in La Jolla (AHI > 15) [22] to 87% in the multicenter Sleep AHEAD trial (AHI > 5) [23]. Ethnic differences in the prevalence of sleep apnea among patients with diabetes seem to be explained more by obesity than diabetes [24].

Obstructive sleep apnea activates a cascade of metabolic changes that can contribute to diabetes mellitus including hypertension, sympathetic nervous system activation, and elevated cortisol that in turn cause resistance to insulin action and accelerate gluconeogenesis. In susceptible individuals, this causes metabolically mediated, type 2 diabetes mellitus (T2DM).

Coordination between dedicated centers for both sleep medicine and metabolic disease is necessary to successful care. A recent study [25] underscored not only the high prevalence of undiagnosed sleep apnea in patients with T2DM, but also the problems encountered in the absence of specialists in sleep medicine. Although 90% of patients with T2DM screened by questionnaire for sleep apnea had sleep with high risk of apnea, only 29% agreed to test after contact from a large, primary care clinic. Of this group of 213, 91% had OSA on the basis of predominantly home testing (AHI > 5), but only two-thirds agreed to trial therapy, that is, only one of every six patients thought to have sleep apnea agreed to a trial of treatment [25]. Discussion of results and education of patients to risks and benefits of their decisions should always be handled by a team that is trained and experienced in sleep medicine.

Although this section deals with metabolically mediated, T2DM, I should note a potential relationship among obesity, sleep apnea, and autoimmune-mediated type I diabetes mellitus (T1DM). The incidence of T1DM is increasing worldwide [26]. Whether this increase in incidence is related to a higher incidence of preclinical autoimmunity or faster progression to diabetes after development of autoimmunity is not clear. Rise in T1DM has stimulated speculation that insulin resistance and obesity might modulate autoimmunity. A recent meta-analysis supports the association between childhood obesity and subsequent T1DM [27].

The diagnosis of diabetes mellitus and related hyperglycemia is important to trigger evaluation for microvascular and macrovascular complications that can complicate therapy. From a surgical perspective, diagnosis of diabetes mellitus should not be missed, lest one has the diagnosis forced upon him in the postoperative period, incurring a higher risk of infection and slow wound healing, and necessitating a scramble for resources to immediately control glucose and train a naïve, post-operative patient for high intensity care at home.

Criteria used for the diagnosis of diabetes by the American Diabetes Association [28] are relatively straight forward (See Table 5.4), but practitioners often cut corners and diagnosis of diabetes is missed. For example, over-reliance on Hb_{A1C}, will miss 20-50% of patients with diabetes mellitus [29, 30].

The use of A1C for the diagnosis of diabetes has several advantages. Such testing does not require the patient to be fasting, is more convenient than a 2-hour oral glucose tolerance test, and is less dependent on the patient's health status at the time of testing. The following caveats should be acknowledged [30].

Table 5.4 ADA criteria for diagnosis of diabetes [28]

Tests to establish the diagnosis of diabetes Repeat test or use a different test to confirm		**Notes**
Oral glucose tolerance test	2-h PG ≥200 mg/dL (11 mM)	Oral glucose tolerance test with 75 g glucose in water in the morning after a fast with glucose measured at 0, 60, and 120 minutes
$Hemoglobin_{A1C}$	≥6.5% (48 mmol glc/mol hb).	The test should be performed in a laboratory using a method that is NGSP-certified and standardized to the DCCT assay
Fasting plasma glucose	FPG ≥126 mg/dL (7.0 mM).	Fasting is defined as no caloric intake for 8 hours, though it should be noted that sham feeding can result in insulin release and caffeine can cause gluconeogenesis
Hyperglycemic crisis	Random plasma glucose ≥200 mg/dL (11 mM).	Must be associated with classic symptoms of hyperglycemia or hyperglycemic crisis

Tip

- HbA1C should not be considered the primary criterion for diagnosis of diabetes. Confirmation with another test is recommended.
- HbA1C may be misleading in several ethnic populations (for example, African–American patients).
- HbA1C may be misleading in the setting of various hemoglobinopathies, iron deficiency, hemolytic anemias, thalassemias, spherocytosis, and severe hepatic and renal disease.

As with other metabolic disease, once the diagnosis of diabetes or prediabetes is established, patients should be referred to a multidisciplinary specialty clinic to educate the patient- and design-specific therapy. Centers accredited by the American Diabetes Association or the American Association of Diabetes Educators have the added advan-

tage of payment from third-party payers such as Medicare for these services. These centers may include nurse educators, registered dietitians, exercise physiologists, physical therapists, pharmacists, psychologists, social workers, and physicians. Administrators should appreciate that from an institutional perspective, certified centers for diabetes education can be used also for comprehensive and efficient treatment of obesity, hypertension, steatohepatitis, dyslipidemia, and other metabolic disease.

5.5 Fatty Liver Disease

Fatty liver disease occurs when pathological deposition of fat in the liver leads to metabolic dysfunction, inflammation, fibrosis, and cirrhosis. It has emerged as the major cause of cryptogenic cirrhosis and is currently the second most common indication for addition to the transplant wait-list in the United States [31]. It is the leading cause of chronic liver disease and occurs in at least two-thirds of patients with obesity [32]. Like other metabolic disease, prevalence and severity worsen with sleep apnea [33–35]. We note that severity of sleep apnea associates with severity of non-alcoholic fatty liver disease (NAFLD) even in patients without obesity [36] or metabolic syndrome [36].

Fatty liver disease, also referred to as non-alcoholic fatty liver disease (NAFLD), constitutes a spectrum of disease from benign, reversible hepatosteatosis to chronic non-alcoholic steatohepatitis (NASH) and cirrhosis. Fat deposition contributes to inflammation of the liver (steatohepatitis) and subsequent fibrosis or cirrhosis. The process is accelerated by liver insulin resistance [37].

Although much of the literature focuses on the relationship of OSA to non-alcoholic fatty liver disease, it is our impression that much of the fatty liver seen in our sleep disorders center has a contribution from alcohol consumption, past or present. We must remember, alcohol consumption can exacerbate NAFLD at even low levels of consumption [38]. Many patients with obstructive sleep apnea have concurrent insomnia, which they self-treat with alcohol. Historical screening for alcohol consumption is an important step in diagnostic and therapeutic considerations for fatty liver.

Alcohol consumption is an important diagnostic consideration, especially when one considers its effect on perioperative metabolism of anesthetics as well as risk of bleeding associated with decreased synthesis of coagulation factors, thrombocytopenia, and effects of alcoholic disease on platelet function. In our clinic, all patients with sleep apnea are instructed on the relationships among alcohol, sleep apnea, and metabolic disease, and are reminded not to drink alcohol until these problems are fully addressed.

Tip

Fatty liver disease in sleep apnea often associates with a history of alcohol consumption. Patients should be instructed to avoid alcohol until metabolic problems and sleep apnea are fully addressed.

Development of diagnostic tools for both early development of NAFLD and NASH with fibrosis and cirrhosis are rapidly evolving. MR proton density fat fraction (PDFF) has become the gold standard for liver fat quantification and liver biopsy remains the gold standard for fibrosis. We have, however, fairly good tests to screen for likelihood of fatty liver or fibrosis based on commonly measured anthropometric and serological variables including aspartate amino transferase (AST), gamma-glutamyl transferase (GGT), alanine amino transferase (ALT), and platelet content (PLT). (See ◘ Table 5.5).

As PDFF and liver biopsy cannot be performed universally, we have selected some screening tests that can be easily programmed into an electronic health record (EHR) using simple field codes, to calculate risk for NAFLD or NASH, and thereby add these diagnoses to the list of problems to address in therapy. Although the emphasis has drifted away from quantifying liver fat because the amount of the fat in the liver is not related to liver-related outcomes, in the context of sleep apnea and related metabolic disease, we continue to recommend screening for risk of hepatosteatosis or NAFLD, as this is a potentially reversible problem and treatment can lead to prevention of irreversible fibrosis and cirrhosis.

For assessment of risk for fatty liver, we recommend either Fatty liver index (FLI) or the NAFLD liver fat score (NAFLD-LFS). FLI is given by the following calculation:

$$\mathrm{FLI} = \frac{\left(e^{0.953*\log e(\text{triglycerides})+0.139*\mathrm{BMI}+0.718*\log e(\mathrm{GGT})+0.053*\text{waist circumference}-15.745}\right)}{\left(1+e^{0.953*\log e(\text{triglycerides})+0.139*\mathrm{BMI}+0.718*\log e(\text{ggt})+0.053*\text{waist circumference}-15.745}\right)}*100$$

Table 5.5 Diagnosis of hepatosteatosis (fatty liver) and steatohepatitis with fibrosis (cirrhosis) – see text for details

	Citation	Cut-off	Sensitivity (%)	Specificity (%)
Hepatosteatosis				
Screening test				
Fatty liver index (FLI)	[41]	≥30	87	64
NAFLD liver fat score (for liver fat>5.6%)	[39]	≥-0.64	86	71
Definitive test				
Magnetic resonance proton density fat fraction (PDFF)	*C.f.*, [42]			
Steatohepatitis with fibrosis				
Screening test				
Fibrosis-4 (FIB-4)	[43, 44]	<1.3	85	65
Vibration controlled transient elastography in NAFLD (kPa)	*C.f.*, [45]	≥10.3	90	87
Definitive test				
Liver biopsy				

NAFLD Liver Fat Score (for liver fat defined as >5.6%) is given by the following calculation [39]:

$$\begin{aligned}\text{NAFLD}-\text{LFS} = &-2.89+1.18\left(\text{metabolic syndrome}\left[\text{yes}=1/\text{no}=0\right]\right)\\&+0.45\left(\text{type 2 diabetes}\left[\text{yes}=2/\text{no}=0\right]\right)\\&+0.15\left(\text{fasting serum insulin}\left[\text{mU/L}\right]\right)\\&+0.04\left(\text{AST}\left[\text{U/L}\right]\right)-0.94\left(\text{AST/ALT}\right)\end{aligned}$$

The FLI has the disadvantage of requirement for measurement of waist circumference, a potentially troublesome measurement in patients with no waist. Although the NAFLD liver fat score avoids this measurement, it relies on assessment of metabolic syndrome and accurate diagnosis of T2DM. In this calculation, the metabolic syndrome was defined according to criteria of the International Diabetes Federation [40]: central obesity (waist circumference ≥94 cm in men and ≥80 cm in women) and at least two of the following factors: (1) serum triglycerides ≥1.70 mmol/L or specific treatment for this lipid abnormality; (2) serum high-density lipoprotein (HDL) cholesterol <1.03 mmol/L in men and <1.29 mmol/L in women or specific treatment for this lipid abnormality; (3) systolic blood pressure (BP) ≥130 mm Hg or diastolic BP ≥85 mm Hg or treatment for previously diagnosed hypertension; and (4) fasting plasma glucose ≥5.6 mmol/L or previously diagnosed type 2 diabetes. Although the requirement to screen for components of metabolic syndrome seems to add some complexity to the calculation, data for screening should be available from standard order sets for automatic analysis within the EHR.

For validated assessment of risk for liver fibrosis and cirrhosis in the context of NAFLD, we recommend additional calculation of Fibrosis-4 (FIB-4) with subsequent study by vibration-controlled transient elastography or biopsy.

Fibrosis-4 (FIB-4) is calculated by the following equation (McPherson, Stewart, Henderson, Burt, & Day, 2010; Sterling et al., 2006):

$$\text{FIB}-4=\frac{\left(\text{Age}\left[\text{year}\right]\right)\left(\text{AST}\left[\text{U/L}\right]\right)}{\left(\left(\text{PLT}\left[10(9)/\text{L}\right]\right)\left(\text{ALT}\right)^{1/2}\right)}$$

A patient with fibrosis in the context of hepatosteatosis can be expected to have an element of irreversible liver dysfunction. Treatment to arrest progression and to reduce fat content of the liver should be aggressive. Anesthesiologists will need to be aware of the likelihood of cirrhosis and adjust anesthesia and perioperative protocols as needed. When in doubt, prudence dictates consultation with a hepatologist and liver biopsy to fully define liver disease and risks.

5.6 Conclusions

- Recognition of metabolic disease associated with obstructive sleep apnea allows modification of therapy for sleep apnea to avoid risk of injury to the patient and to improve chances for a successful outcome. Specific strategies for management are covered in parts III and IV of this book.

- Documentation of associated disease and complexity of care has fast become important to sufficient reimbursement for services.
- Diagnosis of obesity and associated morbidities will allow not only detection of additional metabolic disease such as diabetes and fatty liver, but also requires recognition of mechanical problems such as arthritis and anatomical compromise of upper airway by adipose tissue, which in turn justifies therapy specific to obesity.
- Diagnosis and treatment of hypertension are important in the design of therapeutic programs for diet and exercise, as well as reduction in perioperative risks associated with surgical therapy.
- Recognition of prediabetes and diabetes allows reimbursement for treatment through multidisciplinary clinics that specifically address metabolic disease, and allows more accurate calculation of cardiovascular risks associated with exercise or surgical intervention.
- Recognition of simple hepatosteatosis allows therapy aimed at reversal and prevention of steatohepatitis, irreversible fibrosis, and hepatic dysfunction.
- Recognition of steatohepatitis with fibrosis and cirrhosis allows recognition of risk for cardiovascular disease, metabolic dysfunction, coagulopathy, and thrombocytopenia, and thereby better management of perioperative risks.
- Diagnosis and treatment of metabolic disease can be easily addressed through efficient use of electronic health records, problem-generated order sets, and a multidisciplinary team.

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Diagnostic Considerations for OSA

Contents

Clinical Evaluation of the Obstructive Sleep Apnea Patient

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Contents

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6.1 Background

Individuals with obstructive sleep apnea typically present to clinical attention with a chief complaint of excessive daytime sleepiness, snoring, or witnessed apneas during sleep. Excessive daytime sleepiness (EDS) is defined as the inability to stay awake and alert during the major waking episodes of the day, resulting in unintended lapses into drowsiness or sleep [1]. This symptom is common among the population, with estimates that more than 30% of the population in the United States has daytime sleepiness that interferes with their quality of life [2]. The daytime sleepiness may affect important activities such as work or taking care of children, or may occur at times that are dangerous such as driving a vehicle. The best initial step in evaluation of patients with suspected obstructive sleep apnea includes a detailed history and physical examination [3].

6.2 History Taking

Because many patients presenting for evaluation of obstructive sleep apnea report excessive daytime sleepiness, it is important to further investigate the presence and severity of excessive daytime sleepiness. Subjective scales, such as the Epworth Sleepiness Scale (See Fig. 6.1), can quickly attempt to measure the level of sleepiness during the day as reported by the patient. This can be further assessed (many times more effectively) by directly inquiring about different circumstances where sleepiness occurs or affects them. Providers can ask patients if they feel sleepiness affects activities such as driving, work, school, or social activities. Many times, the patient may underestimate the severity or even presence of excessive daytime sleepiness. This can be due to the chronic nature of the symptoms, as it may be difficult for the patient to know what normal alertness during the day is supposed to be if symptoms have persisted for years. Sleepiness also affects the ability of the brain to self-assess performance, hence leading to inaccurate judgments by the patient on their ability to stay alert. It can be helpful to ask any family members, friends, or coworkers about the patient's level of sleepiness, as they may have a different and more accurate perspective. Inquiring about when and how often daytime sleepiness occurs can also be helpful in the evaluation, especially because there are numerous causes for a presenting complaint of excessive daytime sleepiness that also need to be considered in the differential diagnosis (See Table 6.1). Insufficient sleep (getting less than 7 hours per a day on average) is the most common cause of excessive daytime sleepiness in the United States.

It can also be helpful to ask if the patient's sleepiness is more consistent with fatigue or decreased energy, as there are other possible causes of these symptoms that can be difficult for patients or clinicians to distinguish. Mood disorders, endocrinopathies, and certain rheumatological disorders commonly cause fatigue. Specifically asking the patient if they actually doze off or fall asleep in sedentary situations versus not wanting to get up and do something active can sometimes help determine if the patient has hypersomnia from a primary sleep disorder or fatigue from another medical cause. Of note, even with a good history, many patients with sleep disorders will still describe their sleep disorder with presenting symptoms of fatigue or lack of energy, so these symptoms should still be taken seriously in the evaluation of sleep apnea [4]. Obstructive sleep apnea can also cause symptoms of decreased attention, cognition, and poor memory. Hyperactivity and inattention are a common presenting complaint of obstructive sleep apnea, especially in children [5].

In addition to asking about sleepiness, it is essential to investigate other common clinical symptoms of obstructive sleep apnea. The presence, frequency, and intensity of snoring are key features in patients with obstructive sleep apnea. Snoring may not be apparent to the patient, and it is important to ask a bed partner or collateral source about the presence of snoring or noisy breathing. Patients may report that their bed partner or someone sharing a room with them notices apneas or pauses in their breathing during sleep. Other symptoms that can be attributed to obstructive sleep apnea include nighttime awakenings, night sweats, nocturia, acid reflux, and morning headaches. These symptoms are noted more commonly in patients with sleep apnea, but are nonspecific and can be related to numerous other etiologies. Finally, many patients with obstructive sleep apnea may present with insomnia or disrupted sleep.

It is important to consider evaluation for other causes of hypersomnia, especially because patients with obstructive sleep apnea may also have other contributing sleep disorders also responsible for poor sleep quality (See Table 6.1). Evaluation for insufficient sleep by asking about bedtime, wake times, and sleep schedules is essential. Adults require at least seven hours of sleep for optimal health, and insufficient sleep is the most common causes of sleepiness [6]. Restless legs syndrome can be a cause of excessive daytime sleepiness by causing nighttime sleep disruption. This diagnosis can be evaluated by asking the patient about extremity discomfort or an urge to move their legs that is worse at night. The symptoms improve with movement and cause disruption of their sleep. Though rare, screening for narcoleptic symptoms such as cataplexy, sleep-related hallucinations, and sleep paralysis can be helpful in the appropriate clinical scenario. Cataplexy is brief episodes of transient muscle weakness triggered by emotion, typically positive emotion such as laughter. Sleep paralysis

Fig. 6.1 Epworth sleepiness scale (From: Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14(6):5400–545)

Epworth Sleepiness scale

Using the following scale, circle the most appropriate number for each situation.

0=would doze, less than once a month

1=slight chance of dozing

2=moderate chance of dozing

3=high chance of dozing

Situation	Chance of Dozing			
Sitting and reading	0	1	2	3
Watching TV	0	1	2	3
Sitting inactive in a public place (theatre, in a meeting)	0	1	2	3
As a passenger in a car for an hour without a break	0	1	2	3
Lying down to rest in an afternoon	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after a lunch without alcohol	0	1	2	3
In a car, while stopped for a few minutes in traffic	0	1	2	3

8 numbers you have circled TOTAL = _____

From: Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14(6):5400-545.

Table 6.1 Important causes of excessive daytime sleepiness to consider

Insufficient sleep
Obstructive sleep apnea
Central sleep apnea
Narcolepsy
Idiopathic hypersomnia
Circadian rhythm disorders
Restless legs syndrome
Medications
Drugs of abuse
Traumatic brain injury
Neurodegenerative disorders (e.g., dementia, Parkinson's disease)

is a transient feeling of being awake but not being able to move, usually occurring while falling or waking up from sleep. Cataplexy is rarely seen outside a diagnosis of narcolepsy, but both sleep paralysis and sleep-related hallucinations can be seen in a variety of sleep disorders, including sleep apnea and sleep deprivation.

Certain medical diagnoses place patients at higher risk for obstructive sleep apnea. These include pulmonary conditions such as chronic obstructive pulmonary disease and asthma as well as neurological conditions such as stroke or neuromuscular disorders. Patients with craniofacial disorders or midface hypoplasia, such as patients with cleft palate or Down's syndrome are at higher risk of sleep-disordered breathing. Patients with cardiac diseases such as heart failure and cardiac arrhythmias (atrial fibrillation) have very high rates of both central and obstructive sleep apnea [7]. It will also be important to ask about any previous upper airway

surgeries that the patient may have undergone for a previous diagnosis of sleep apnea or airway difficulties.

Patients may try to mask their symptoms of sleepiness by using caffeine or tobacco. While completing the social history, the provider should ask about any use of caffeine, tobacco, or other recreational drugs which can cause sleepiness during the day or insomnia at night. A history of smoking also puts the patient at risk for sleep apnea.

It is important to learn more about the patient's employment, as certain occupations come with specific regulations in regards to sleepiness and a diagnosis of obstructive sleep apnea. This typically includes occupations such as pilots or drivers with commercial driver's licenses. Federal agencies have regulations in regards to adherence to therapy or possibly time off of work during evaluation for sleep apnea if there is perceived risk that the symptoms may put the worker or society at risk. It may also be helpful to ask if any family members have a history of obstructive sleep apnea (or symptoms suspicious for sleep apnea), as obstructive sleep apnea can run in families, especially if related to craniofacial or upper airway anatomical abnormalities.

6.3 Physical Examination

The clinical history as described above is critical in the evaluation of possible obstructive sleep apnea. There are key features on the physical examination that are also very helpful in the clinical evaluation. One of the most helpful objective measure is an elevated body mass index (BMI) or a finding of obesity. The higher the BMI, the higher the risk for obstructive sleep apnea (due to increase of fatty tissue in oropharyngeal structures including tongue). It is also important to measure the neck circumference as an increased neck circumference >17 inches in men and >16 inches in women are also considered risk factors and should be included in the initial clinical evaluation [8]. An elevated blood pressure (or a history of hypertension), abnormal respiratory signs, or low oxygen saturations can be associated with sleep-disordered breathing.

A detailed upper airway and craniofacial examination will be helpful in looking for characteristics that can predispose the patient to a narrow airway. The clinician should document the presence and size of tonsillar tissue as well as a gauge of how crowded the airway is upon visual inspection. One common method is the Mallampati classification which grades the airway from 1(least crowded) to 4(most crowded). The Mallampati classification or score was initially utilized by clinicians in determining ease of intubation but was later found to correlate with risk of obstructive sleep apnea. The score is obtained by asking the patient while sitting to open their mouth and fully protrude their tongue (no phonation) and examining the airway. Mallampati 1 classification is when you can see the soft palate, hard palate, uvula, and tonsillar pillars. A Mallampati 2 classification is when you can view the other three structures, but not the tonsillar pillars. In a Mallampati class 3, only the soft and hard palate, and base of the uvula is visualized. In a Mallampati class 4, only the hard palate is noted, suggesting a crowded airway putting the patient at highest risk for sleep apnea [9].

More detailed visualization of the upper airway can be performed by nasal endoscopy, though this is not routinely performed at most sleep centers unless surgical intervention is being considered. Craniofacial abnormalities such as retrognathia, micrognathia, macroglossia, scalloping of the tongue, or significant overjet also place the patient at high risk for obstructive sleep apnea. Physical examination of nasopharynx should include evaluation for nasal obstruction from nasal turbinate hypertrophy, septal deviation, nasal polyps, or other obstructing lesions.

Due to the common occurrence of cardiac disease and sleep apnea, and detailed cardiovascular examination listening for murmurs, abnormal heartbeats, or signs of heart failure (lower extremity edema or elevated jugular venous distention) should be performed. An abnormal pulmonary exam may also suggest heart failure (rales or crackles) or other pulmonary disorders (wheezing) putting the patient at risk for obstructive sleep apnea or other sleep-disordered breathing such as sleep-related hypoventilation or hypoxemia. Evaluating the distal extremities for any clubbing or cyanosis is also helpful to determine if there is any underlying cardiovascular or pulmonary disease.

A detailed neurological examination, especially focusing on cranial nerve and motor function, can assist in the evaluation of obstructive sleep apnea. If patients have significant motor weakness or signs of a central nervous system injury, this puts them at risk for not only obstructive sleep apnea, but also more complicated sleep-disordered breathing such as sleep-related hypoventilation or central sleep apnea which may require more complex evaluation and treatment.

6.4 Conclusion

Though objective testing is necessary to confirm a diagnosis of obstructive sleep apnea, the history and physical examination is key to a comprehensive evaluation of the patient with suspected sleep apnea. The history and physical not only helps guide which testing is necessary as the next step, but also evaluates for other possible causes for the patients presenting sleep symptoms.

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Further Reading

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Diagnostic Testing for Obstructive Sleep Apnea

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Contents

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7

7.1 Background

Obstructive sleep apnea (OSA) is characterized by complete or partial closure of the upper airway during sleep and is the most common form of sleep-related breathing disorders. OSA is highly prevalent in the community; recent epidemiologic data suggest that 14% of men and 5% of women in the general population have OSA that is associated with excessive daytime sleepiness. Certain high-risk populations such as those with resistant hypertension, pulmonary hypertension, coronary artery disease, congestive heart failure, cardiac arrhythmias, stroke, and diabetes mellitus type 2 have significantly higher rates of OSA.

OSA is associated with several adverse individual and population health consequences. Hypoxemia, hypercapnia, sympathetic dysregulation, intrathoracic pressure swings, and increased arousals from sleep are thought to be the pathophysiologic mechanisms underlying these increased risks. OSA has been shown to be linked with adverse neurocognitive, cardiovascular, and metabolic sequelae. These include excessive daytime somnolence, enhanced risk of motor vehicle and work place accidents, mood disorders, and dementia. There is a heightened risk of systemic and pulmonary hypertension, coronary artery disease, congestive heart failure, arrhythmias, and stroke. OSA is associated with metabolic dysregulation and increased risk for diabetes. Additionally, OSA is linked with increased healthcare utilization.

OSA is generally well treated with continuous positive airway pressure (CPAP) and has been shown to decrease symptoms, rate of motor vehicle accidents, the adverse medical consequences noted above, and healthcare utilization as well as improve quality of life. However, a CPAP device may be cumbersome and difficult to tolerate for many patients. Oral appliance treatment is used as an alternative therapeutic option. Generally, this is less efficacious than CPAP in terms of eliminating disordered breathing events, but may be more acceptable to some patients. Oral appliance treatment is beneficial in reducing daytime sleepiness and blood pressure to a degree equivalent to CPAP.

Thus, OSA is a common medical disorder that is associated with significant injurious health consequences and increased healthcare costs. A false-negative test may leave symptomatic patients untreated, adversely affect their quality of life, and increase the risks of poor health outcomes. On the other hand, treatment of OSA may be difficult for many patients to tolerate. A false-positive test may expose patients to unnecessary inconvenience and expense. Therefore, it is imperative that the diagnosis of OSA be established as accurately as possible. Access to testing, ease and cost of the diagnostic procedures are other relevant factors that are important to consider from the patient perspective.

In this chapter, we describe the testing modalities that are currently available for the diagnosis of OSA in adults. Diagnosis of OSA in children and other sleep-related breathing disorders such as central sleep apnea syndromes and sleep-related hypoxemia/hypoventilation is beyond the scope of this chapter.

7.2 Diagnosis of OSA

7.2.1 History and Examination

The evaluation of OSA starts with a comprehensive sleep evaluation, comprising of a detailed history for symptoms suggestive of OSA, and to assess for the possibility of other sleep disorders, and presence of comorbid conditions. Physical examination should include an assessment of the body mass index (BMI), neck circumference, blood pressure, focused ear, nose, and throat examination (e.g., the presence of nasal septal deviation, nasal turbinate hypertrophy, nasal mucosal erythema/discharge, nasal polyps, nasal valve collapse, micro- and/or retrognathia, maxillary hypoplasia, high-arched palate, overbite, cross-bite, overjet, Friedman palatal position, decreased anteroposterior and lateral dimensions of the oropharynx, macroglossia, ankyloglossia, dental marks on the tongue, and tonsillar hypertrophy) as well as an examination of the cardiovascular and respiratory systems. See ► Chap. 6 for further information on the clinical evaluation of OSA.

7.2.2 Screening Tools

Screening questionnaires and prediction algorithms are not sufficient to make the diagnosis of OSA as they have been found to have a low accuracy when compared to traditional diagnostic tests in several studies. Many of these studies were performed in high-risk populations for OSA such as the elderly, bariatric surgery candidates, and commercial vehicle drivers and thus may not be generalizable to other patient groups. In general, the specificity of these screening tools was noted to be low, resulting in a large number of false positives. Furthermore, the rate of predicted false negatives was more than 1 in 10, which would render these measures as unacceptable for the purposes of diagnosing OSA. The most recent American Academy of Sleep Medicine (AASM) clinical practice guideline for the diagnostic testing for adult OSA recommended that these tools may be used in clinical settings, but not as a substitute for objective sleep testing. Examples of questionnaires and algorithms used in screening for OSA include the Epworth Sleepiness Scale, Berlin Questionnaire, Stop-BANG, sleep apnea clinical score,

Kushida Index, OSAS score, OSA50, Multivariable Apnea Prediction Questionnaire, and morphometric models.

7.2.3 Diagnostic Tests

The diagnosis of OSA involves measurement of respiratory parameters in sleep. Attended or in-laboratory polysomnography (PSG) is considered the gold-standard test for making the diagnosis of OSA and requires the simultaneous measurement of eight or more physiological parameters during sleep, including electroencephalography (EEG), electrooculography (EOG), electromyography (EMG), electrocardiography, air flow (with an oronasal thermistor and nasal pressure transducer), chest/abdominal muscle effort (usually with respiratory inductance plethysmography), pulse oximetry, heart rate, snoring (with a microphone), and body position. Time-synchronized audio and video recording is generally available as well. Due to issues with access to in-laboratory PSG requiring the presence of skilled personnel and associated cost, home sleep apnea tests (HSATs) have emerged as a viable option for the diagnosis of OSA in well-selected patients.

7.2.4 Types of Sleep Studies

Sleep studies are traditionally classified as types I–IV (Table 7.1). Type I is attended in-laboratory PSG. Unattended sleep studies are categorized into types II–IV. Type II studies are similar to type I except that they are unattended studies and can be performed outside of the sleep laboratory. Type III studies utilize oximetry, two respiratory, and one cardiac channel. Type IV studies use only one or two sensors, for example, airflow or oximetry and pulse rate. There is considerable device variation even within the same sleep study category. In addition, since this original classification was devised, newer technologies such as those incorporating peripheral arterial tonometry to diagnose OSA have emerged. The SCOPER (sleep, cardiovascular, oximetry, position, effort and respiratory parameters) classification, proposed by the AASM more recently after a comprehensive technology evaluation, is an alternative and more complex classification system that includes these newer technologies.

7.2.5 Definition of OSA

The International Classification of Sleep Disorders, Third Edition, defines OSA as being present if a respiratory index (RDI) of ≥5 per hour is noted on PSG or HSAT, associated with typical symptoms of OSA, that is, loud snoring, choking/gasping/breath-holding episodes, witnessed apneas, unrefreshing sleep, sleepiness, fatigue, insomnia, and/or a diagnosis of mood disorder, cognitive dysfunction, hypertension, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, stroke, or type 2 diabetes mellitus. Alternatively, an RDI of ≥15 per hour on PSG or HSAT in the absence of these symptoms or comorbid medical conditions is sufficient to make the diagnosis of OSA.

7.2.6 Scoring of Respiratory Events

The RDI comprises apneas, hypopneas, and respiratory effort-related arousals (RERAs) per hour of sleep, whereas the apnea-hypopnea index (AHI) includes only apneas plus hypopneas per hour of sleep. The criteria for defining hypopneas have changed over the years, rendering an evaluation of the medical literature regarding the diagnosis and outcomes of OSA difficult. The AHI (and therefore the RDI) may be considerably different in an individual person depending upon which definition of hypopnea is employed. The most recent AASM Manual for the Scoring of Sleep and Associated Events version 2.4, 2017, recommends scoring hypopneas in adults when there is a ≥ 30% reduction in nasal pressure

Table 7.1 Types of sleep studies

	Tech	EEG	EOG	EMG	ECG	Airflow	Resp effort	SpO_2
Type 1	X	X	X	X	X	X	X	X
Type 2		X	X	X	X	X	X	X
Type 3					X	X	X	X
Type 4								X

Abbreviations: *tech* sleep technologist, *EEG* electroencephalography, *EOG* electrooculography, *EMG* electromyography, *ECG* electrocardiography, *Resp* respiratory, *SpO_2* pulse oximetry

signal excursion (alternative hypopnea sensor if this is unavailable) or positive airway pressure (PAP) device flow for at least 10 seconds accompanied by a ≥ 3% oxyhemoglobin desaturation or arousal. However, many laboratories are also using ≥4% desaturation criterion (with no arousal criteria), along with a ≥ 30% or ≥ 50% reduction in nasal pressure excursion, in keeping with the "acceptable" definition of hypopneas in the current scoring manual and the definition in the older version of the manual from 2007 respectively. This is largely due to the lack of reimbursement from certain medical insurance companies (mainly Centers for Medicare and Medicaid Services) for OSA diagnosed based on the latest recommended scoring criteria and paucity of data demonstrating a difference in long-term clinical outcomes with the different criteria in use.

7.2.7 Clinical Guidelines

The AASM published practice parameters for the indications for PSG in 2005 and initial clinical guidelines for the use of HSATs in 2007. In some geographic areas, there has been a significant upswing in the number of HSATs performed relative to PSGs for the diagnosis of OSA; there are a number of potential reasons for this including changes in payor policies. As noted above, AASM published an updated clinical practice guideline for the diagnostic testing for adult OSA in 2017, incorporating a meta-analysis of 87 randomized controlled trials (RCTs) and observational studies from 2005 to 2016 evaluating the accuracy of diagnosis of OSA using clinical prediction rules, HSAT, and PSG as well as relevant clinical outcome measures. Four recommendations in this guideline were graded "strong" based on the quality of the evidence, benefits versus harms, patient values and preferences, and utilization of resources. The remaining two recommendations were rated "weak." These recommendations will be discussed throughout the chapter.

7.3 Home Sleep Apnea Test (HSAT)

This type of sleep study is one that can be conducted outside of the sleep laboratory setting in the absence of a trained sleep technologist during the recording period.

7.3.1 Advantages

Potential benefits of performing a sleep study in the home setting include greater convenience and comfort to the patient, increased access to testing and possibly decreased cost. HSAT may be particularly advantageous in some situations where it might be difficult for the patient to leave the home or healthcare setting due to complexity of cares. Aside from being able to sleep in a familiar environment, generally, fewer sensors are applied during a HSAT, which may also serve to enhance patient comfort. In one RCT in which both HSAT and PSG were performed in the same patient, over three-fourths of the subjects preferred HSAT.

7.3.2 Disadvantages

HSAT is less accurate when compared to PSG in the diagnosis of OSA and tends to underestimate severity. Usually, EEG is not recorded; therefore, the degree of sleep fragmentation secondary to OSA cannot be determined. Other sleep disorders cannot be assessed on a HSAT. Furthermore, central sleep-disordered breathing (SDB) events may not be well differentiated from obstructive events, and in most cases, RERAs cannot be detected. There is no opportunity to titrate positive airway pressure (PAP) and assess response to this or troubleshoot in the home setting. Chain of custody issues may arise, for example, in a commercial vehicle driver or pilot, in ensuring that the patient for whom the study is being ordered is the one undergoing the test. Lastly, if the HSAT is negative for OSA, a PSG is indicated for further evaluation in the patient with suspected moderate-to-severe OSA (assuming they were appropriately selected for HSAT). In this situation, one study suggested that there were a significantly high proportion of patients who did not follow through with the recommended second round of testing with PSG, thus leaving their OSA untreated with potential attendant long-term risks.

7.3.3 Patient Selection

The AASM recommends that HSAT be performed in an uncomplicated patient with an increased clinical pre-test probability of moderate-to-severe OSA.

An uncomplicated patient is one that does not have a condition that would place them at a greater risk of nonobstructive SDB, such as central sleep apnea, sleep-related hypoxemia, or hypoventilation (e.g., cardiopulmonary disease, potential respiratory muscle weakness due to a neuromuscular condition, history of stroke, or current chronic opioid medication use). Patients with significant safety-related issues such as driving or workplace accidents due to sleepiness may also best be studied by PSG. If a screening oximetry suggests the presence of significant hypoxemia and/or hypoventilation or the patient has other risk factors for hypoventilation for example, a BMI in excess of 40 kg/m^2, PSG would be preferable. In some geographic areas, third-party payors have their own criteria for coverage of HSAT versus

PSG. In patients in whom there is a suspicion for other sleep disorders that require evaluation (e.g., central disorders of hypersomnolence, parasomnias, sleep-related movement disorders) or can interfere with the conduct or accuracy of HSAT (e.g., insomnia), PSG is the recommended test. Other situations that may preclude the conduction of HSAT may involve personal (e.g., cognitive dysfunction, physical limitations) or environmental (unsuitable living conditions) factors that can limit the acquisition and interpretation of data.

There is very limited medical literature evaluating the validity of HSAT in patients with significant cardiopulmonary/neuromuscular conditions, insomnia, and those on opioid medications or at high risk of hypoventilation. HSAT may result in an inaccurate assessment of SDB in these situations, and thus PSG is the recommended test of choice. If there are other extenuating circumstances, such as an inability to leave the hospital or home setting, then it may be reasonable to proceed with HSAT than to perform no testing at all.

According to the recent AASM clinical practice guideline for the diagnostic testing for adult OSA, an increased pretest probability of moderate-to-severe OSA is indicated by the presence of excessive daytime sleepiness with at least two of the following three factors: (1) habitual loud snoring, (2) witnessed apnea, or (3) hypertension.

7.3.4 Data Obtained

The recording channels usually comprise a combination of respiratory (including oximetry) and pulse rate parameters. Newer technologies may employ measures of peripheral arterial tonometry to determine disordered breathing events and estimate sleep time/stages. All of these technologies generally include snoring and body position sensors as well.

A technically adequate device per AASM guidelines is one that has at least nasal pressure (for airflow), chest and abdominal respiratory inductance plethysmography (for effort), and oximetry. In the case of devices that utilize peripheral arterial tonometry, these measures in addition to actigraphy are required. Detailed requirements for the sensors are described in the most recent edition of the AASM Manual for the Scoring of Sleep and Associated Events.

Typically, sleep staging channels such as EEG, EOG, and EMG are not present in HSAT, thus the number of disordered breathing events is calculated per hour of recording time and not sleep time. To reflect this difference, the HSAT usually reports severity of SDB as a "respiratory event index" (REI), as opposed to an RDI or AHI. The REI represents a potential underestimation of events that might be calculated on PSG. Secondly, due to the absence of sleep staging, respiratory events resulting in cortical arousals per current recommended scoring criteria cannot be determined, which may also result in a lower severity of SDB than that gauged by PSG.

7.3.5 Conduct and Interpretation of Test

It is recommended by the AASM (and required by most third party payors) that the HSAT be administered by an accredited sleep center under the supervision of a board-certified or board-eligible Sleep Medicine physician. Similarly, the test should be interpreted by a board-certified or eligible Sleep Medicine physician.

A single HSAT is conducted over one night. Studies of single versus multiple nights of recording have demonstrated a marginal increase in accuracy and increased probability of insufficient information with multiple recordings compared to a single night of data. It should be noted that the recordings in these studies utilized only a single channel (nasal pressure transducer or oximetry) and efficiency of care as well as long-term clinical outcomes were not assessed.

Based on available studies, a technically adequate HSAT requires a minimum of 4 hours of recorded data obtained for a duration that includes the patient's habitual sleep period. This includes a minimum of 3 hours of oximetry data and 2 hours of airflow information. There is no literature regarding the accuracy of results obtained from less than 4 hours recording on a HSAT compared to PSG and the influence of the number of recording hours on any long-term clinical outcomes.

7.3.6 Accuracy of Results

There is moderate evidence of the potential for misclassification of severity of SDB in either direction, based on 27 studies that assessed the accuracy of HSAT versus PSG. This is partly due to night-to-night variability of OSA and possibly due to different hypopnea definitions used in the two test types.

The accuracy of type II and III studies compared to PSG (AHI cut-off of ≥5 per hour or ≥ 15 per hour) is in the range of 80–90%, for patients thought to be at high risk for OSA. The accuracy deteriorates in low-risk groups. The use of single-channel oximetry has significantly high false-positive and false-negative rates when compared to PSG (more than 1 in 5). Three studies assessed HSAT using peripheral arterial tonometry and actigraphy against PSG. These studies showed a misdiagnosis rate of about 1 in 10 in high- as well as low-risk patients, a low specificity of about 0.45 for an AHI of ≥5 per hour and ranging from 0.77–1.0 for AHI cutoffs of ≥15 or ≥ 30 per hour.

7.3.7 Discussion of Results

After a positive HSAT, the patient can be commenced on auto-titrating PAP (APAP) if thought to be appropriate by the treating provider. Alternatively, the management pathway may include titration PSG after a positive HSAT. If the results are complex, then an in-laboratory split-night PSG (described below) or titration PSG may be required, depending upon the individual patient.

Based on currently available literature, the chance of a technically inadequate study is approximately 20%. If the HSAT is negative, inconclusive, or technically inadequate in a patient with a high pretest probability of OSA, then attended PSG is recommended as the next step rather than a repeat HSAT. This is because evidence from one study suggested that the likelihood of a second inconclusive or technically inadequate is about 40%. Furthermore, in this study, the rate of nonadherence with the recommended next step of a PSG was high (approximately 20%); to maximize the rate of a definitive diagnosis in this situation after a failed first study, PSG is the recommended test. However, patient preference, available resources, and clinician judgment regarding the possibility of a second failed HSAT will need to be taken into account before making this decision in an individual patient.

7.3.8 Recommended Follow-Up

Early follow-up is recommended following the initiation of APAP after HSAT. Most RCTs examining the HSAT-APAP pathway included a follow-up APAP visit within 2–7 days after HSAT with skilled technical staff. It should be noted that these RCTS were conducted in tertiary care or academic settings comprising of highly skilled medical and technical personnel teams.

7.3.9 Clinical Outcomes

In seven RCTs, after CPAP was commenced, patient-reported outcomes (sleepiness, quality of life, and PAP adherence) did not differ between HSAT and PSG groups. Information regarding cardiovascular and other outcomes is currently not available.

7.3.10 Cost-Effectiveness

The overall cost-effectiveness of an HSAT versus PSG pathway of management of OSA is not fully clear. In the long term, the PSG pathway has been noted to be more beneficial in patients with moderate-to-severe OSA in some studies due to the favorable cost-effectiveness of treatment of OSA in this group of patients. False negatives with HSAT that leave patients untreated with downstream costs relating to adverse health consequences and healthcare utilization, the cost of retesting with PSG in the setting of negative, inadequate, inconclusive or complex HSAT results, and the potential for false positives with unnecessary treatment may tilt the balance in favor of PSG. Conversely, in the one RCT that evaluated the expense associated with HSAT versus PSG, there was a 25% lower cost with HSAT.

If HSAT is utilized in appropriately selected patients and within the care management pathway described above, it is likely to be more cost-effective than if the recommended guidelines are not followed. From the provider perspective, cost may not always be lower with the HSAT pathway because a large number of components are required to ensure that the quality of the HSAT pathway-mediated care for OSA is similar to a PSG pathway.

7.3.11 Summary of HSAT

The use of HSAT in an uncomplicated patient with a high pretest probability of moderate-to-severe OSA, using a technically adequate device and recording period, under the supervision of personnel with the requisite expertise and with a clear management pathway in place, can provide similar clinical outcomes as PSG when used in the diagnosis of OSA.

7.4 Polysomnography (PSG)

In-laboratory PSG, consisting of the simultaneous monitoring of multiple physiological parameters in sleep, in the presence of skilled technical personnel, is the current gold-standard recommended test for the detection of OSA.

7.4.1 Patient Selection

Currently available evidence includes only patients with comorbid heart failure and chronic obstructive pulmonary disease. The utility and validity of HSAT in patients with other comorbidities, environmental and personal factors that can affect testing have not been systematically studied. In the research that has been conducted to date, the specificity of HSAT in identifying central SDB or hypoventilation was low or not evaluated. Since these respiratory abnormalities are potentially associated with significantly increased risk of morbidity and mortality, and may not be adequately assessed by HSAT and/or require treatment modalities other than CPAP/APAP,

PSG is recommended for the diagnosis of SDB in patients with the comorbidities or complicating factors described above.

7.4.2 Number and Duration of Tests

A split-night protocol (where PAP is applied after the diagnostic portion of the study) is generally appropriate and may be used instead of a full night diagnostic study for the purposes of detecting OSA. For a split-night study, a moderate-to-severe degree of OSA needs to be observed during a minimum of 2 hours of recording time on the diagnostic portion of the PSG and at least 3 more hours should be available for PAP titration.

The accuracy of split-night PSG has been found to be comparable to full night diagnostic PSG, even in those with milder degrees of OSA. Many of these investigations were not RCTs and the types of sensors utilized were inconsistent across studies. Currently, there is no definitive data regarding the optimal AHI threshold at which to initiate PAP after the diagnostic portion of the PSG.

The split-night protocol, in theory, leads to decreased cost and increased efficiency of care by facilitating diagnosis and treatment of OSA during a single night's recording. One study did demonstrate lower cost with the split-night PSG protocol compared to the full night pathway, based on cost per quality of life year gained. However, further research regarding cost-effectiveness is needed. It is worth noting that the studies evaluating split-night versus full night diagnostic PSG excluded certain patient groups such as those with severe insomnia, claustrophobia, and other suspected sleep disorders. Thus, individual patient factors determining eligibility for the split-night pathway are not fully known at this time. Additionally, if the diagnostic and/or titration portions of the study are inadequate or inconclusive, these may need to be repeated; alternatively, in the case of an inadequate/inconclusive titration, APAP may need to be used if thought to be appropriate by the treating clinician.

Most studies have shown no significant differences in patient-related outcomes such as the rates of adherence to CPAP or the residual AHI on CPAP treatment in subjects who underwent split-night or full night diagnostic PSG.

7.4.3 Conduct and Interpretation of the Study

As with HSAT, it is recommended that PSG be administered by an AASM accredited facility with appropriately trained personnel and the study interpreted by a physician who is board-certified or board-eligible in Sleep Medicine.

7.4.4 Follow-Up

After undergoing PSG for the diagnosis of OSA, discussion of the results must take place within a reasonable time frame after the study and the patient commenced on treatment if appropriate.

7.4.5 Discussion of Results

If the results of PSG are negative for OSA and there is still a high clinical suspicion for OSA, it is recommended that a second PSG be considered for the diagnosis of OSA. There are a few studies of two-night versus one-night PSG that have shown significant night-to-night variability in AHI in a subset of patients, although there were no overall differences in AHI between the groups. Up to a third of individuals had a change in the classification of severity of their OSA in either direction after the second study. Body position was not noted, but an increased proportion of rapid eye movement sleep was noted on the second PSG in one of these studies. The available evidence indicates that 8–25% of symptomatic patients with an initial negative PSG will have OSA diagnosed after the second PSG.

A false-negative study may exclude a patient from therapy and expose them to increased morbidity in the long term. On the other hand, repeat PSG after an initial negative study carries with it the potential for increased expense and inconvenience to the patient and the possibility of a false-positive test. If the patient is symptomatic, the potential benefits of this approach may outweigh the risks, but the evidence supporting this recommendation is weak. A thorough discussion with the patient is warranted in this situation so that they can make an informed choice about undergoing a second PSG.

7.4.6 Repeat Testing in the Long Term

There is a general lack of evidence regarding the performance of repeat PSG in patients with OSA with stable symptoms, weight, and comorbidities who are adherent to PAP, in terms of whether this affects classification of type or severity of SDB in an individual patient or has any influence on long-term clinical outcomes. According to current Centers for Medicare and Medicaid Services (CMS) coverage criteria for PAP devices in the United States, a repeat diagnostic and/or titration PSG showing an AHI ≥5/hour is required if more than 10 years have elapsed since the time of the original diagnostic study and the patient has not obtained a new PAP device in this time frame,.

If there is a significant change in medical comorbidities (and there is a suspicion for change in type/severity of SDB or PAP device type/pressure requirements) or ≥ 10% change in body weight from the time of the diagnostic or titration study, repeat PSG could be considered. CMS requires a titration PSG demonstrating adequate control of SDB (AHI <10/hour) on PAP titration PSG before nocturnal supplemental oxygen treatment can be prescribed for persistent hypoxemia.

7.4.7 Summary of PSG

PSG is considered the gold-standard test for the diagnosis of OSA. While HSAT may be appropriate in certain situations, PSG is recommended for the evaluation of OSA in patients with coexisting cardiopulmonary/neuromuscular comorbidities and/or other complicating medical, environmental, or personal factors, and when other sleep disorders are suspected.

7.5 Conclusions

More accurate screening tools may help identify which patients are candidates for testing with HSAT or PSG for the diagnosis of OSA. The advent of biomarkers that can screen for OSA and/or identify patients at increased risk for adverse outcomes may also help with the prioritization and individualization of testing and treatment for OSA. Research on the factors influencing inadequate/inconclusive/negative/complex ("failed") HSAT in patients with a high pretest probability of moderate-to-severe OSA as well as patient preferences regarding mode of testing is needed. Further studies are required regarding the accuracy and long-term outcomes of HSAT versus PSG in more diverse patient populations, including more female and ethnically/racially diverse populations, those with significant comorbid cardiopulmonary and neuromuscular conditions, and patients with other complicated environmental/personal factors. The accuracy, clinical implications and cost-effectiveness of portable monitoring or "HSAT"s versus PSG performed in the hospital setting, single versus multiple-night HSAT, PSG versus repeat HSAT for a failed HSAT, split-night versus full night PSG, second versus no PSG when the first one is negative and the role of repeat PSG in chronic disease management needs clarification.

7.6 Summary Box

Based on the AASM Clinical Practice Guideline for the Diagnostic Testing for Adult OSA

No.	Recommendation	Strength of evidence
1	Questionnaires, clinical prediction tools and algorithms should not be used (in the absence of PSG or HSAT)	Strong
2	PSG should be used, or HSAT with a technically adequate device in an uncomplicated patient with increased clinical pretest probability of moderate-to-severe OSA	Strong
3	PSG should be performed in the event of a single negative/inconclusive/technically inadequate HSAT	Strong
4	PSG should be performed instead of HSAT in patients with significant cardiopulmonary/neuromuscular conditions, suspected hypoventilation, chronic opioid medication use, history of stroke or severe insomnia	Strong
5	Split-night rather than full night PSG should be performed if clinically appropriate	Weak
6	A second PSG can be considered if the initial PSG is negative and there is still a clinical suspicion for OSA	Weak

Abbreviations: *AASM* American Academy of Sleep Medicine, *OSA* obstructive sleep apnea, *No* number, *PSG* polysomnography, *HSAT* home sleep apnea test

Further Reading

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Cone-Beam CT Use for Airway Imaging

Juan Martin Palomo, Tarek Elshebiny, and Kingman Strohl

Contents

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8.1 Introduction

Imaging as always was a key concept in craniofacial diagnosis and treatment planning. Airway imaging techniques used in the diagnosis of obstructive sleep apnea (OSA) have greatly improved especially with understanding OSA pathophysiology [1]. Treatment planning and evaluation of surgical and nonsurgical therapies which target-specific areas of obstruction are now possible with newer imaging modalities. Airway imaging can be done using numerous techniques such as nasal pharyngoscopy, cephalometric radiographs, fluoroscopy, conventional and electron beam computed tomography (CT), acoustic reflection, magnetic resonance imaging (MRI), and cone-beam computed tomography (CBCT) [2].

The importance of the third dimension for airway assessment has been emphasized highlighting important limitations of two-dimensional studies. The high radiation dose of conventional CT devices might have been a limiting factor for its use, but the evolution of a new technology, CBCT, took a significant turn. CBCT has become a well-accepted oral and maxillofacial diagnostic imaging, providing a three-dimensional view of hard and soft tissues of the head and neck with low doses of radiation. CBCT studies allow us to examine areas, volumes, and complex hollow structures, including the airway channel (Fig. 8.1) [3–5]. Several CBCT manufacturers currently use pulse technology where radiation only occurs when taking images, allowing the creation of a three-dimensional (3D) image with less radiation to the patient than a panoramic radiograph [6].

The purpose of this chapter is to describe the possible use of CBCT technology for airway evaluation in OSA patients. Since a CBCT does not provide any information on neuromuscular tone, susceptibility to collapse, or actual function of the airway, one cannot use a CBCT to diagnose sleep apnea alone. A CBCT may be used for monitoring conditions or for treatment considerations, such as the location of the maximum constriction, so the clinician can decide between expansion, if it is located in the nasal area, and mandibular advancement devices, if it located in the oropharynx, for example.

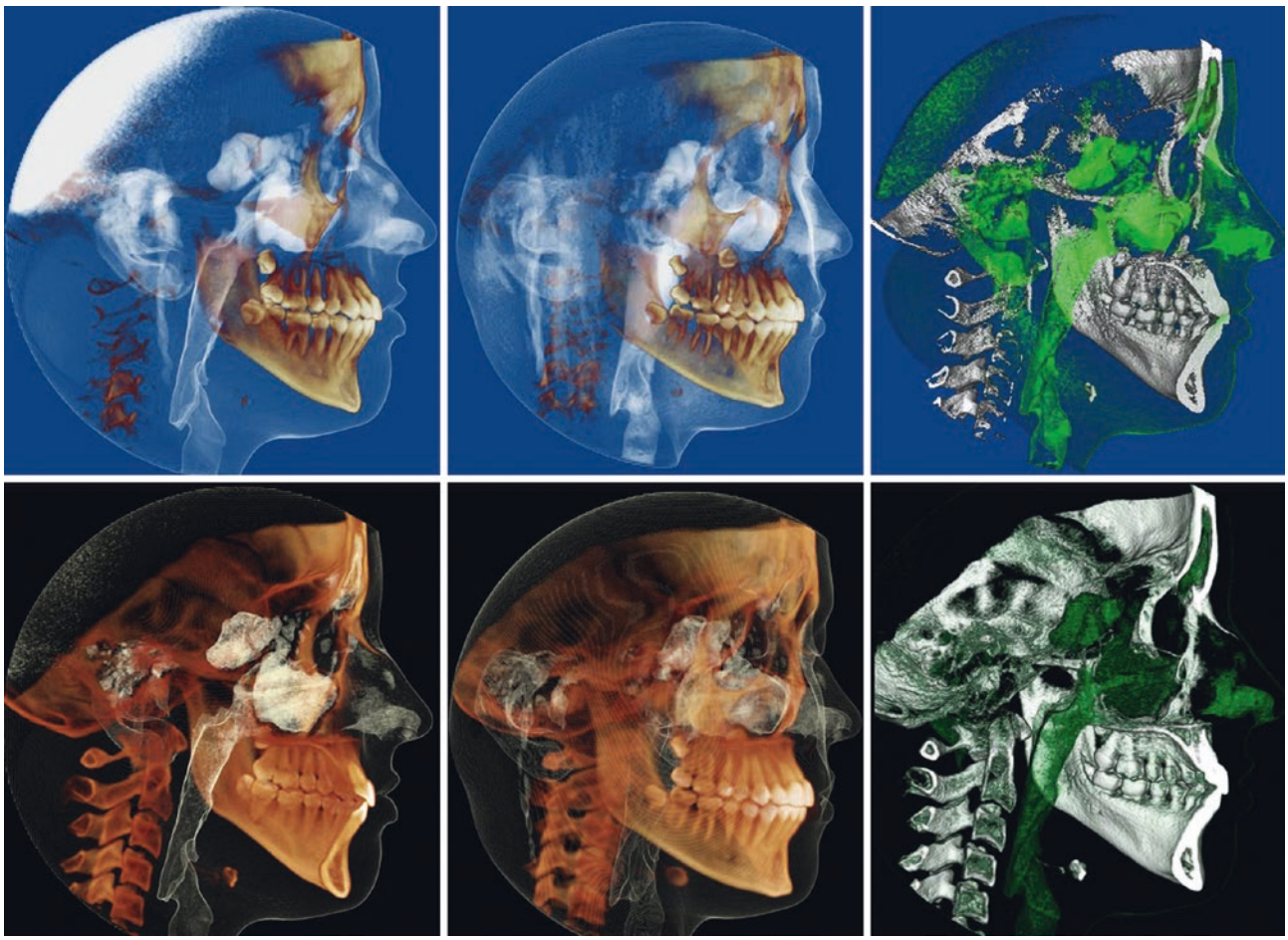


Fig. 8.1 Different views of a CBCT volume showing the upper airway using two different software packages

8.2 Accuracy and Reliability of Measuring the Airway Using CBCT

Validation of CBCT technology to measure the airway has been evaluated in several studies [3, 7–11]. Accuracy and reliability of airway volume digital measurements of CBCT compared with the manual measurements of an airway model was investigated by constructing an acrylic airway model attaching it to a human dry skull in the natural position of the airway passage [7]. The total and internal airway volumes, as well as the most constricted airway area, were measured manually on the model and on the CBCTs taken after the model was attached to the skull. Results suggested that the CBCT digital measurements of the airway volume and the most constricted area of the airway are reliable and accurate [7]. Another article evaluated the accuracy of measurements made on CBCT images compared with the gold standard which is measurements made on a coordinate measuring machine [8]. The authors found that the coordinate correlation coefficient was virtually identical between the 3-3D CBCT images and the coordinate measuring machine measurements [8]. Evaluation of upper airway using airway phantoms report high accuracy and reliability of CBCTs in measuring airway [9]. Amirlak et al. used a manual segmentation program to test the reliability and accuracy of CBCT images in measuring volumes, of artificial defects, subsequently filled with polyvinyl siloxane (PVS). They used a water displacement technique for comparing the CBCT volumes with actual volumes and found that the manually segmented volumes were highly accurate compared to the water displacement technique which is considered as the gold standard [10]. Tsolakis et al. investigated the difference between CBCT and the acoustic reflection (AR) imaging technique in calculating airway volumes and areas. Subjects with prescribed CBCT images as part of their records were also asked to have AR performed. A total of 59 subjects had their upper airway measured from CBCT images, acoustic rhinometry, and acoustic pharyngometry. It was found that CBCT is an accurate method for measuring anterior nasal volume, nasal minimal cross-sectional area, pharyngeal volume, and pharyngeal minimal cross-sectional area [11]. Commercially available digital imaging and communications in medicine (DICOM) viewers for measuring upper airway volumes were compared to show that manual segmentation was more accurate than semiautomatic segmentation, but all of them showed high correlations, suggesting the existence of a systematic error in the derivation of the airway volume [3].

8.3 Evaluation of Upper Airway Using CBCT

This section provides a step-by-step guide for the airway analysis.

(a) Orientation

In order to create different views in a standardized way and to obtain consistent measurements using CBCT images, image orientation should be the first step within the software. A simple way to orient the volume can be done by adjusting the midsagittal plane on the skeletal midline of the face, then adjusting the axial plane on top of the Frankfort horizontal plane, and the coronal plane so it passes through the level of the furcation point of the right maxillary first molar (Fig. 8.2) [12]. The Case Western Reserve University (CWRU) orientation method is a more complex one and uses five biologically relevant anatomic structures and one plane [13].

(b) Segmentation

Upper airway segmentation can be performed accurately either manually which is more time consuming or semiautomatically which is significantly faster. In the semiautomatic approach, the software automatically differentiates the air and the surrounding soft tissues by using the differences in density values of these structures because the air space is of a greater negative Hounsfield unit than the more dense surrounding soft tissue. A new tool in commercial software products allows us to visualize the different densities in different tissues in the craniofacial complex with the aid of the Hounsfield Unit Color Mapping, which helps distinguish between different biological structures according to their radiolucency measurements. A graph displays the colors as they fall on the Hounsfield Scale as a reference, while the image on the screen is colored accordingly (Fig. 8.3). Automatic segmentation also allows the airway to be measured along a curved path, instead of simply along horizontal slices and an airway color coded by constriction is presented. Another method to measure the airway by setting boundaries to the region of interest based on specific anatomical landmarks. Although it is mainly automatic, adjusting the airway sensitivity scale and placing seeds in the regions of interest to allow accurate segmentation is required in some commercial software products. Figures 8.4, 8.5, and 8.6 show the different methods to measure the upper airway.

Segmentation of Different Regions of Interest. The below regions of interest are areas commonly studied with suggested anatomic limits for proper reliability. There may be slight variations found in different studies, and slight variations with the true anatomic definition of such region.

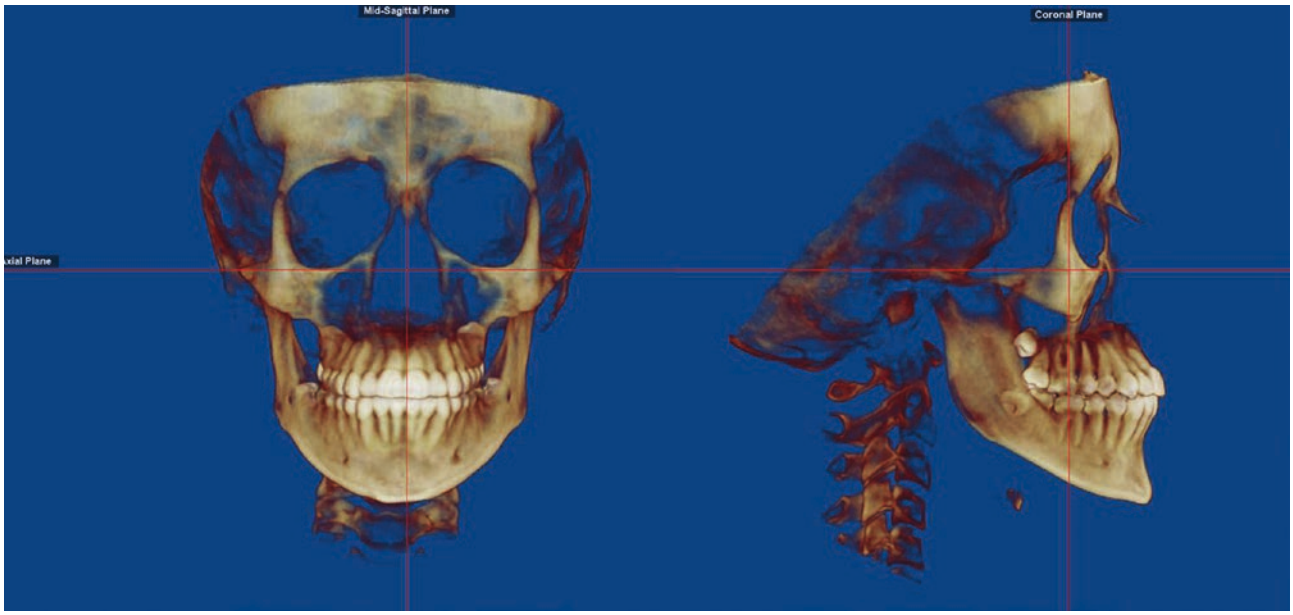


Fig. 8.2 Volume orientation can be done by adjusting the mid-sagittal plane on the skeletal midline of the face, then adjusting the axial plane on top of the Frankfort horizontal plane, and the coronal plane, so it passes through the level of the furcation point of the right maxillary first molar

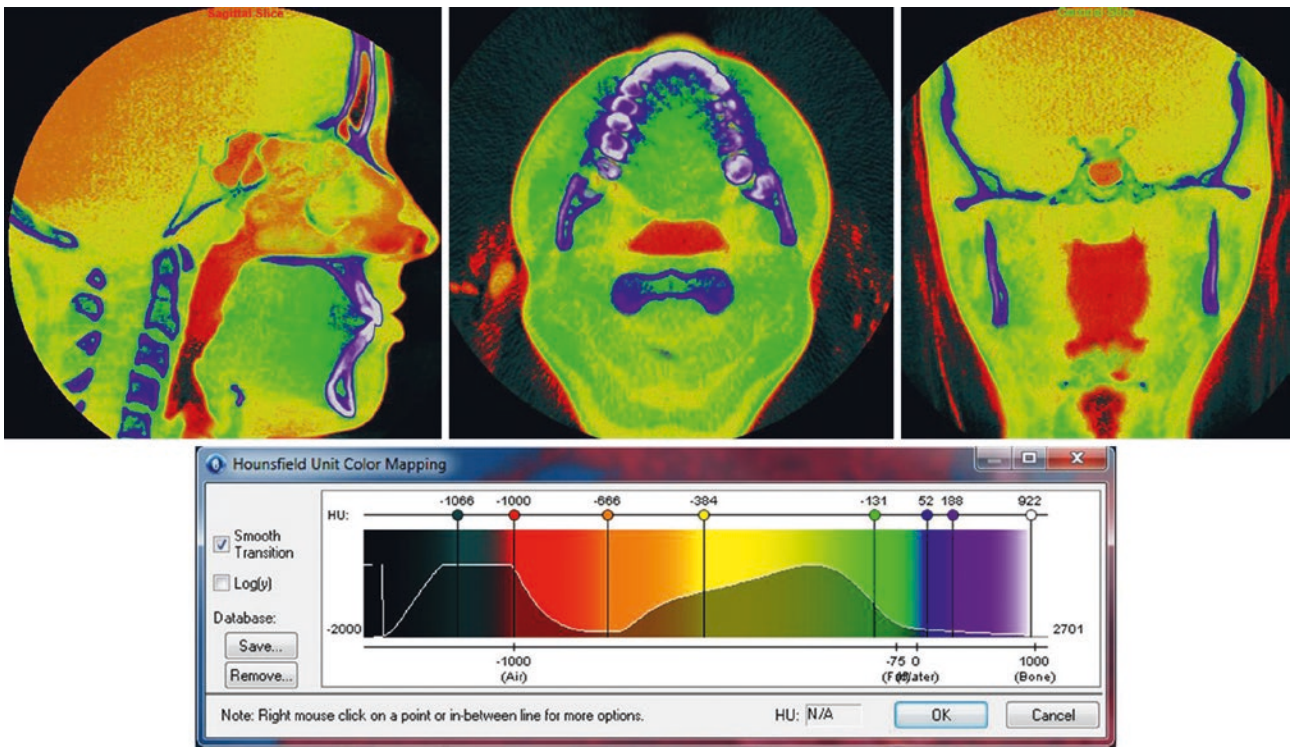


Fig. 8.3 Three-dimensional reconstructed images in different planes of space showing different densities in different tissues in the craniofacial complex with the aid of the Hounsfield Unit Color Mapping tool, which helps you to distinguish between different biological structures according to their radiolucency measurements. A graph displays the colors as they fall on the Hounsfield Scale as a reference, while the image on the screen is colored in accordingly

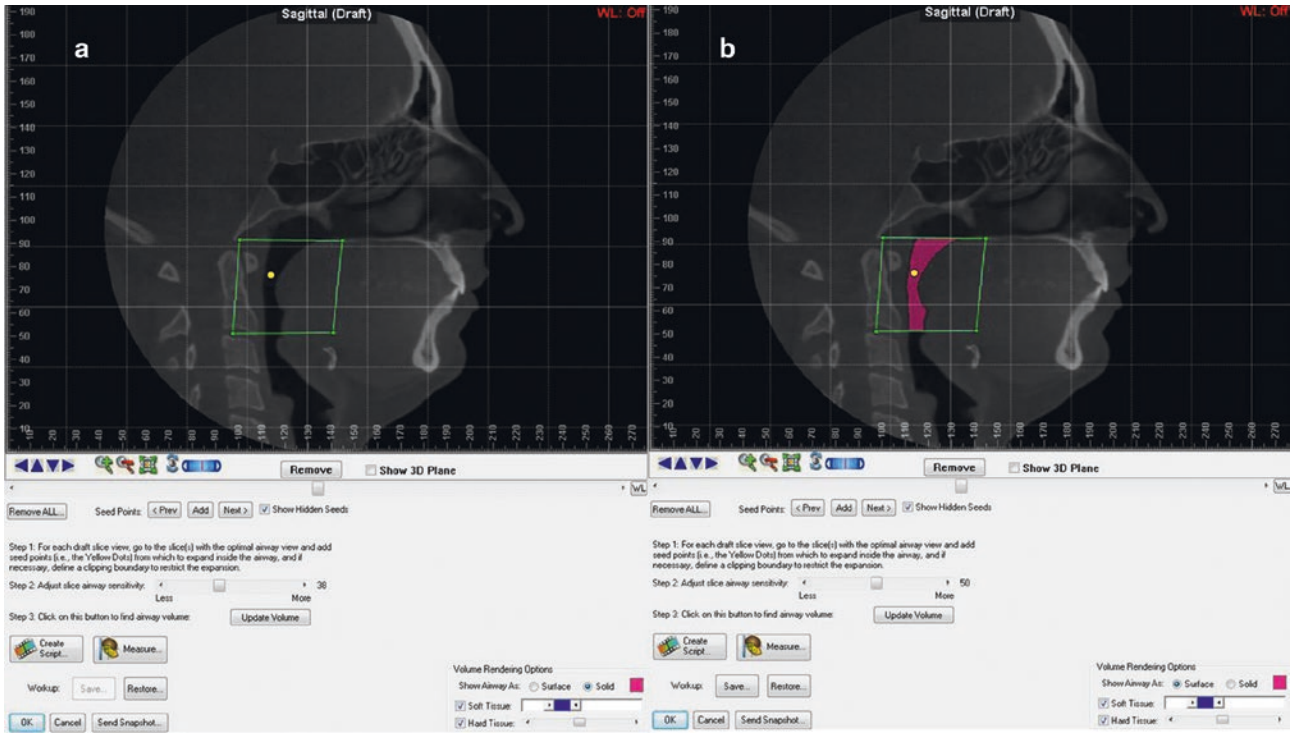
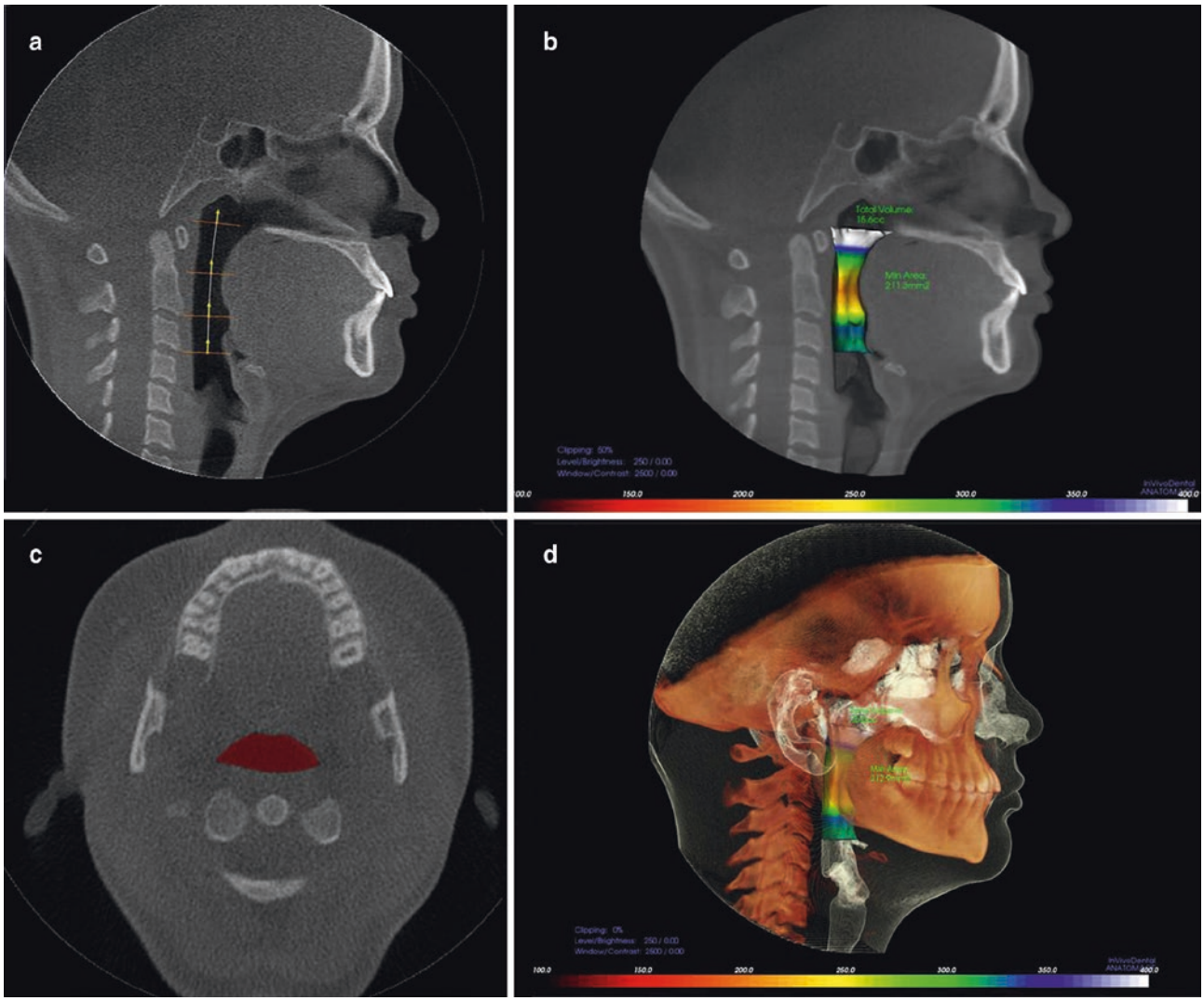


Fig. 8.4 Setting boundaries to the region of interest based on specific anatomical landmarks. Although it is mainly automatic, adjusting the airway sensitivity scale and placing seeds in the regions of interest to allow accurate segmentation is required

1. Nasal Passage: The volume is defined as being the pharyngeal volume located between the palatal plane and a parallel plane passing through the last axial slice before the nasal septum fused with the posterior pharyngeal wall. Once boundaries are outlined in the sagittal plane, additional boundaries outlining the respective airway are made in the axial and coronal planes (Figs. 8.7 and 8.8).
2. Oropharynx Volume: The volume is defined as the pharyngeal volume located between a plane passing through the palatal plane (PNS-ANS) and a parallel plane passing through the most antero-inferior point of the second cervical vertebrae (Fig. 8.9).
3. Hypopharynx Volume: The volume is defined as the pharyngeal volume located between the inferior limit of the oropharynx volume and a parallel plane passing through the most antero-superior point of the hyoid bone (Fig. 8.10).
4. Retro-palatal Volume: From the level of posterior nasal spine to the lower edge of the soft palate (Fig. 8.11a).
5. Retro-glossal Volume: From the lower edge of the soft palate to the hyoid bone (Fig. 8.11b).
6. The minimum axial area, also known as the area of maximum constriction (mm^2), can be determined for the whole airway volume or just in specific region of interest. Maximum constriction area can be presented in an automatic setting with volume measurement or can be enabled through features in different software packages (Fig. 8.12).

8.4 CBCT and OSA

OSA is a common disorder characterized by collapse of the upper airway during sleep resulting in hypoxemia and arousal [14]. CBCT can be employed to assess the location of obstruction in OSA patients. One study compared the upper airway structure in OSA patients and control subjects using CBCT images [15]. It was shown that OSA subjects presented lower total airway volume, smaller anterior-posterior dimension of the minimum cross-section segment, and smaller minimum cross-section area. Also, the OSA group showed elliptic-shaped airway, while the non-OSA group showed round or square airway [15]. Another study compared CBCT scan measurements between patients with OSA and snorers to develop a prediction model for OSA based on CBCT imaging and the Berlin questionnaire. It was found out that the upper airway dimension was significantly smaller in the OSA patients [16]. Recent study evaluated the upper airway dimensions of OSA and control subjects using CBCT. Results showed that OSA subjects had a significantly smaller average airway area,



Figs. 8.5 and 8.6 Automatic segmentation of the airway measured along a curved path, instead of simply along horizontal slices and an airway color coded by constriction is presented

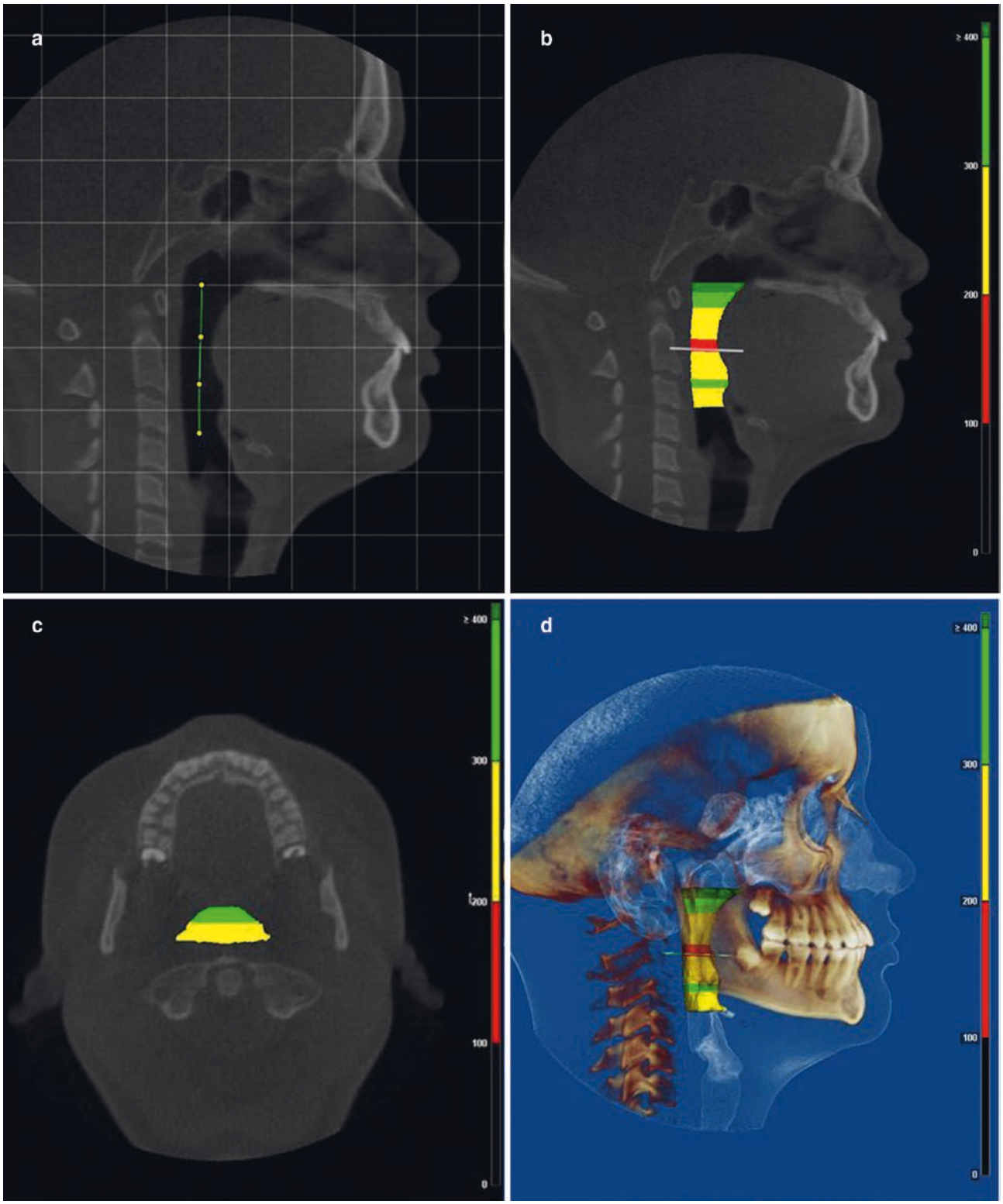


Fig. 8.5 and 8.6 (continued)

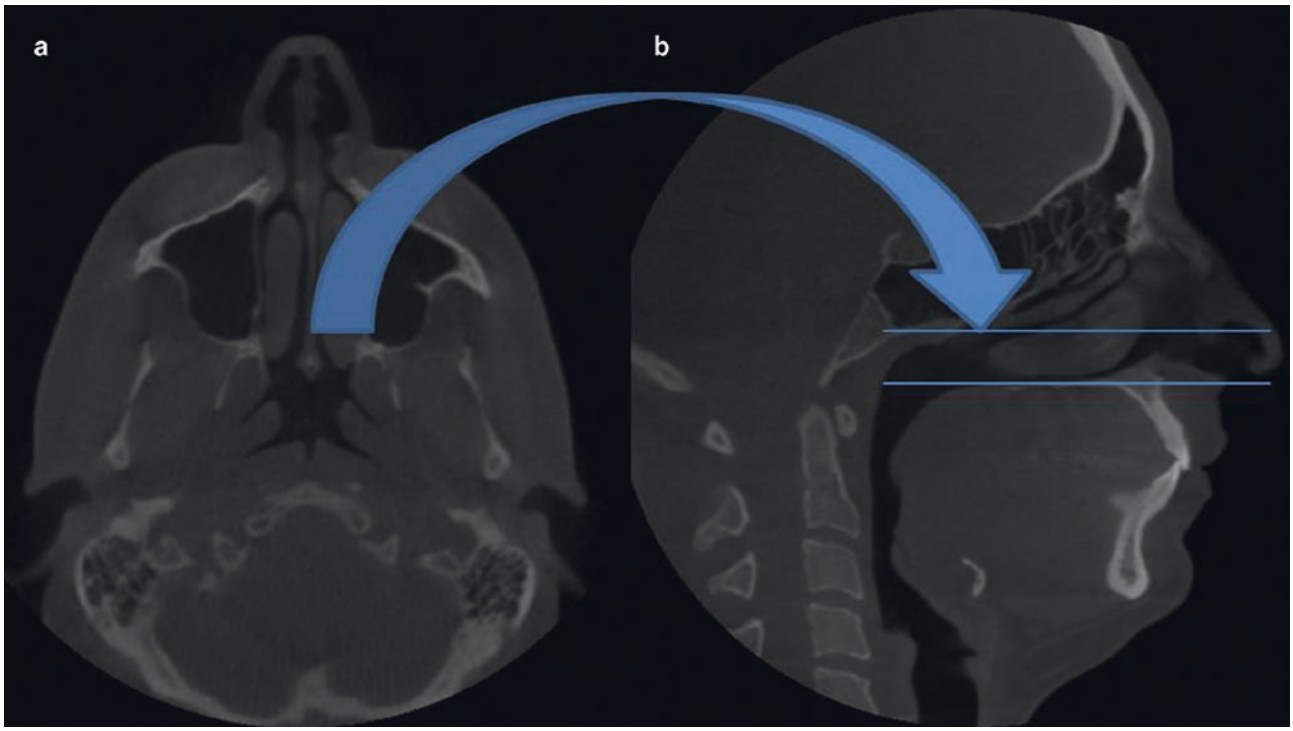


Fig. 8.7 Nasal passage volume is defined as being the pharyngeal volume located between the palatal plane and a parallel plane passing through the last axial slice before the nasal septum fused with the posterior pharyngeal wall

average airway volume, total airway volume, and mean airway width. OSA subjects had a significantly larger airway length measurement [17].

The hyoid bone which is a predictor of airway obstruction plays an important role in the airway patency. Links between hyoid position and airway resistance have been demonstrated in the literature [18–20]. The increased distance of the hyoid bone to mandibular plane has shown to be correlated to OSA in the literature; more than 15 mm is considered abnormal and associated with OSA [21]. One study demonstrated the correlation of upper airway resistance with the posterior airway space and the vertical/horizontal position of the hyoid bone. A more downward position of the hyoid bone was demonstrated in OSA subjects compared to normal subjects [22].

8.5 Evaluation of OSA Treatment Approaches Using CBCT

Continuous positive air pressure (CPAP) is the standard, first-line therapy for treating OSA; however, the general effectiveness of initial CPAP therapy is dependent on patient acceptance and adherence to treatment [23]. Other options include oral appliances, nerve stimulation, and surgical procedures to anatomically improve airway function.

8.5.1 CPAP

CPAP is the most effective method to manage OSA. It improves subjective and objective measures of sleepiness [24].The most significant effect is enlargement of the airway by dimensional changes of the lateral pharyngeal walls. Our study in the 1980s showed that CPAP acts as a pneumatic splint and passively open the upper airway to prevent obstructive apnea [14]. A recent study evaluated OSA patients by taking CBCT scans during application of positive and negative pressures to the respiratory system while awake and seated [25].The first scan was taken during a cycle of resting breathing. Two other images were obtained when pressure was applied using a full facemask also in the seated posture during wakefulness. The mask was connected to a positive/negative pressure source. One scan was taken while breathing on a mask pressure of +10 cm H_2O and the other scan was taken while the patient breathed against a mask pressure of – 2 cm H_2O (Figs. 8.13 and 8.14). Positive pressure application of +10 cm H_2O showed significant airway volume increase in all regions (36%). The hypopharynx volume increased the most with 50%, followed by oropharynx with 23%, and the nasopharynx with 17.7%. The minimal cross-section area changed from 100.57 ± 38.74 mm^2 to 130.64 ± 64.01 mm^2.There was no significant change of the tongue length when using

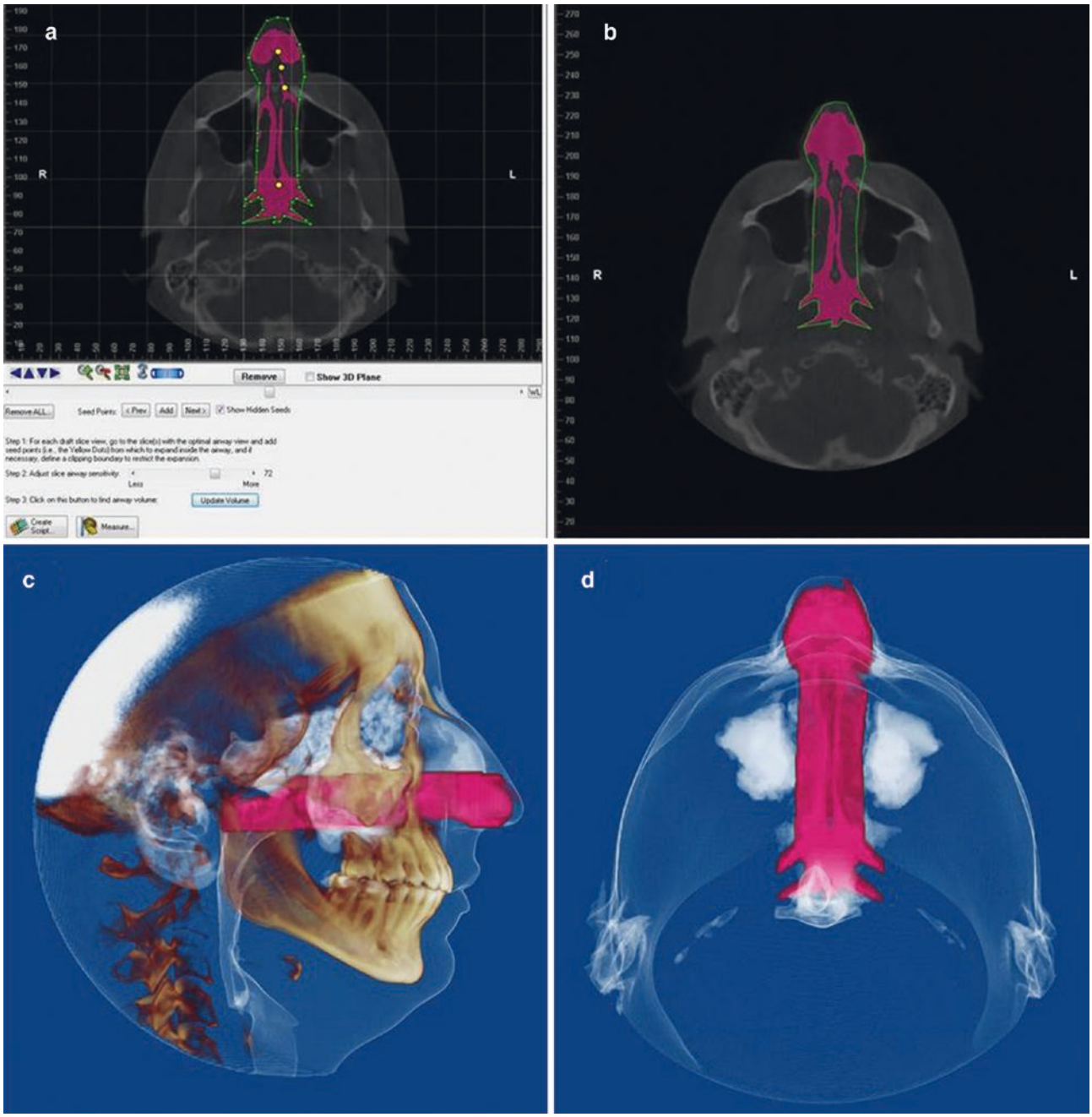


Fig. 8.8 Nasal passage volume segmentation and outcome

positive pressure. Superimposition on the cranial base showed no change in the hyoid bone position vertically or horizontally. Negative pressure showed a significantly airway volume decrease in all regions (−28%). The volume of the upper airway was nearly collapsed with the negative pressure. The oropharynx decreased the most, with −31%, followed by the hypopharynx with 30%, and the nasopharynx with 19.0%. The average minimal cross-section area changed from 100.57 ± 38.74 mm^2 to 52.00 ± 23.01 mm^2 with no change in hyoid bone position and tongue length. It was concluded that increases and decreases in intraluminal pressure alter the airway geometry but do so without changing hyoid position.

8.5.2 Oral Appliances

An oral appliance is fitted to the upper and lower teeth and is designed to work by fixing and/or anterior positioning the mandible, preventing the collapse of the

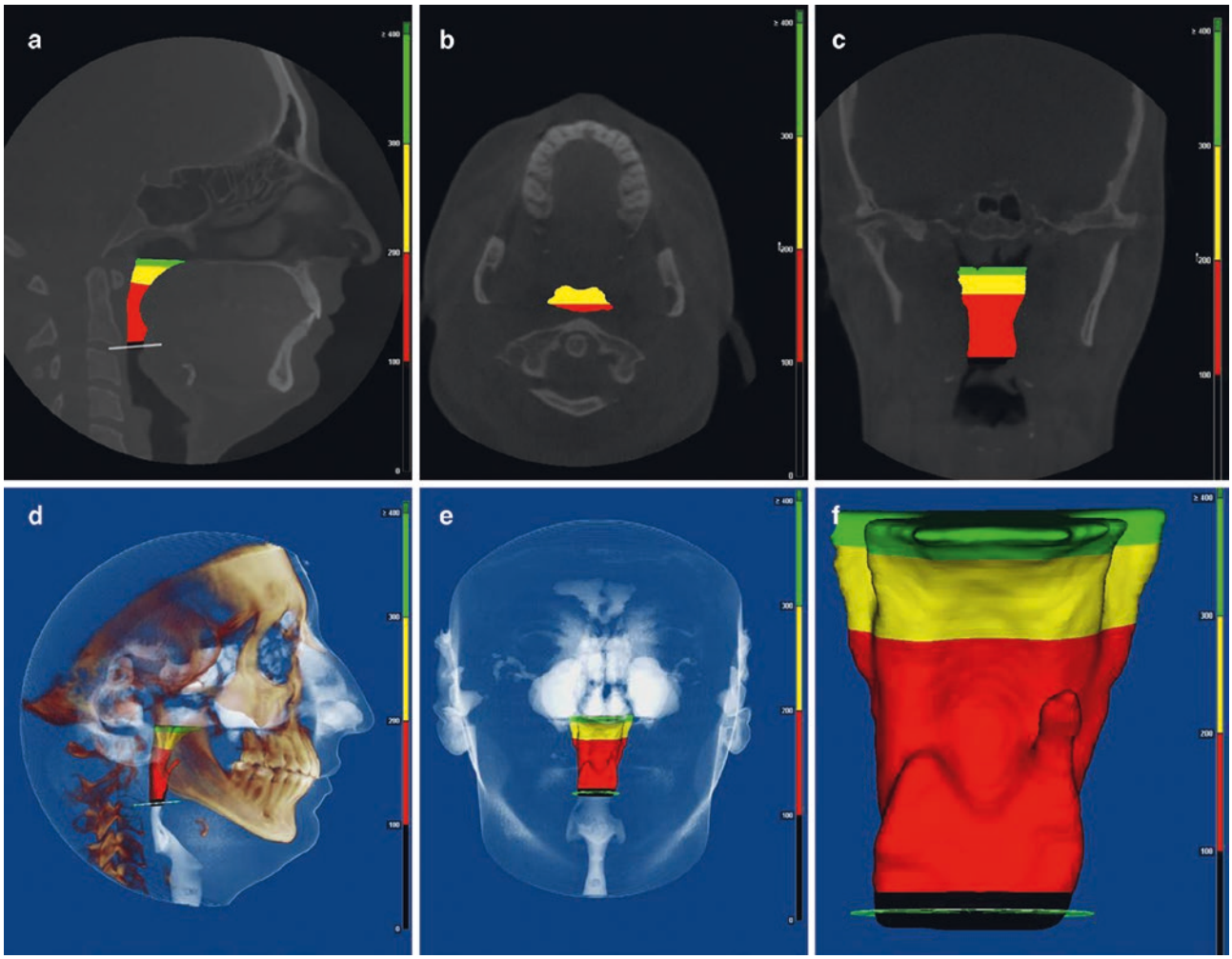


Fig. 8.9 Oropharynx volume is defined as the pharyngeal volume located between a plane passing through the palatal plane (PNS-ANS) and a parallel plane passing through the most antero-inferior point of the second cervical vertebrae

tongue and/or increasing the posterior oropharyngeal airway space, therefore reducing the collapse of the upper airway during sleep [26]. Oral appliances can be first-line therapy but are more commonly used for patients who are not compliant with CPAP and diagnosed with mild and moderate OSA; and oral appliance also treats simple snoring. Recently, the American Academy of Sleep Medicine recommended the oral appliances as a first line of therapy in patients with mild-to-moderate OSA. In a randomized clinical trial, the treatment outcome of oral appliances and CPAP therapy of OSA patients was reported to show that oral appliance should be considered as an alternative to CPAP in patients with mild-to-moderate OSA. While patients with severe OSA, CPAP should remain the first line of treatment [26].The most commonly used oral appliances are the mandibular advancement devices which reposition the mandible, tongue, and hyoid bone anteriorly to increase dimensions of the upper airway [27, 28]. The upper airway structures were evaluated in patients with OSA by using MRI scans of upper airway with and without oral appliances. It was shown that the upper airway was increased mainly by increasing the volume of the velopharynx [29].

Figure 8.15 shows airway volume changes for one patient with and without oral appliance.

Dental side effects associated with oral appliances and CPAP were assessed in a randomized clinical trial to show that there is a small dental change with oral appliances but significant if compared with CPAP. Different studies showed decrease in overbite and overjet, proclination of lower incisors, retroclination of upper incisors [30–35].

8.5.3 Maxillomandibular Advancement

Maxillomandibular advancement (MMA) surgery is a well-established treatment of obstructive OSA [36]. The rationale for MMA is to increase the anteroposterior and the lateral dimensions at multilevels of the upper airway [37], and reduce upper airway collapsibility with the superior and anterior movement of the hyoid

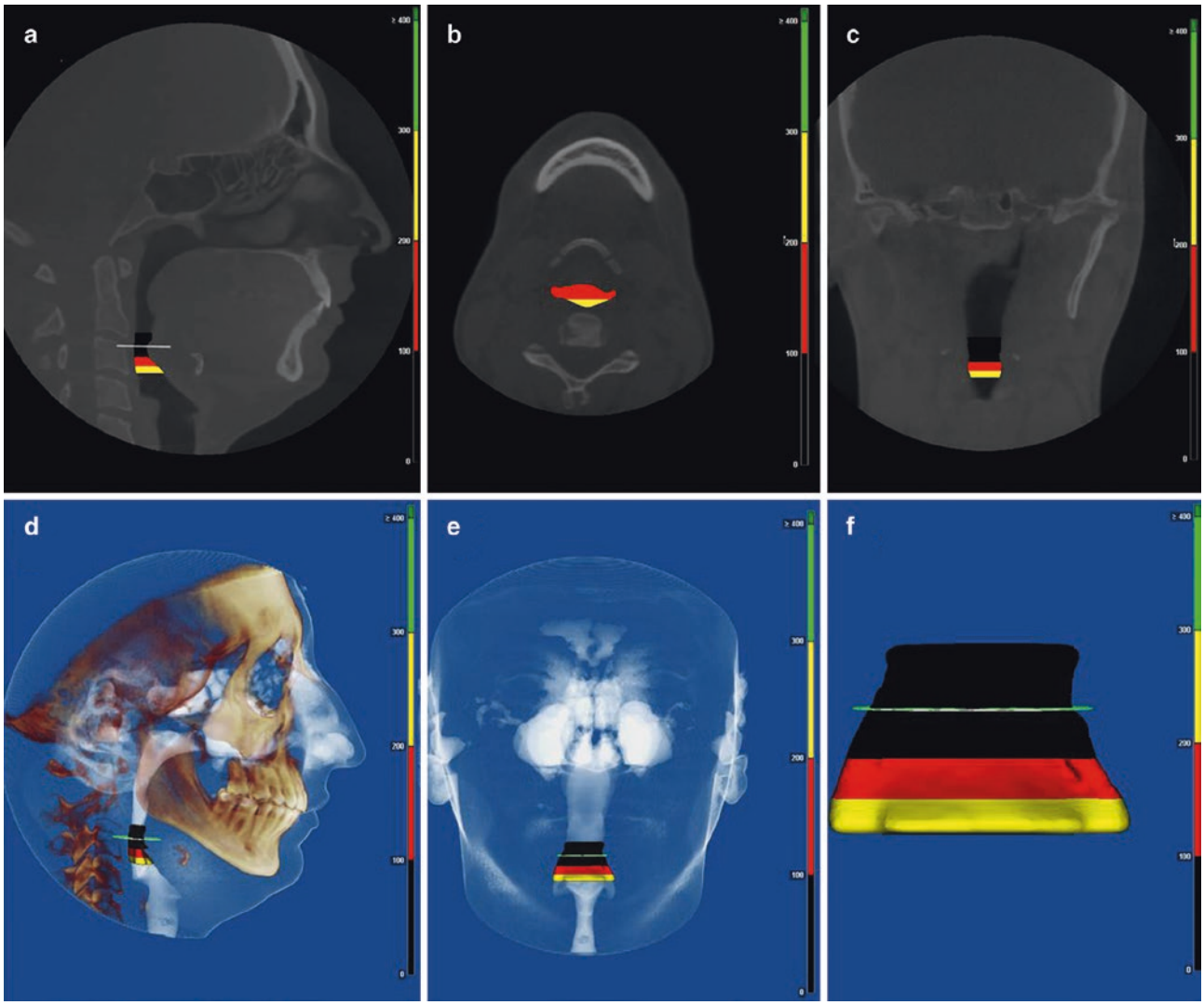


Fig. 8.10 Hypopharynx volume is defined as the pharyngeal volume located between the inferior limit of the oropharynx volume and a parallel plane passing through the most antero-superior point of the hyoid bone

bone [38]. In systematic review of data regarding MMA advancement for OSA treatment, it was shown that MMA advancement is the most successful surgical therapy for OSA [39]. Schendel et al. evaluated 10 patients with moderate or severe OSA who underwent MMA surgery by preoperative and postoperative cone beam computed tomography scans and polysomnograms. The volume of the UAS increased significantly by 237% as a result of the MMA. The retropalatal volume increased more than retroglossal volume, 361% to 165% [21]. Linear and volumetric morphological changes of upper airway after MMA for OSA patients were assessed using CBCT. It was shown that MMA increased the airway total volume, minimal cross-sectional area, anteroposterior and lateral dimensions, airway index, airway length, posterior airway space morphology, Apnea–hypopnea index (AHI), and Epworth sleepiness score [36]. CBCT images are recommended for three-dimensional airway and soft tissue evaluation in treatment of obstructive sleep apnea syndrome (Fig. 8.16) [40].

8.6 Upper Airway Stimulation

A relatively novel and cutting edge treatment is upper airway electrical stimulation (UAS) therapy using a fully implanted system. The Inspire implant (Inspire Medical Systems, Inc., Maple Grove, MN and FDA approved in April 2014) is offered for the treatment of moderate-to-severe obstructive sleep apnea who cannot use CPAP therapy, and is known to decrease the severity and symptoms of OSA in selected patients [41–44]. The therapeutic approach, as initially described, is to deliver stimulation to the hypoglossal nerve, synchronized with breathing efforts [45]. The patient can turn the therapy on before bedtime, and off in the morning

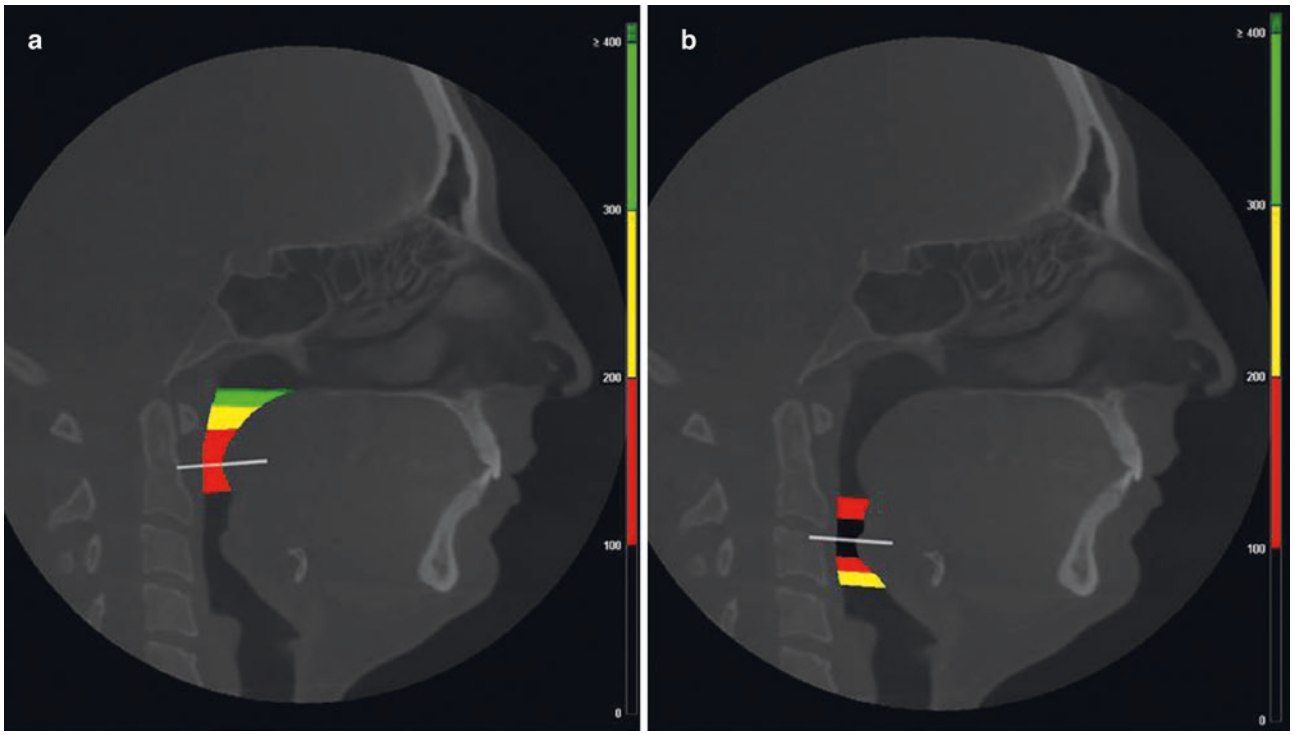


Fig. 8.11 **a** Retropalatal volume: From the level of posterior nasal spine to the lower edge of the soft palate. **b** Retroglossal volume: From the lower edge of the soft palate to the hyoid bone

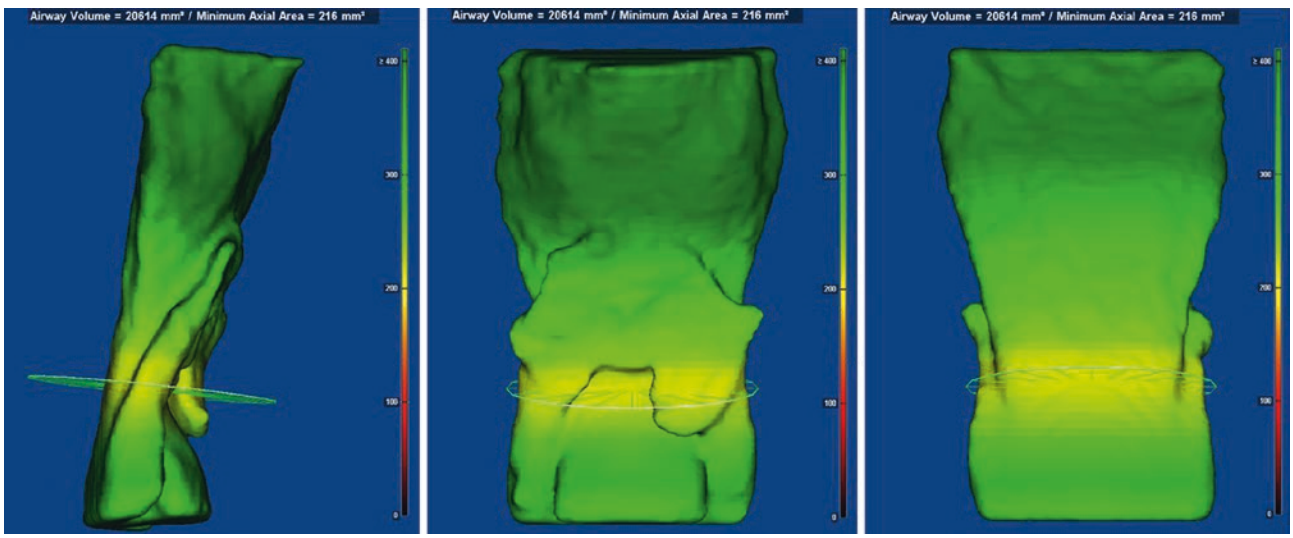


Fig. 8.12 The minimum axial area, also known as the area of maximum constriction (mm^2), which can be determined for the whole airway volume or just in specific region of interest

using a remote control. When the device is activated, it senses the person's breathing patterns, delivering mild stimulation in order to keep the airway open, acting in a similar way than a pacemaker. The level of stimulation can be custom to each patient depending on patient's unique BMI and AHI. The UAS system is implanted on the right-hand side of the patient while under general anesthesia, through three surgical incisions. The median time for implantation has been reported to average 140 minutes, with most patients spending the night at the hospital [43]. A recent study evaluated seven patients who had previously undergone surgical implantation for UAS therapy at the University Hospitals Case Medical Center (Cleveland, OH); all were regularly using therapy. Each had been deemed a candidate on the basis of (a) CPAP intolerance, (b) an

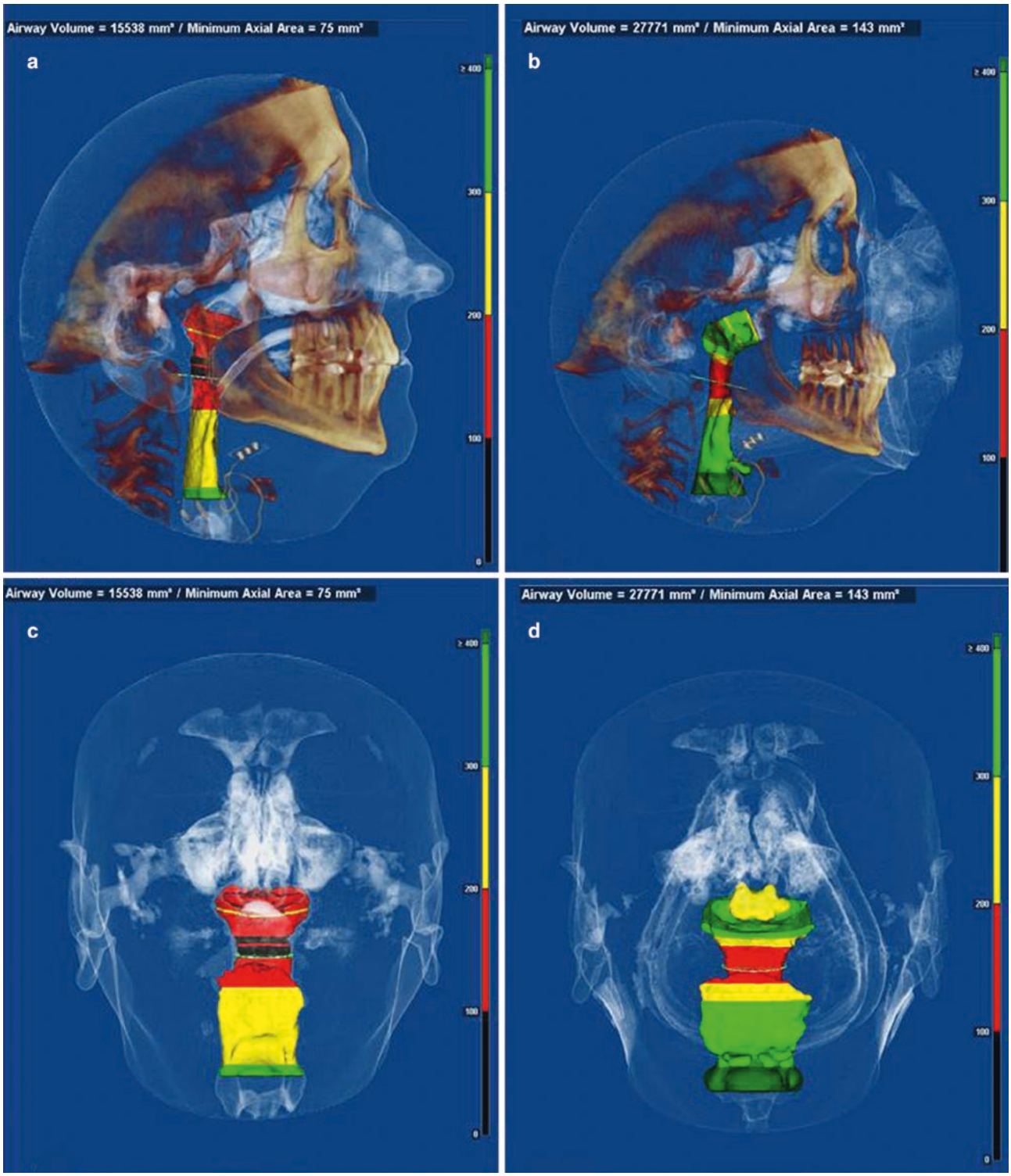


Fig. 8.13 Three-dimensional rendering images of the upper airway with cone-beam computed tomography for one patient. **a** Resting breathing: Sagittal view during a cycle of resting breathing showing the upper airway volume. **b** Application of positive pressure (+10 cm H_2O) by facemask. Sagittal view during application of +10 cm H_2O with an increase in the airway volume. **c** Frontal view in resting breathing. **d** Frontal view in positive pressure of +10 cm H_2O

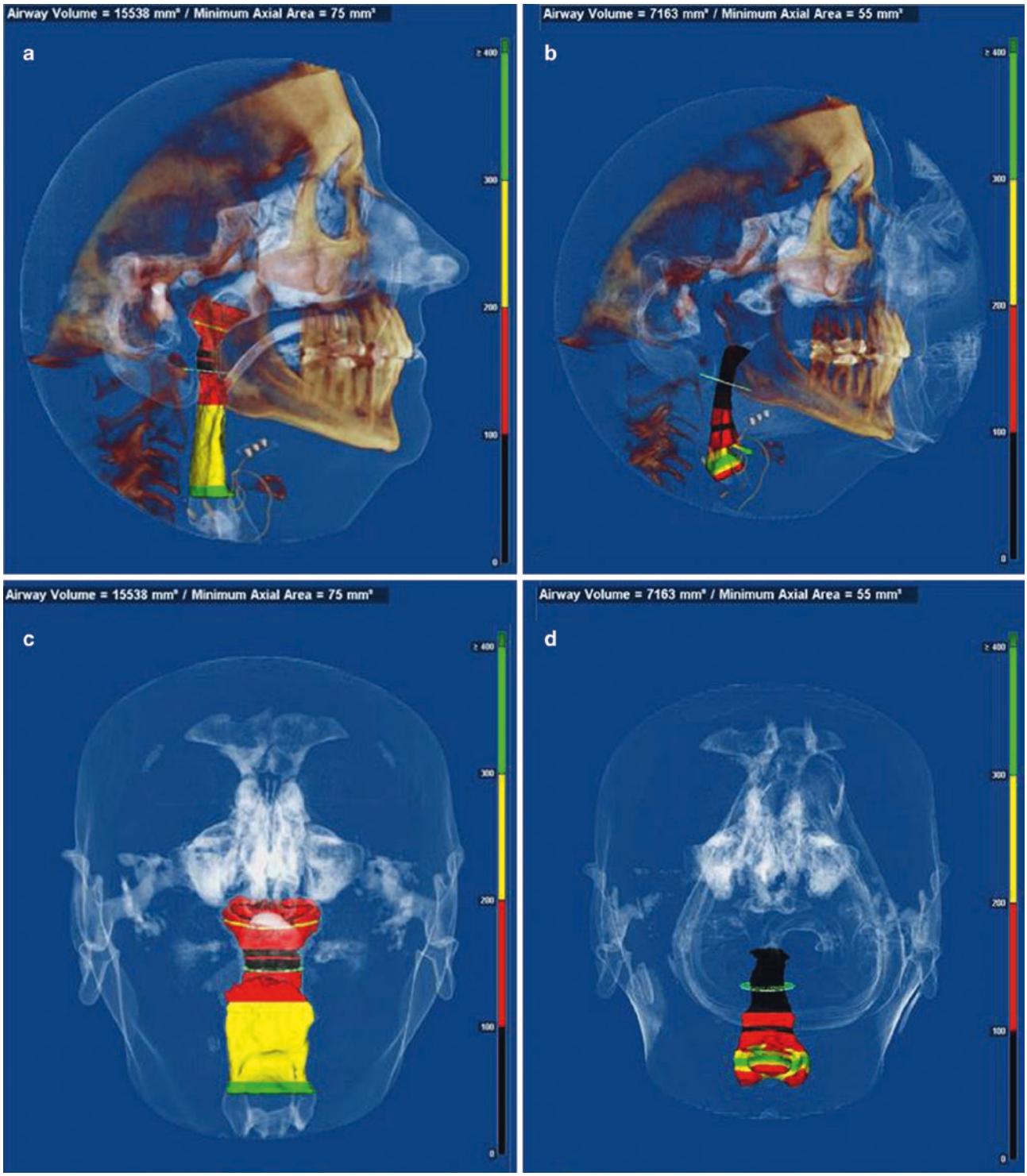


Fig. 8.14 Three-dimensional rendering images of the upper airway with cone-beam computed tomography for one patient. **a** Resting breathing: sagittal view during a cycle of resting breathing showing the upper airway volume **b** Application of negative pressure (−2 cmH_2O) by facemask. Sagittal view during application of -2 cm H_2O with an increase in the airway volume. **c** Frontal view in resting breathing **d** Frontal view in negative pressure of −2 cm H_2O

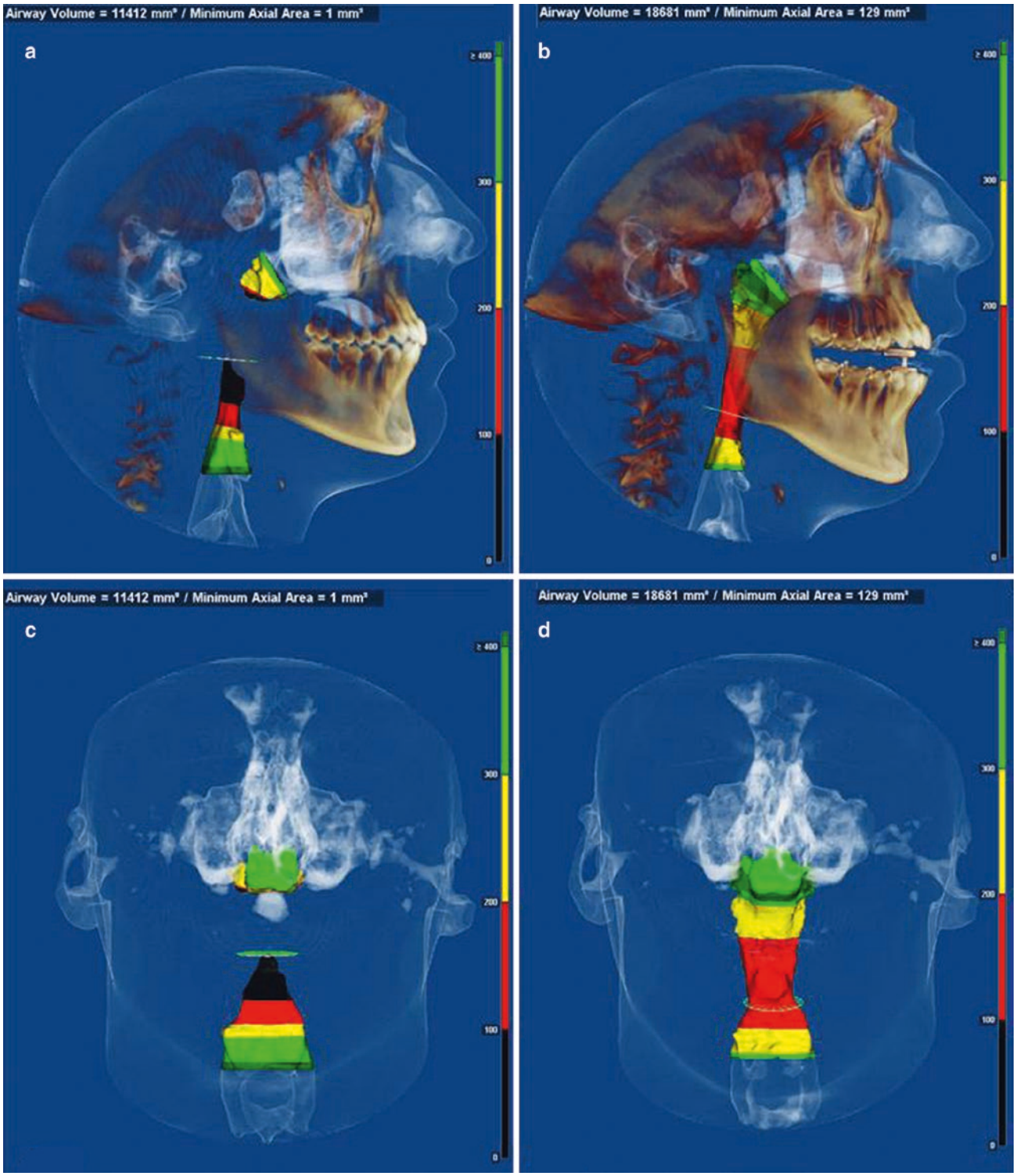


Fig. 8.15 Three-dimensional rendering images of the upper airway with cone-beam computed tomography for one patient. **a** Resting breathing: Sagittal view during a cycle of resting breathing showing the upper airway volume. **b** Same patient with oral appliance showing an increase in the airway volume. **c** Frontal view in resting breathing. **d** Frontal view with an oral appliance in place

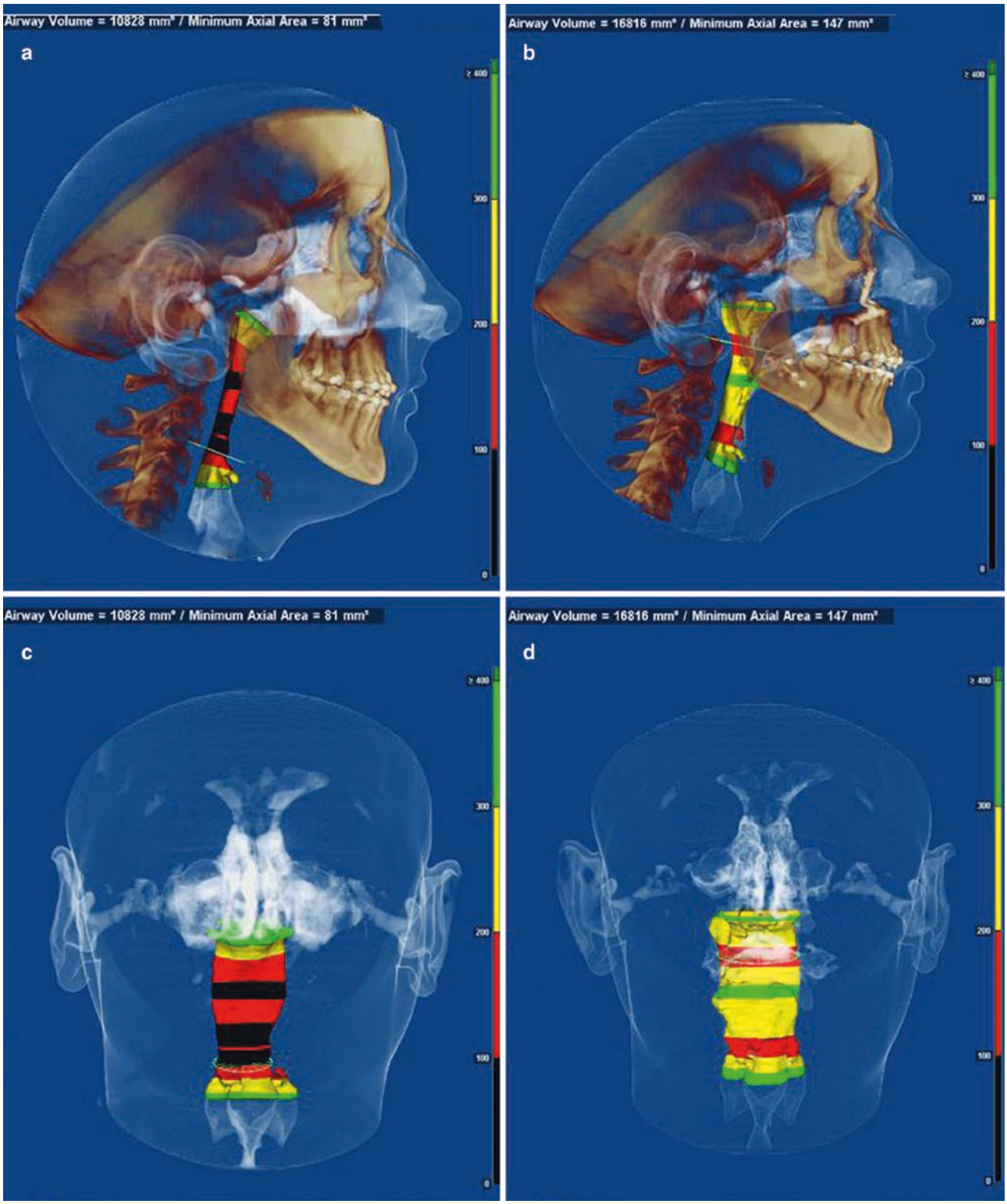


Fig. 8.16 Three-dimensional rendering images of the upper airway with cone-beam computed tomography for one patient. **a** Resting breathing: sagittal view during a cycle of resting breathing showing the upper airway volume. **b** Same patient after MMA showing increase in the airway volume. **b** Frontal view in resting breathing. **d** Frontal view after MMA

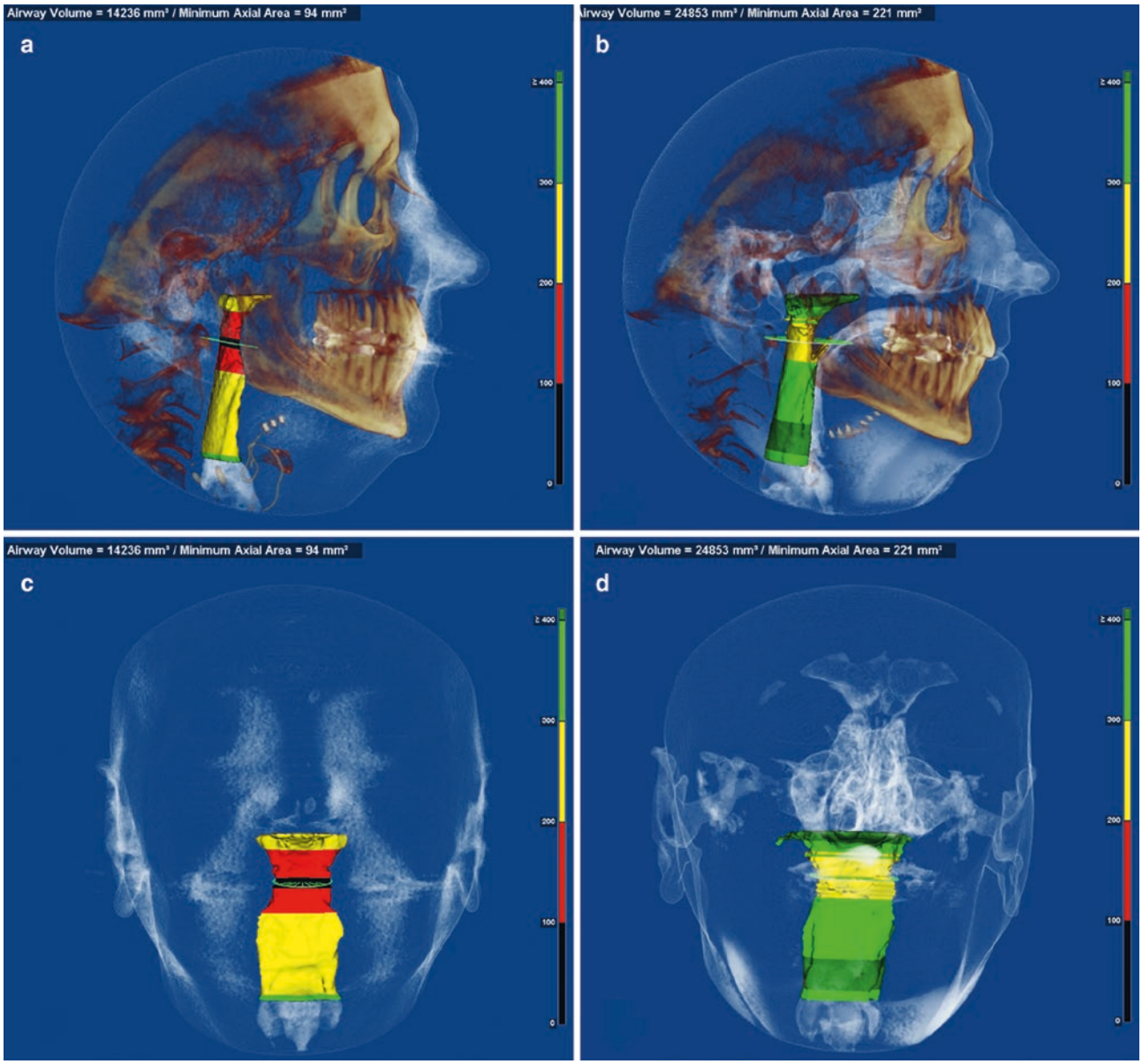


Fig. 8.17 Three-dimensional rendering images of the upper airway with cone-beam computed tomography for one patient. **a** Resting breathing: Sagittal view during a cycle of resting breathing showing the upper airway volume. **b** Sagittal view during stimulation of the hypoglossal nerve with an increase in the upper airway volume. **c** Frontal view in resting breathing. **d** Frontal view during hypoglossal nerve stimulation

AHI generally between 20 and 60/hour, (c) >75% of the AHI being obstructive apneas and hypopneas, and (d) closure during drug-induced sedation endoscopy showing a predominant anteroposterior collapse at the level of the velopharynx. Subjects were evaluated by CBCT scans and lateral cephalograms in regular breathing, during UAS therapy. The first scan was taken during a cycle of resting breathing. The second scan was taken during stimulation at voltage amplitude at or near that used therapeutically during sleep in that patient. The CBCT volumes taken under UAS of the hypoglossal nerve showed a significant increase along the upper airway (+48%). The hypopharynx increased 63%, followed by the oropharynx with 54%, and the nasopharynx with a 15% increase (Fig. 8.17). In six of seven subjects, the minimal cross-section area was found in the retropalatal airway, while for the others, it was in the nasopharynx. The average minimal cross-section area before stimulation was 100.5 mm^2 and after stimulation it was 139.2 mm^2 [25].

8.7 Summary

This chapter outlines how CBCT can be used to assess the airway for both diagnosis and treatment outcome assessment. A CBCT alone cannot provide a diagnosis for sleep apnea, but it has its uses, specially in monitoring and helping with treatment considerations. A lateral cephalogram should not be used to assess the airway, since it does not portray mediolateral information or changes.

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Craniofacial Morphology Related to Obstructive Sleep Apnea: Growth of Craniofacial Bones and the Upper Airway

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Contents

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9.1 Upper Airway Development with Normal Craniofacial Growth

Upper airway, which comprises the nasal cavity, pharynx, and larynx, is more relevant to craniofacial structural environment than the lower airway. The pharynx is a tube-shaped structure that extends from the cranial base to the level of the inferior surface of the sixth cervical vertebra [1]. It lies dorsal to the nasal and mouth cavity and is cranial to the esophagus, larynx, and trachea. The pharynx can be anatomically separated into three parts: the nasopharynx, oropharynx, and hypopharynx. In a midsagittal image, the nasopharynx is shown to extend from the nasal turbinates to the hard palate. The oropharynx can be subdivided into the retropalatal pharynx (from the hard palate to the caudal margin of the soft palate) and the retroglossal pharynx (from the caudal margin of the soft palate to the base of the epiglottis). The hypopharynx spans from the base of the epiglottis to the larynx (Fig. 9.1) [2].

As upper airway is located below the skull base and behind the face, the growth and developmental changes of craniofacial structures will affect the development of the upper airway, and subsequently the dimension and function of the upper airway. It is necessary to understand the normal growth pattern of the upper airway and its contents in healthy children, which varies from year to year, to assess the significant variations from the normal.

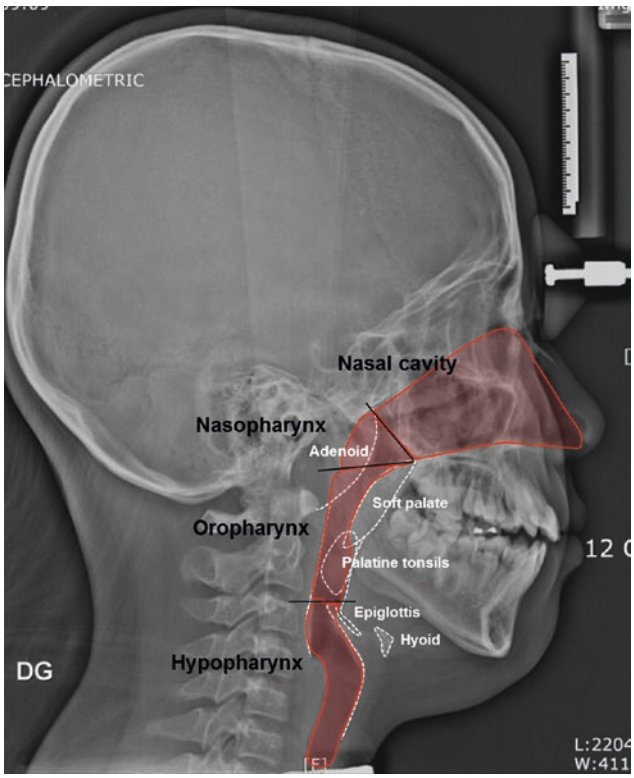


Fig. 9.1 Upper airway on the lateral cephalogram comprising nasal cavity, nasopharynx, oropharynx, and hypopharynx

9.1.1 Postnatal Growth of the Cranial Base

It is important to understand the forces within the cranial base that drive facial growth and upper airway development. The cranial base provides the platform around which the nasomaxillary complex and mandible develop, both of which influence craniofacial morphology and function.

Although the sutural growth on the cranium and cranial base accounts for multidirectional expansion of the cranial base, overall postnatal growth of the cranial base depends on endochondral growth on the synchondrosis, differential sutural growth of the calvaria wall, and surface cortical drift on the endocranial floor in response to the growth of cerebral lobes and sinuses. Among these mechanisms, endochondral growth of two principal synchondroses directly determines growth of the cranial base after birth. The sphenoethmoidal synchondrosis is most active in relation to growth of the anterior cranial base through approximately 7–8 years of age. The spheno-occipital synchondrosis, which fuses shortly after puberty (16–17 years in females and 18–19 years in males), is most prominent throughout the period of active craniofacial growth. Once synostosis occurs, growth of the cranial base length in the anteroposterior direction has mostly completed, and subsequent changes in the form of the cranial base may be attributable to bone remodeling. The cranial base undergoes a dramatic shift in its growth pattern during the first 2–3 postnatal years, and growth changes, thereafter, are smaller and steadier. Both cranial base lengthening and cranial base flexion are important growth mechanisms.

9.1.1.1 Cranial Base Lengthening

Up to the end of the first year of life, the intrasphenoidal synchondrosis defines the junction of the anterior and posterior cranial bases. The anterior cranial base grows primarily due to the growth of sphenoethmoidal synchondrosis by 6 years in concert with the frontal lobes of the brain and continues to increase after its fusion at 7–8 years of age. This is due to bony apposition on the outer surface of the frontal bone associated with the development of frontal sinus. The posterior cranial base lengthens primarily due to growth at the spheno-occipital synchondrosis, and it represents differential maturation from the anterior cranial base. The anterior cranial base grows more and is also more mature than the posterior cranial base throughout the postnatal growth between birth and 17 years of age. According to longitu-

dinal analyses, the anterior cranial base has attained nearly 90% of its adult size by 4.5 years of age, while the posterior cranial base has attained only about 80% of its adult size [3]. As a consequence, anteroposterior growth of cranial base length is almost complete during the first 6 years of life. Thereafter, any additional lengthening occurs by bony apposition, affecting the forward displacement of nasomaxillary complex.

9.1.1.2 Cranial Base Flexion

In newborns and infants, the cranial base is quite flat. With growth into childhood, a more convex superior or flexed appearance emerges. The cranial base angulation decreases more than twice as much during the first 2 years than between 2 and 17 years of age, primarily due to the differential growth of spheno-occipital synchondrosis. Between 2 and 6 years of age, cranial base flexion occurs because of bone remodeling that results in the clockwise rotation of the sphenoid bone and counterclockwise rotation of the occipital bone, which is accompanied by shortening and widening of the cranial base. The degree of bony rotation and its direction tends to be determined during the first 6 years of life when facial dynamics begin to compete with cranial dynamics.

Individuals with impaired cranial base flexion tend to keep their cranial bases narrow and long (a dolichocephalic pattern), and accordingly have narrow and long faces (Fig. 9.2). A combination of a counterclockwise sphenoidal and a clockwise occipital rotation may develop maxillary protrusion with deep and narrow maxillary arch and locate mandibular condyles backward, representing skeletal Class II. In contrast, individuals with large cranial base flexion by combined clockwise sphenoidal and counterclockwise occipital rotation tend to exhibit wide and short cranial bases (a brachycephalic pattern) with deficient midface and anteriorly located mandibular condyles, representing skeletal Class III.

On the other hand, a longitudinal study from Burlington Growth Centre with the annually examined Caucasian sample [4] found no significant differences in cranial length, cranial width, cephalic index, and anterior cranial base length between the 10% of the children with the most open cranial base angles and the 10% with the most closed cranial base angles. Children with the flattest cranial bases had a slightly shorter posterior cranial base, mandibular condyles located further backward and upward, and retrognathic maxilla, showing

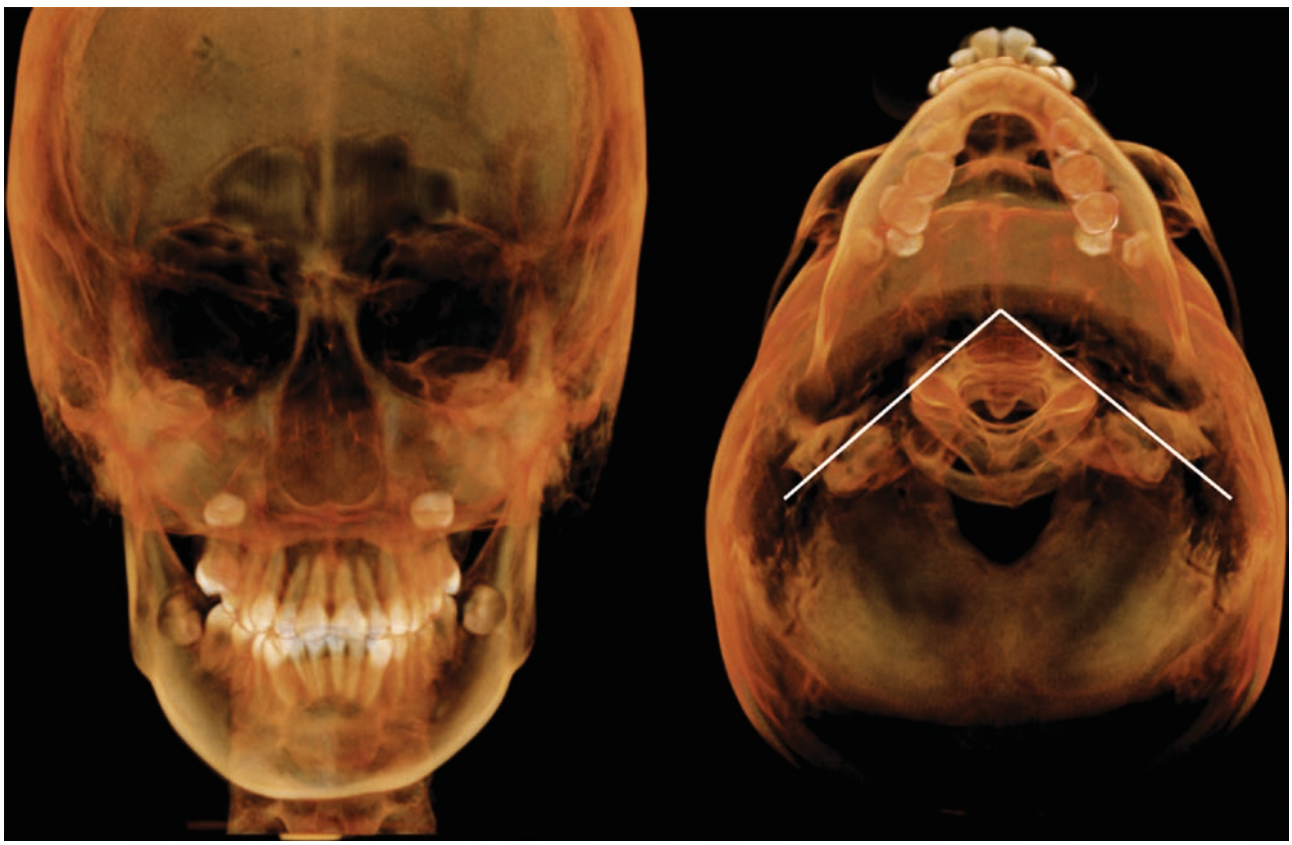


Fig. 9.2 CBCT volume images of an individual with impaired cranial base flexion, showing a dolichocephalic pattern and long face

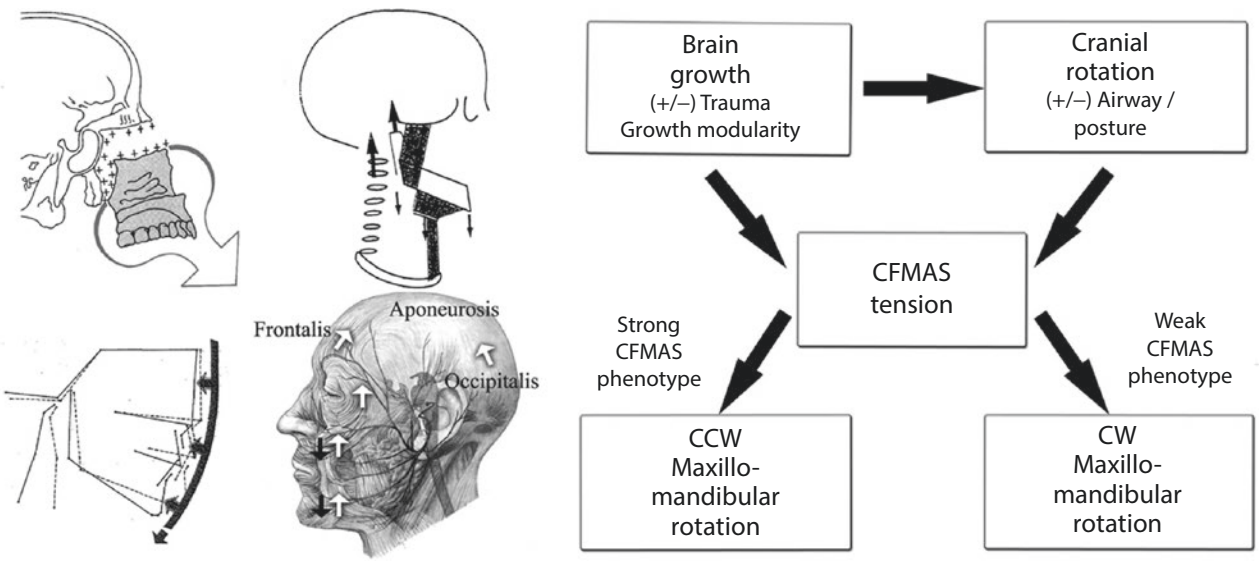
strong tendency to Class II relationships, which is in agreement with Bjork [5] and Enlow and McNamara [6]. In this population, large cranial base angles might be caused by a clockwise rotation of sphenoid bone and a much greater clockwise rotation of occipital bone. In contrast, none of the children with the smallest cranial base angles had Class III occlusions and most were Class I, even though their condyles were located more forward and downward (inconsistent with the findings of Enlow [7] and Lavelle [8]). A likely explanation to explain this is that their small cranial base angles might be formed mostly by greater counterclockwise rotation of occipital bone with less clockwise rotation of sphenoid bone.

Likewise, it should be noted that the individual bony units of the cranial base can be remodeled or rotated independently of each other, resulting in different mechanisms of cranial base flexion and different types of craniofacial morphology. This morphometric diversity is controlled primarily by genetics, but also affected by environmental factors, such as head posture and respiratory function, before 6 years of age.

9.1.1.3 Growth of Cranial Base Affecting the Upper Airway

Under the genetic influence, the growth of brain, skull, and cranial base may impact the size of upper airway by defining the relationship between the maxilla and the mandible. Both cranial base lengthening and flexion are essential for the forward and downward displacement of the nasomaxillary complex determining maxillary depth and height, additionally contributing to the development of maxillary width. Volumetric increment of nasomaxillary complex with forward and downward displacement will increase the dimension of upper airway, at least maintaining the relative airway space and passage in spite of the growth changes of parapharyngeal soft tissues, as will proper positioning of the mandible (mostly determined by the early growth of cranial base).

According to the aponeurotic tension model of craniofacial growth, brain growth and cranial rotation create cephalad tension within the interrelated craniofacial musculoaponeurotic system (CFMAS) of head and face, adding clarity to the mechanism of maxillomandibular rotation, temporomandibular joint development and positioning, and airway development (◘ Fig. 9.3) [9]. Rotation of the cranium at the occipital condyles and asymmetric growth at the Sphenooccipital synchondrosis (SOS) are proposed to cause the face to rotate counterclockwise, opening the airway with normal jaw rotation and extension of tissues, such as lingual tonsils and velum. Transverse development of the maxilla and nasal cavity, with greater posterior relative to anterior increase, is believed to be a result of muscle mass increases leading to greater CFMAS tension, thereby increasing the transverse airway dimension and providing room for the tongue.



◘ **Fig. 9.3** Algorithmic adaptation of the proposed CFG model. Displayed is brain growth modulation of cranial rotation and CFMAS tension. Brain growth can display temporal regional growth and mylenization with normal development and as a result of trauma (e.g., concussion, drug use). Cranial rotation modulates CFMAS tension and itself is influenced by brain development and postural control. CFMAS tension manifests as a strong or weak phenotype. A strong CFMAS phenotype will be expected to develop a *counterclockwise/forward maxillomandibular rotation*, while a weak CFMAS phenotype will be expected to develop a *clockwise/backward maxillomandibular rotation pattern*. (Standerwick and Roberts [9])

9.1.2 Postnatal Growth of Nasomaxillary Complex

9.1.2.1 Growth Mechanisms of Nasomaxillary Complex

The nasomaxillary complex (NMC), or midface, includes the circummaxillary suture system connecting it to the neurocranium and intermaxillary suture system, which are comprised of the midpalatal, transpalatal, intermaxillary, and internasal sutures. Postnatal development of NMC occurs principally by intramembranous ossification on the circummaxillary and intermaxillary sutures, although the nasal septum contributes to forward and downward displacement of the midface by cartilaginous bone growth, especially during the first 3–4 years [10]. The compensatory sutural growth in response to midface displacement by the growth of surrounding soft tissues leads to the majority of vertical, sagittal, and transverse changes of midface during both childhood and adolescence by lengthening of the anterior cranial base during the first 6 years of age. In addition, extensive surface modeling takes place over the entire NMC along its posterior and superior aspects.

9.1.2.2 Differential Growth of the Nasomaxillary Complex

The midface undergoes differential growth in its width, length, and height. The midface increases the most in height, followed by increases in depth, and the least in width. Inversely, the growth of midface ends earliest in width, next in depth, and the latest in height.

The growth in width, which is mostly associated with the expansion of brain and eyes separating the intermaxillary sutures between the two halves of nasal bones, maxillae, and palatine bones, essentially diminishes at about 7 years of age when the central nervous system completes growth. Thereafter, maxillary basal width slowly increases from 7 to 16 years of age by growth of the midpalatal suture, as well as the buccal surface bone apposition and expanding growth of the maxillary alveolar process. The growth activity on the midpalatal and transpalatal sutures drops down at around 15–16 years of age [11], although the biological fusion age of those sutures varies among studies [12–14].

In the meantime, the midface increases more dramatically in depth and height since the typical forward and downward growth of the midface relative to the anterior cranial base continues to be significant, with substantially greater vertical growth potential compared to the anteroposterior cranial base (Fig. 9.4). As the midface is displaced forward and downward, compensatory bony deposition occurs along the posterior surface of the maxillary tuberosity increasing the maxillary length despite resorptive surface bone modeling on the anterior surface of maxilla. Bony resorption is associated with inferior drifts of the anterior nasal spine and the inferior–posterior drift of A-point. Resorption occurs along the floor of nasal cavity with apposition on the roof of palate, which increases vertical displacement of NMC and increases nasal cavity volume. Although vertical maxillary growth rates peak during adolescence at the same time as stature, anteroposterior maxillary growth remains more or less constant with no distinct adolescent spurt.

9.1.2.3 Growth of the Nasomaxillary Complex Affecting the Upper Airway

Three-dimensional changes of the nasomaxillary complex show increases in nasal cavity volume and the forward and downward displacement of the posterior nasal spine on the hard palate. This increases the size of the bony nasopharynx and contributes to the developing nasopharyngeal and velopharyngeal airway, compensating the growth of adenoids and soft palate tissues and the nasopharyngeal walls (Fig. 9.5).

9.1.3 Postnatal Growth of Mandible

9.1.3.1 Growth Mechanisms of Mandible

The mandible has the greatest postnatal growth potential of any craniofacial complex structure. In contrast to the nasomaxillary complex, both endochondral and periosteal activities are significantly influential in mandibular growth, whereas displacement created by growth of the cranial base (moving the mandibular condyle) plays a negligible role. The overall growth pattern of the mandible can be represented as forward and downward translation of the chin in reference to the cranium (Fig. 9.6), while the body and the corpus of the mandible grow longer and wider. The actual growth occurs at the secondary cartilage on the mandibular condyle by endochondral bone formation and along the entire surface of the mandible by differential formation and resorption. At the same time, the mandible undergoes substantial true vertical rotation but limited transverse rotation. The typical pattern is forward rotation due to greater inferior displacement of the posterior rather than anterior aspects of the mandible, ultimately leading to a decreased mandibular plane angle.

9.1.3.2 Differential Growth of Mandible

The greatest changes in mandibular growth occur during the first few years to secure the pharyngeal airway in infancy and make room for the rapidly developing dentition. Corpus length is the most consistently mature region (closely approximating maturity of midfacial height), followed by overall length, and ramus height is least mature [16]. The greatest increases in mandibular length (from

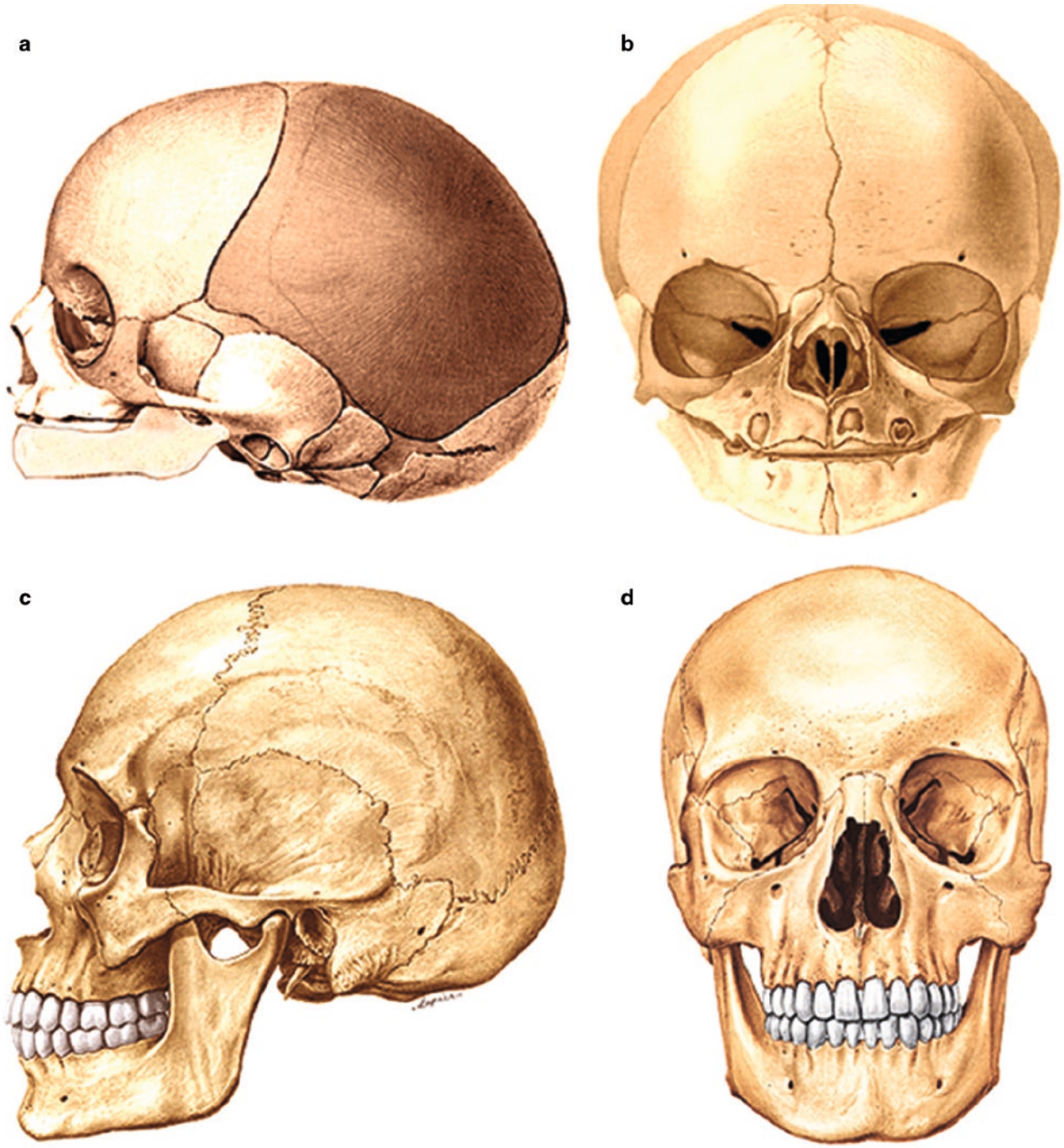


Fig. 9.4 Result of differential growth of nasomaxillary complex in width, length, and height from neonate to adult

condyle head to chin) occur between 4 and 17 years of life, followed by corpus length and ramus height. By the age of 5 years, ramus height grows to approximately 70% its adult size in males and 74% in females.

Like the maxilla, growth in mandibular width is typically complete prior to the spurt in adolescent growth. Increases in length and height continue throughout puberty. The condyle demonstrates the most growth superiorly compared to posteriorly during later childhood and adolescence. The juvenile growth spurt prior to the adolescent spurt is a key reason why careful evaluation of physiologic age is required to determine the timing of orthodontic intervention, particularly in females. Treatment planning should also consider differences between rapid-maturing girls and slow-maturing boys because of their differences in mandibular growth,

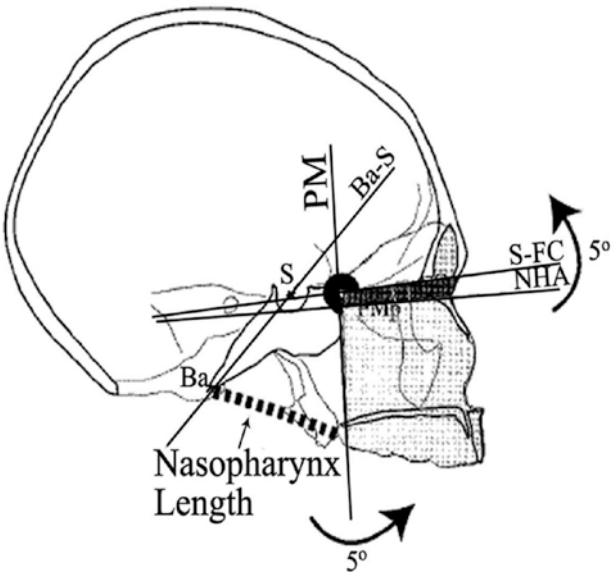


Fig. 9.5 The aponeurotic tension model explains the effects of angular invariance between the back of the face (behind the PM plane) and the top of the face, which is also the bottom of the anterior cranial base (S-FC). Changes in cranial base angle cause the face to rotate together around an imaginary axis through the PM point. Counterclockwise rotation of the midface increases the bony nasopharynx affecting the nasopharyngeal airway length. (Standerwick and Roberts [9])

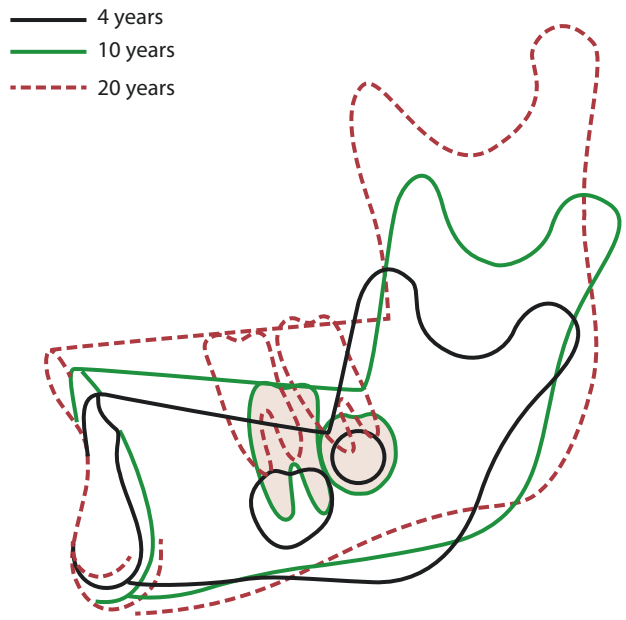


Fig. 9.6 Superimposition of implants for an individual with a normal pattern of growth, showing surface changes in the mandible from ages 4 to 20 years. As a variation between individuals, approximately a negative 10 to negative 15 degrees of internal rotation is compensated and concealed by external rotation with surface remodeling, as is expressed by 2 to negative 4 degrees change in the mandibular plane angle. (Bjork and Skieller [15])

particularly as they become most pronounced during adolescence.

There is more true mandibular rotation that occurs during childhood than adolescence compared to any time thereafter, particularly in the transition period to early mixed dentition at 6–7 years of age. Furthermore, the mandible rotates transversely because of its greater posterior expansion compared to the anterior aspects of the two corpii. Transverse rotation is also related to age. The mandible's posterior aspect expands between 65% and 70% as much as the posterior maxilla expands at the midpalatal suture. The growth of the mandible, therefore, closely follows the growth of the midface. As the midface is translated forward and downward with counterclockwise rotation, the mandible keeps pace in the normally growing face based on that the relative rate and amount of growth of maxilla and mandible differ over time.

9.1.3.3 Growth of Mandible Affecting the Upper Airway

The most critical craniofacial bony structures affecting the upper airway size are mandible and hyoid bone where the related muscles and soft tissues are attached. Along with forward and downward translation of the mandible, normal mandibular rotation is expected to direct the tongue forward through muscle attachment at the genial tubercles (internal surface of the mandibular symphysis) balancing the position of hyoid bone and its related muscle groups, to open the oropharyngeal and hypopharyngeal airway spaces.

9.1.4 Postnatal Growth and Positional Changes of Hyoid Bone

The hyoid bone is located between the mandibular symphysis and the larynx in the front of the neck, joining together the cranial base, tongue, mandible, sternum, scapula, thyroid cartilage, and pharynx [17]. The hyoid bone is suspended in the soft tissues without bony articulation [18]. Hyoid bone position is determined by the combined activity of suprahyoid, infrahyoid, and pharyngeal dilator muscles, as well as changes of head posture, body position, and oral functions. The hyoid maintains the positional balance of the upper airway patency and upright head position in association with tongue activity [18].

9.1.4.1 Growth of Hyoid Bone

The hyoid bone begins to ossify at the end of the third trimester and may never fully fuse until late in life [19]. The growth trend of the different components of the hyoid bone was similar during the first few years of life, while other bony structures seem to have a second phase

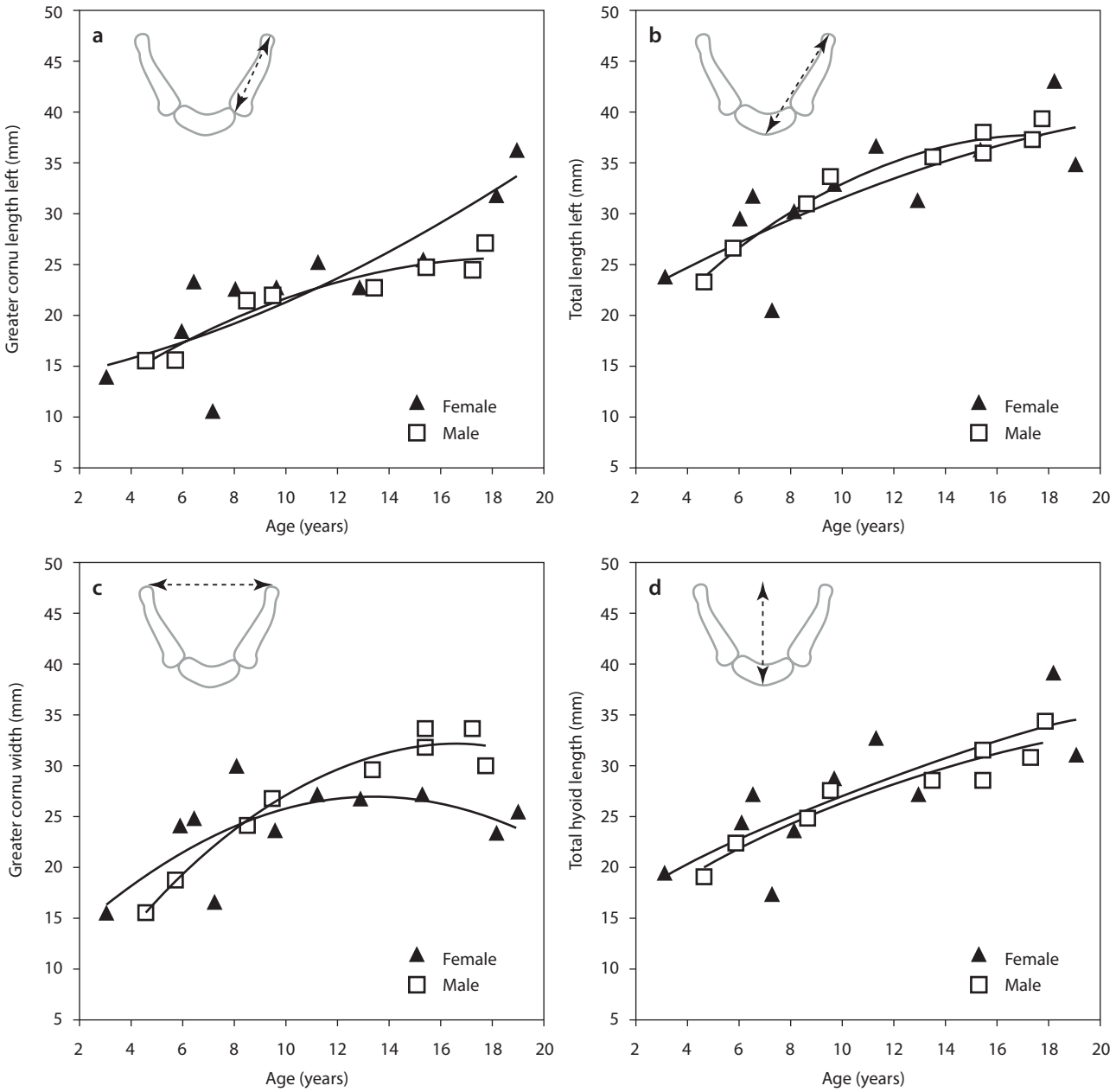


Fig. 9.7 Developmental growth pilot data from ages 2 to 20 from in vivo CT scans. Despite variability in the growth trend of the different measurements, all variables show a rapid period of growth during the first few years of life with discrete differences in growth after 6–8 years of age, along with the known growth differences between sexes. (Cotter et al. [20])

of rapid growth during puberty. Cotter et al. [20] showed a rapid period of growth during the first few years of life, with discrete differences in growth of different portions of the hyoid bone after about age 6–8 years showing gender-specific differences as well (Fig. 9.7).

9.1.4.2 Positional Changes of Hyoid Bone in Relation to Mandible, Tongue, and Upper Airway

The hyoid bone descends and moves slightly forward up to age 18 years [21], although the relative position of hyoid bone is constant within the individual through growth periods. Under normal conditions, forward translation of the hyoid is likely associated with the forward displacement of mandible, while the descent of the hyoid is reported to occur mostly by vertical growth of the cervical vertebrae [22]. According to Cotter et al.'s longitudinal study [20], the hyoid bone descended at a faster rate than the lowest point of mandibular symphysis, which closely parallels the behavior of the fourth cervical vertebra in growth increment between ages 2 and 7, and thereby, the relative position of the hyoid was maintained between the third and the fourth cervical vertebrae afterward despite its size increase (Fig. 9.8).

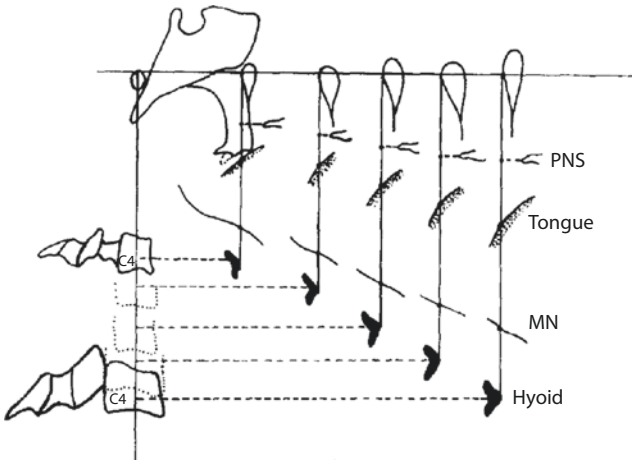


Fig. 9.8 Vertical growth of fourth cervical vertebra in various age groups was compared with descent of hyoid bone, mandibular border, tongue, and posterior nasal spine. Hyoid bone remained relatively stable to fourth cervical vertebra but moved away from border of mandible. However, all areas displayed consistent vertical increase. (Bench et al. [25])

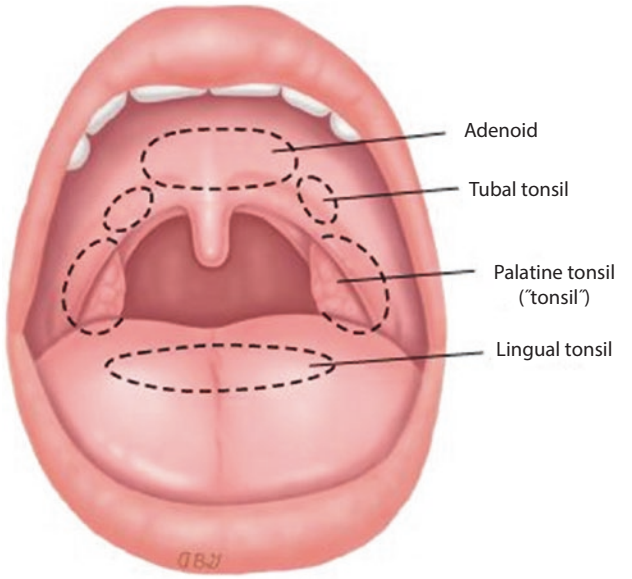


Fig. 9.9 Intraoral view of Waldeyer's ring comprising adenoids, lateral pharyngeal bands, palatine tonsils, and lingual tonsils

In other aspects, Sheng et al. [23] found that the vertical position of the hyoid bone had a strong relationship with ramus length. They deduced that the suprahyoid muscles play a vital role in relationships between the hyoid bone position and mandibular morphology or position. In addition, growth increment of tongue size relative to the oral cavity may cause downward movement of the tongue, leading to the downward and forward movement of the hyoid bone [24]. Accordingly, the hyoid bone controls the tongue functions when its position is normally related with the cervical column, and stable hyo-cervical relation is important to preserve the upper airway patency, especially the retroglossal airway.

9.1.5 Postnatal Growth of Pharyngeal Soft Tissues

The complex of lymphoid tissues encircling the pharynx is referred to as Waldeyer's ring, which includes (1) pharyngeal tonsils (adenoids), (2) lateral pharyngeal bands, (3) palatine tonsils, and (4) lingual tonsils (Fig. 9.9). The growth of adenoids and tonsils shows considerable variation in size among all age groups, suggesting that these tissues are more responsive to environmental stress than to the classical lymphoid growth curve published by Scammon et al. [26], which did not include measurements of the adenoids and tonsils [25].

9.1.5.1 Growth of Pharyngeal Tonsils (Adenoids)

In a 1954 cephalometric study, Subtelny [27] found that adenoid tissues follow a definite growth pattern without significant gender difference (Fig. 9.10).

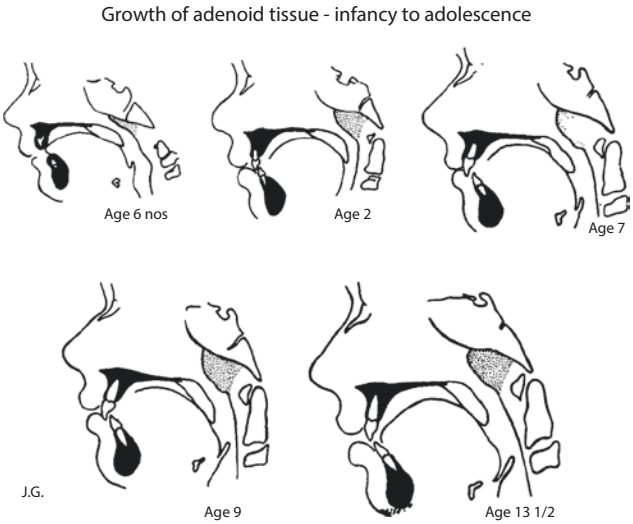


Fig. 9.10 Serial tracings of cephalometric head plates revealing changes in the adenoid tissue mass with age. The stippled area represents adenoid tissue as well as the soft tissue underlying the roof of the bony nasopharynx. (Subtelny [27])

Adenoids are not radiographically evident until 6 months to 1 year of postnatal life. The sloping nasopharyngeal roof looks straight or concave in configuration as it faces the superior surface of the soft palate. The contour is replaced by convex prominence by rapid growth of adenoids by 2–3 years of age, occupying one-half of the nasopharyngeal cavity. At this stage, the growth of adenoids occurs predominantly downward and forward possibly affected by the growth of upper face. Thereafter, the adenoids continue to grow dominantly in a downward direction, but at a retarded rate until their greatest bulk are attained as

early as 10–11 years or as late as 14–15 years of age. After the peak of adenoids growth, reverse growth occurs with progressive decrease in size, and usually, they are atrophied completely in adults.

9.1.5.2 Growth Changes of Palatine and Lingual Tonsils

Arens et al. reported that tonsil size increases linearly in the first decade of life and achieved the maximum size between 7 and 10 years of age [28]. The palatine tonsils may continue growth until 15 years of age, then tend to involute [29]. Currently, however, Oztürk [30] assessed the palatine tonsil size using transcervical ultrasonography in 680 healthy children ranging from 1 to 17 years of age, and proposed a positive correlation between tonsil size and age in the pediatric population. There is consensus that objective tonsil size shows wide variation that is influenced by both body mass index (degree of obesity) and age [31].

The lingual tonsils, located in the dorsal surface at the base of the tongue behind the foramen cecum and the terminal sulcus, develop later than other lymphoid tissues in the pharynx and persist into adulthood. Enlarged lingual tonsils have been identified as a significant cause of persistent obstructive sleep apnea (OSA), based on the report suggesting that obese children exhibit a higher prevalence of enlarged lingual tonsils [32].

9.1.5.3 Tongue Growth

The tongue in newborns is large relative to its small oral cavity. At birth, the entire tongue is positioned forward and lies in the oral cavity. As the oral cavity enlarges in concert with mandibular growth and the larynx descends over the first 5 years of life, the base of the tongue eventually moves backward to sit in the oropharynx. After this descent, the base of the tongue becomes the anterior wall of oropharynx. The tongue continues to increase in size during deciduous and mixed dentition, keeping pace with the dropping of the hyoid. The tongue's overall size doubles from birth to adolescence in thickness, length, and width. Its position is maintained or slightly dropped during 3–12 years of age [25].

The volume of the tongue and the suprahyoid muscle groups influence the anterior growth of the symphysis. During the first 2 years of life, the tongue changes to a rounder shape which may modify the position of the suprahyoid muscle insertions at the lingual side of the mental region by pushing them downward. Thus, the mandibular symphysis inclines posteriorly, which increases the prominence of the chin, and the hyoid bone is relocated forward and downward relative to the inferior border of the symphysis [33].

9.1.6 Postnatal Development of Pharyngeal Airway

A newborn's pharynx is approximately one-third the size of an adult [34] and continuously enlarges to adult dimensions at 6 years of age [35]. Compared to adults, the nasopharynx and oropharynx in children include more lymphoid tissues extending into the airway [36].

9.1.6.1 Developmental Change of Nasopharynx

The size of the soft-tissue nasopharyngeal wall and the adenoids relative to the size of the bony nasopharynx determines the dimension of the nasopharyngeal airway. Jeans et al. [36] evaluated the longitudinal changes in growth of the nasopharyngeal soft tissue, the area of the nasopharynx, and the nasopharyngeal airway from 3 to 19 years of age in 41 normal children using lateral cephalometry. The area of the nasopharyngeal bony framework steadily increased during this period, but the nasopharyngeal soft tissue (adenoids) grew from 3 to 5 years of age, but then remained unchanged until 19 years of age. Accordingly, the nasopharyngeal airway dimensions decrease following this same pattern. However, the current consensus is that overall change of nasopharyngeal airway dimension during growth is not significant since the growth change of the bony nasopharynx affected by craniofacial growth may cancel out the age-dependent difference of the soft tissues. The forward and downward displacement of the posterior nasal spine (PNS) of the hard palate with the descent of nasal cavity floor will move the soft palate forward (decrease of angle of soft palate inclination to the palatal plane) to compensate the pharyngeal airway dimension. Moreover, the age-dependent growth changes cannot exceed the overwhelming interindividual variation [37].

9.1.6.2 Developmental Change of Oropharynx

During the development of upright body posture in the first few years after birth, the oropharynx and the hypopharynx are at the crossroads between the forward positioning of the cervical vertebrae alongside the foramen magnum under the influence of the cranial base flexion. Hence, during early postnatal growth, the oropharyngeal airway may not increase substantially because the mandible, the tongue, and the suprahyoid muscles are tightly packed superiorly by the ethmomaxillary complex and posteriorly by the cervical column.

The Bolton-Brush Growth Study [38] examined 32 healthy children to investigate the pattern of skeletal and soft-tissue growth of the oropharynx. Lateral ceph-

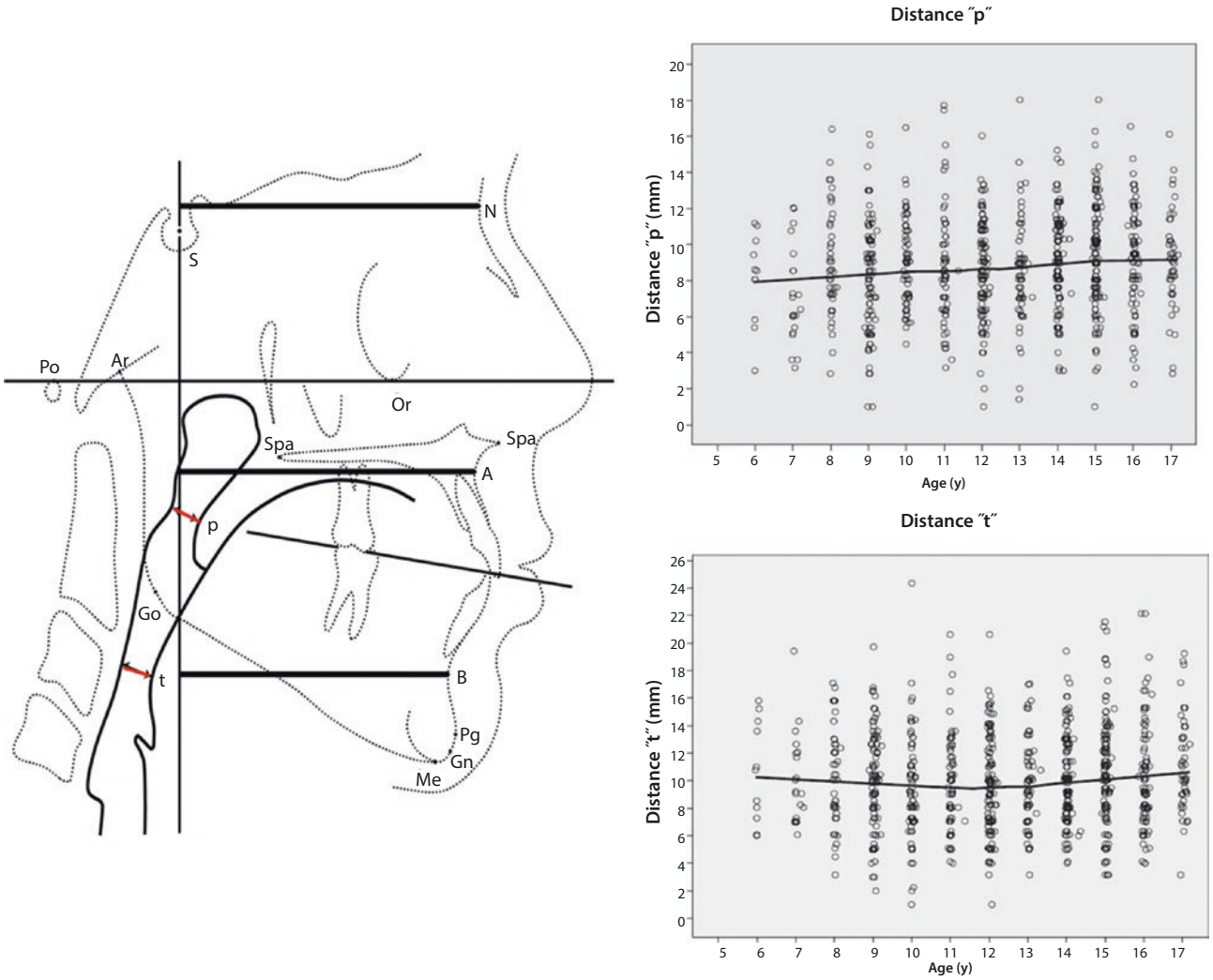


Fig. 9.11 Graphical distribution of airway distance (p) and (t) corresponding to the age of the subjects ($n = 880$) and Loess interpolation line. The small yet statistically significant age-related increase was found, but a large interindividual distribution was apparent. (Mislik et al. [39])

alometric analysis of untreated normal subjects at 6, 9, 12, 15, and 18 years of age showed that pharyngeal soft tissues exhibited two periods of accelerated growth periods (ages 6–9 years and 12–15 years) and two periods of quiescence (ages 9–12 years and 15–18 years). The increase in oropharyngeal width is thought to occur due to the continued growth of the pharyngeal region and natural involution of palatine tonsils. On the other hand, Arens et al. [28] used MRI to assess linear dimensions of the upper airway structures in 92 normal children from 1 to 11 years of age and found that the soft tissues in the oropharyngeal airway (i.e., the tongue, soft palate, and tonsils) maintained growth proportional to the craniofacial skeleton growth. Any deviations in the linearity of these relationships represent critical risk factors for Obstructive sleep apnea (OSA) in childhood.

Mislik et al. [39] reported on a large sample size of 880 healthy Caucasian children (Fig. 9.11), finding that the dimensions of the pharyngeal airway are established in early childhood and remain stable from 6 to 17 years of age. Mean retropalatal width (p) increased continuously from age 6 (8.12 mm) to age 17 (9.15 mm), which is probably related to the size decline of the adenoids during this growth period [40]. Mean retroglossal width (t) decreased from age 6 (10.61 mm) to age 10 (9.31 mm), but increased thereafter up to age 17 (11.19 mm), a finding similar to that of prior research [41–43]. The initial decrease of "t" might be attributed to the distinct growth of the tongue resembling a neural growth pattern. Thus, during the juvenile phase, tongue growth will be more intense compared to the growth of all its surrounding structures following visceral growth pattern.

Caution should be applied when interpreting the small yet statistically significant age-related increases, since a large interindividual distribution was apparent. No difference was found between the genders and no correlations of distances "p" and "t" with Point A-Nasion-Point B angle indicating the maxillomandibular relationship (ANB) in a population of individuals with normal growth.

9.2 Upper Airway Impairment with Abnormal Craniofacial Growth

Although still somewhat controversial, it makes sense that abnormal position of the jaws would influence the airway space. Craniofacial discrepancies developed during the growth period, such as a short cranial base, mandibular and/or maxillary retrognathism, a short mandibular body, backward and downward rotation of the mandible, and constricted maxillary width, may all result in the reduction of pharyngeal airway dimension and passage alone or combined [44]. Decreased space between the mandibular corpus and the cervical column may lead to changes in posterior posture of the tongue and soft palate, leading to not only respiratory dysfunction during the day but also nocturnal problems, such as snoring, upper airway resistance syndrome, and OSA [45].

Likewise, abnormal development of the upper airway is closely related to abnormal craniofacial growth as well as abnormal respiratory function. Considering that genetic and environmental factors have similar impact on craniofacial growth and dimension of the upper airway; however, it is not easy to discern if the impaired development of the upper airway, even when abnormal craniofacial development occurs, might be caused by heredity or by environmental functions resulting in abnormalities. When assessing craniofacial and pharyngeal development, differences in ethnicity and gender should be considered. Despite the controversies, it has been predominantly reported that there were no significant gender differences in pharyngeal dimensions influenced by abnormal craniofacial growth and development in children [46–49]. Besides, a subject's height [50] and body weight [51, 52] are other factors positively correlated with pharyngeal airway volume.

Moreover, it is impossible to clarify a causal relationship between craniofacial deformation and abnormal upper airway development. Nonetheless, it is important to know that further abnormal development of craniofacial structures by delayed growth modification treatment or myofunctional therapy would be a key factor to induce upper airway constriction and secondary development of sleep-disordered breathing (SDB).

9.2.1 Extended Head and Cervical Posture Affecting Craniofacial Deformation

It is fundamentally important to understand the contribution of postural mechanisms to normal or abnormal craniofacial development for the diagnosis and treatment of morphological and functional disorders of the stomatognathic system. Solow and Siersbaek-Nielsen's "soft-tissue stretching" hypothesis [53] links postural-induced stretching of the soft-tissue facial layer, craniofacial morphology, and airway adequacy into a cycle of factors related to craniofacial morphogenesis. Soft-tissue layer of facial skin and muscles passively stretches when the head is extended in relation to the cervical column, which increases the forces on skeletal structures, restricting forward growth of the maxilla and mandible and redirecting them caudally.

Two longitudinal studies found a clear pattern of associations between head and cervical posture with craniofacial growth determined by the structural superimposition method [54, 55]. Subjects with greater cervicohorizontal and smaller craniocervical angles have been associated with a horizontal facial growth pattern characterized by reduced backward displacement of the mandibular condyle, increased growth in length of the maxilla, increased maxillary and mandibular prognathism, and larger-than-average forward true rotation of the mandible. In contrast, subjects with lower cervicohorizontal and large craniocervical angles were associated with a vertical facial development and are likely to exhibit large backward displacement of the mandibular condyle, reduced growth in length of the maxilla, increased maxillary and mandibular retrognathism, and backward true rotation of the mandible, impinging the upper airway dimension. A recent systematic review by Gomes et al. [56] found that greater craniocervical and craniovertical angles were significantly associated with shorter maxillary and mandibular lengths and greater maxillary and mandibular retrognathism, and a skeletal Class II pattern (Fig. 9.12) [57]. In vertical aspects, greater craniocervical and craniovertical angles were associated with a large anterior face height and a small posterior face height, thereby presenting with a reduced face-to-height ratio. Greater craniocervical and craniovertical angles were also associated with a high mandibular plane angle and a high occlusal plane angle, as well as a hyperdivergent vertical pattern, portending increased risks of pharyngeal constriction and breathing problems. However, because correlation coefficients ranged from low to moderate, the authors cautioned careful interpretation of such associations [56]. Although posture appears to influence facial development, many other factors must also affect this mechanism.

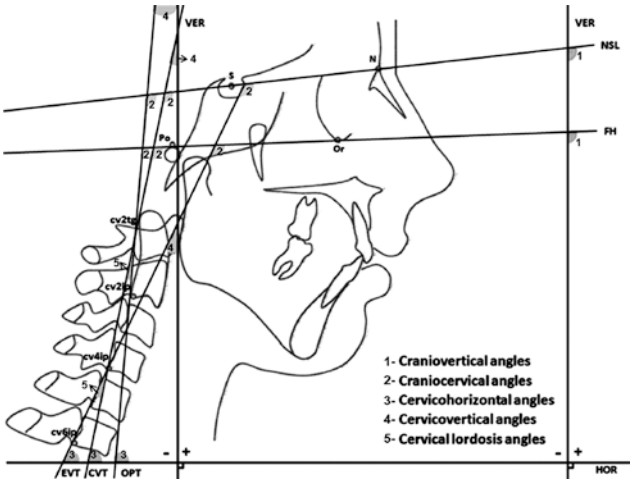


Fig. 9.12 Cephalometric postural variables. The convention employed for angles related to the true vertical was that downward opening angles formed behind the true vertical (VER) were taken as negative, whereas angles formed in front were positive. (Gomes et al. [56])

9.2.2 Pharyngeal Airway in Different Sagittal Craniofacial Discrepancy

Class II subjects with retrognathic mandible have been reported to have significantly smaller airway volume than Class I subjects [58, 59]. Relatively short and/or posteriorly placed mandibles with respect to the cranial base might force the tongue and soft palate back into the pharyngeal space, causing a reduction in oropharyngeal airway volume [60]. More specifically, oropharyngeal airway space has been reported to be more vulnerable to the sagittal skeletal pattern than nasopharyngeal airway space [47, 61, 62].

Zhong et al. [61] supported the notion that skeletal discrepancy may predispose to upper airway obstruction. They analyzed 370 healthy Chinese children (ages 11–16 years) and found that widths of both the oropharyngeal and hypopharyngeal airway were greater in Class III vs. Class I and Class II patients, though no difference was found at nasopharyngeal level. Uslu-Akcam [63] assessed the pharyngeal airway dimension of 124 children with skeletal Class II division 1 and division 2 patterns during the prepeak, peak, and postpeak growth periods for comparison with a skeletal Class I group. They reported that the nasopharyngeal and oropharyngeal airway spaces showed a significant difference among the groups throughout the growth periods, with the smallest dimensions in the skeletal Class II division 2 during the prepubertal period.

Alves et al. evaluated pharyngeal airway dimensions of the 50 healthy Caucasian children (mean age 9.16 years, range 8–10 years) with Class I or Class II skeletal patterns using Cone-beam computed tomography (CBCT). They reported that the Class II children with mandibular deficiency had less airway volume, less minimum axial area, and narrower retropalatal airway width than the Class I children. Zheng et al. [64] suggested the significance of the site of minimal cross-sectional area (Min-CSA) in the upper airway among different sagittal discrepancies (Fig. 9.13). They analyzed three-dimensional volume and the Min-CSA of the pharyngeal airway of 60 Japanese patients (between 14 and 18 years of age) with different sagittal skeletal patterns. Class II showed the smallest mean Min-CSA, which was usually in the upper oropharyngeal region, whereas Class III had the largest mean Min-CSA, which was generally found in the lower hypopharyngeal region. Normally, Class I subjects tend to have the Min-CSA in the lower oropharynx or upper hypopharynx.

Focusing on the oropharyngeal airway of Class III malocclusion, Iwasaki et al. [65] obtained three oropharyngeal airway shapes by cluster analysis from 45 Class sI (mean age of 8.8 years) and Class III (mean age of 8.4 years) Japanese children. They found that 84% of children with Class I had a square oropharyngeal airway, but 70% of the children with Class III had a relatively flat shape, either left–right (55% wide type) or anteroposterior (15% long type) (Fig. 9.14). The smallest cross-sectional area was observed in the square type. This result suggests that both volume and shape of oropharyngeal airway are affected by tongue posture, soft palate configuration, size of palatine tonsils, and anteroposterior position of the mandible.

A correlation analysis between craniofacial and pharyngeal measurements showed significant positive correlations of the Sella-Nasion-Point B (SNB; indicating anteroposterior position of mandible) angle, indicating anteroposterior position of mandible to the cranial base and the pharyngeal airway space [41]. Ceylan and Oktay [47] reported that oropharyngeal airway area decreases with the increase of the ANB angle from lateral cephalometric analysis. El and Palomo [62] observed that oropharyngeal airway volume measured on CBCT was significantly negatively correlated with ANB angle and positively correlated with the SNB angle. Hwang et al. [66] reported that a constricted nasopharyngeal airway is correlated with detruded mandible and maxilla. Furthermore, Trenouth and Timms [67] found that oropharyngeal airway width was positively correlated with the length of the mandible (between gonion and menton). This was confirmed by a three-dimensional CBCT analysis on the preadolescent Korean children by Kim et al. [68], who showed that mandibular body length had a significant positive correlation with total airway volume.

Cases of mandibulofacial dysostosis are characterized by bilateral and symmetrical underdevelopment of the midface and the mandible along with a hypoplastic, retro-positioned tongue, such as Treacher Collins syndrome and Pierre Robins sequence, which are all known

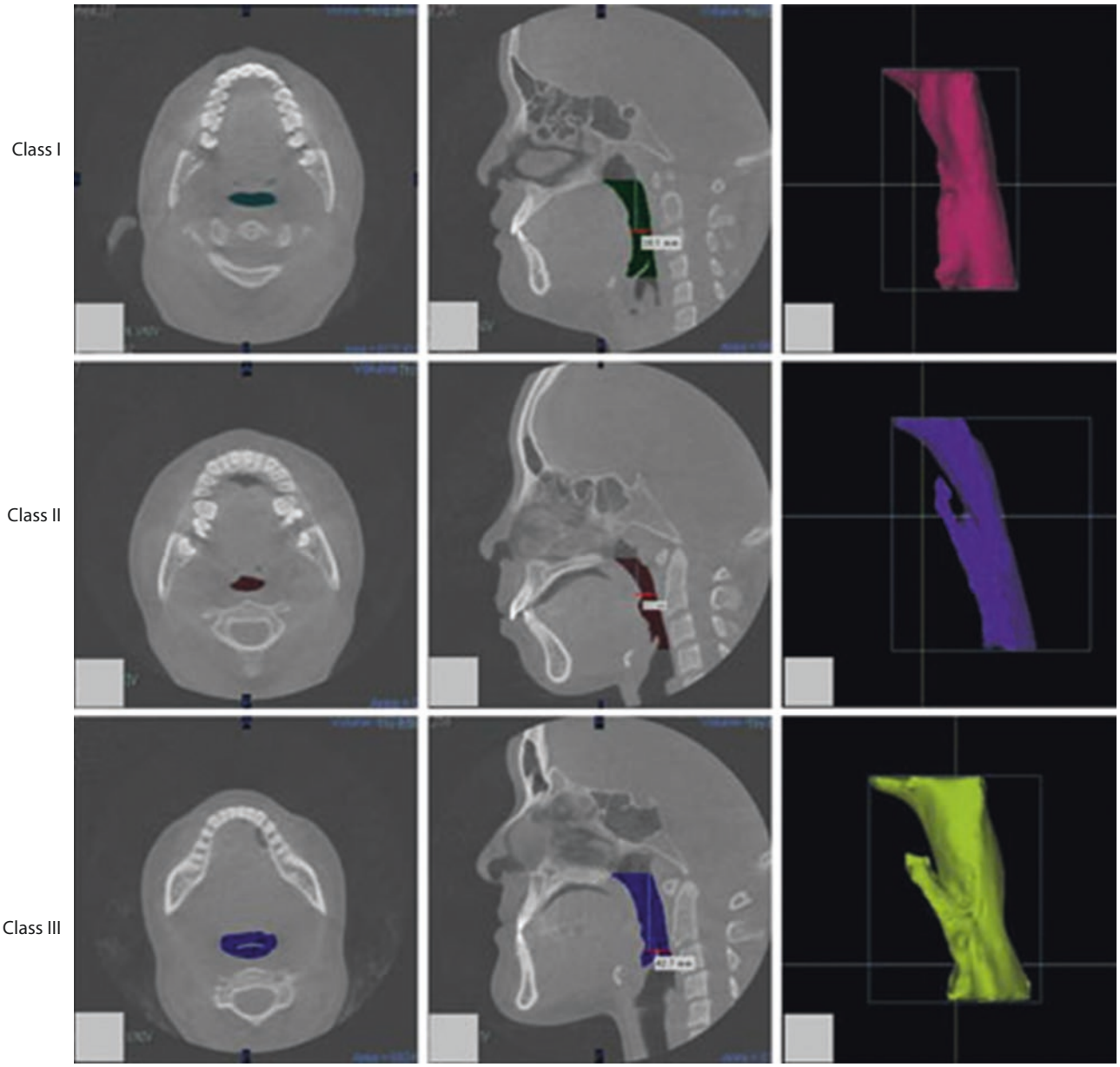


Fig. 9.13 The minimum cross-sectional area (Min-CSA) in patients with different skeletal patterns. Class II showed the smallest Min-CSA in both anteroposterior and transverse dimensions, but Class III showed the largest Min-CSA. (Zheng et al. [64])

causes of pediatric OSA [69]. Pierre Robin sequence includes mandibular hypoplasia, allowing the tongue to be posteriorly located and thereby impairing closure of the palatal shelves that must grow over the tongue to meet in the midline, resulting in U-shaped palatal cleft. The tongue's posterior displacement may impair the action of the genioglossus muscle, an important parapharyngeal dilator known to contribute to airway obstruction.

Studies have compared the pharyngeal airway form in addition to the volume among different skeletal patterns in relation to the craniocervical angle. Grauer et al. [70] assessed the pharyngeal airway form and volume in 62 Caucasian postadolescent subjects (aged between 17 and 46 years) with different craniofacial pattern. They elucidated that skeletal Class II patients had more forward inclination of the pharyngeal airway with smaller volume, whereas Class III patients had a more vertically oriented pharyngeal airway with larger volume (Fig. 9.15). Consistent with these data, Oh et al. [71] reported that the larger the craniocervical angle is, the larger the pharyngeal angle, and the more backward orientation of the oropharyngeal angle is evident (Fig. 9.16) based on the correlation analysis between craniofacial form and pharyngeal airway form in 60 healthy Korean children (mean age 11.79 years, range 10

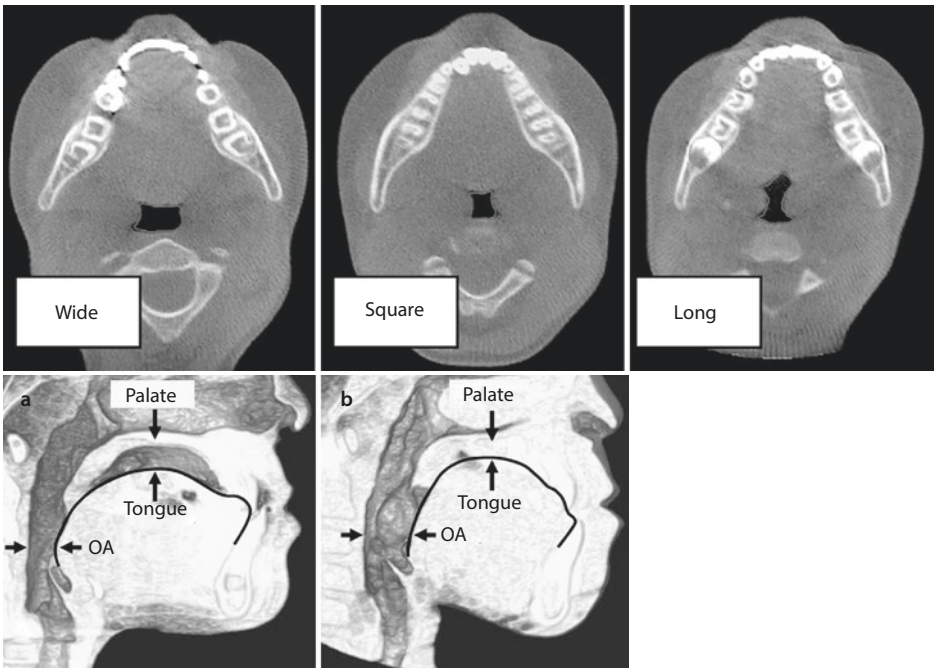


Fig. 9.14 Examples of three airway-shape types with tongue position against the palate and oropharyngeal airway: **a** the wide type has a flat shape and a lower position of the tongue, resulting in reduced oropharyngeal airway; **b** the long type has hypertrophic palatine tonsils. The tongue is positioned more anteriorly to maintain the oropharyngeal airway. (Iwasaki et al. [65])

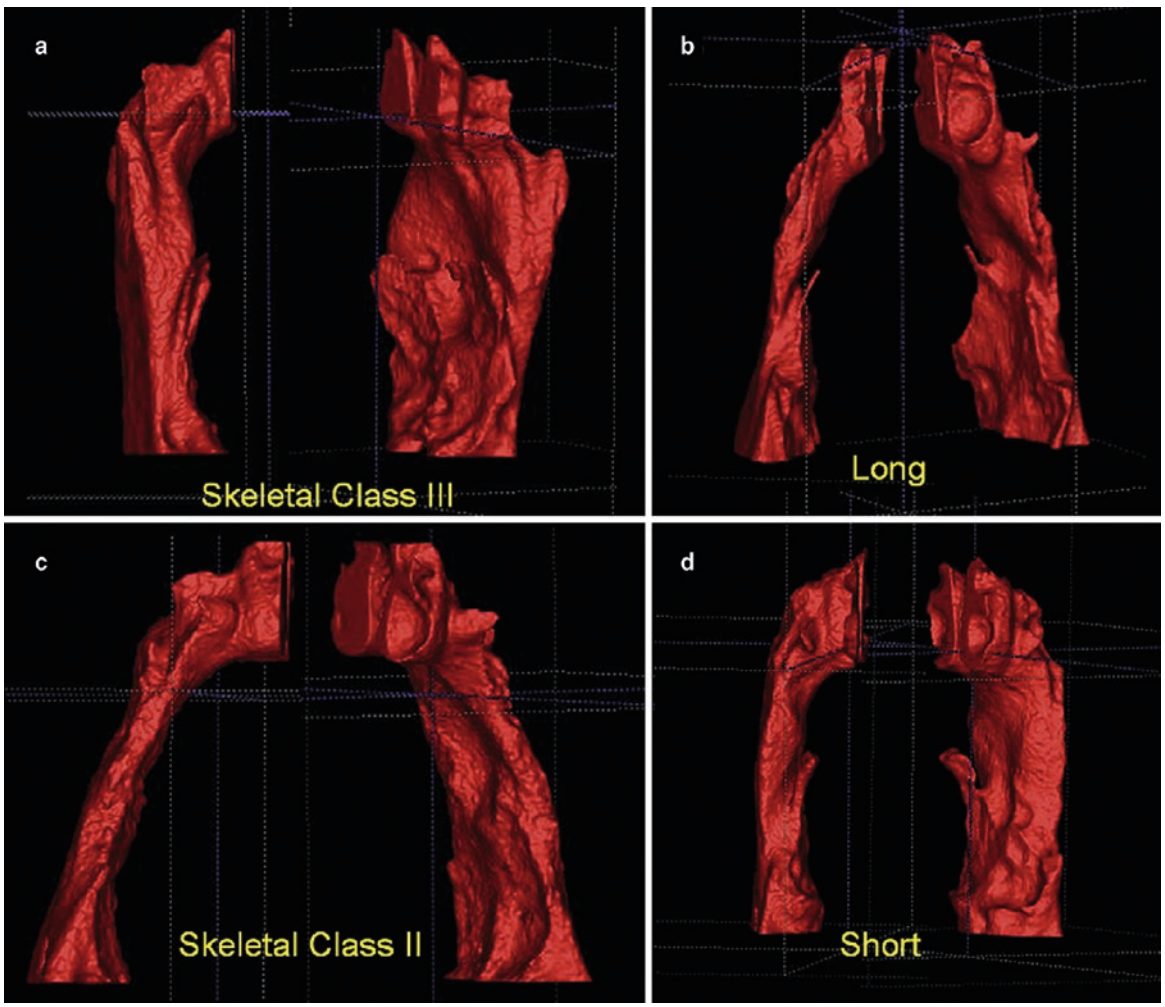


Fig. 9.15 Different airway shapes of skeletal Class II and Class III subjects, depicting a more vertical orientation of the airway in Class III subjects. **a**, **c** This finding was statistically significant. **b**, **d** Differences between subjects in the vertical groups are less apparent; differences not statistically significant. (Grauer et al. [70])

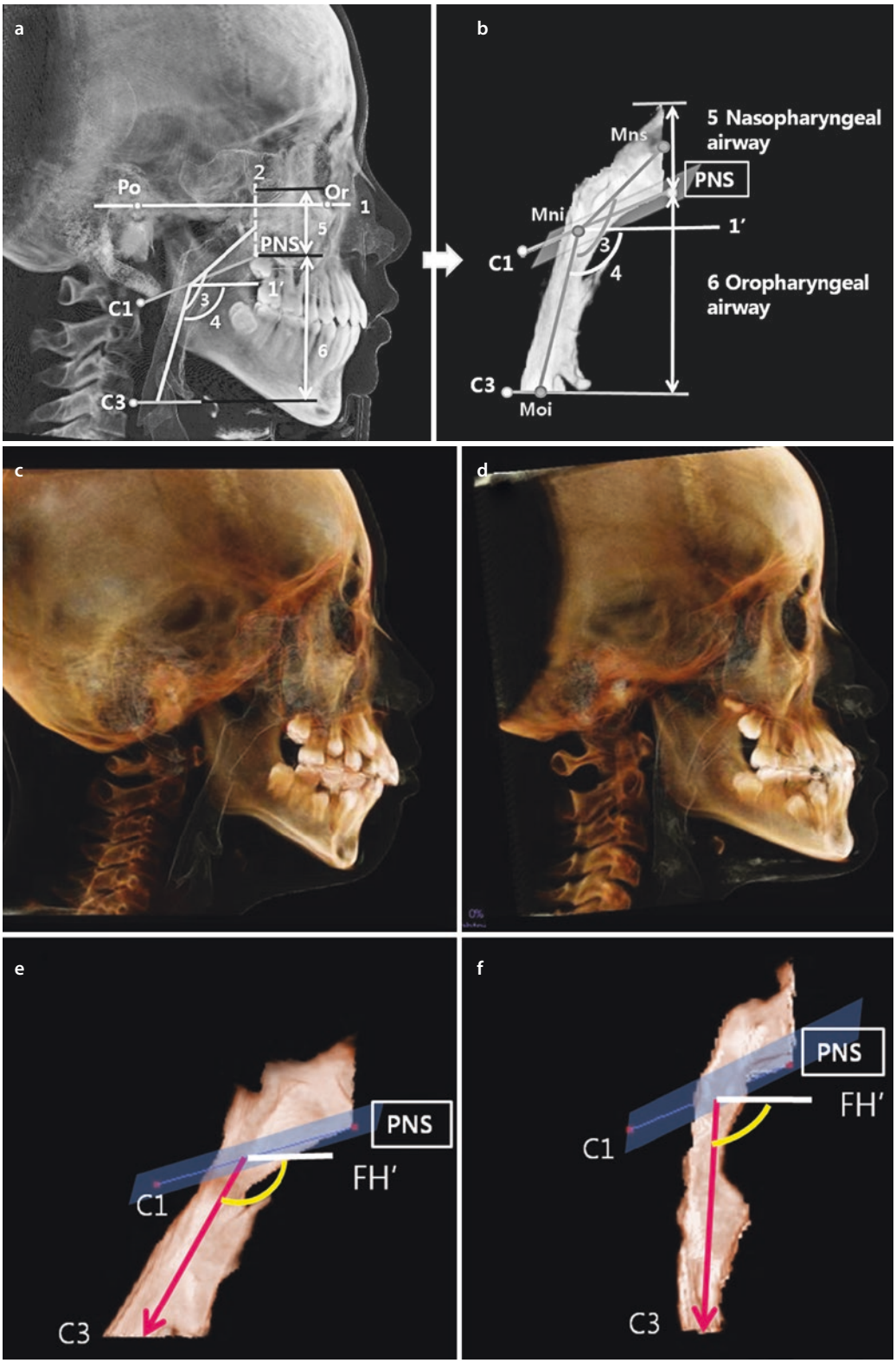


Fig. 9.16 Types of pharyngeal airway form according to anteroposterior facial patterns. **a**, **c** More backward orientation of the oropharyngeal airway to the FH plane in skeletal Class II group. **b**, **d** More vertical orientation of the oropharyngeal airway in skeletal Class III group. The anterior border of the nasopharyngeal airway, which is the coronal plane passing through the posterior nasal spine (PNS) and superior tip of the nasopharyngeal airway: (1) ang-PA, (2) ang-oropharyngeal airway, (3) Vol-NA, and (4) Vol-oropharyngeal airway. (Oh et al. [71])

and 13 years). The inclination of oropharyngeal airway to the FH plane in the sagittal plane (ang-oropharyngeal airway) was greater in Class II with more backward pharyngeal form than in Class III. The ang-oropharyngeal airway had significant correlation with the ANB angle and Pog-N perpendicular at around the puberty of growth.

Currently, however, upper airway dimensions differ in various sagittal skeletal patterns and remain controversial. Some studies found a weak relationship between growth pattern, craniofacial morphology, and pharyngeal airway [59, 72–74]. Allhaija et al. [46] reported that sagittal skeletal patterns were weakly correlated with glosso- and hypo-pharyngeal dimensions, but still statistically significant. De Freitas et al. [75] reported that the sagittal skeletal pattern does not influence retropalatal airway width; furthermore, both sagittal and the vertical growth patterns also do not influence retroglossal airway width.

A recent systematic review on the upper airway dimensions in different sagittal craniofacial patterns included only 11 of 758 identified studies in their final review [76]. Roughly 75% of studies did not report differences in the nasopharyngeal dimensions among craniofacial patterns. Because 5 of the 11 studies found these to be smaller in Class II subjects, and 6 of 11 studies concluded that oropharynx size is larger in Class III pattern, the oropharyngeal dimension findings are controversial. Furthermore, the vertical growth type of the subjects was not considered in five of the investigations, and 45% of the included studies used lateral cephalometry as their only tool for assessing airway dimensions. The clinical significance and reliability of the SNB angle and the ANB angle have been debated in the literature [77].

Although these are still widely used parameters to describe anteroposterior dentofacial discrepancies, it should be recognized that they have limitations influenced by many variables, such as morphology of the nasion area, the vertical dimensions of the face, and the inclination of the anterior cranial base. Therefore, three-dimensional evaluation incorporating the influence of sagittal, vertical, and transverse skeletal patterns simultaneously is recommended when interpreting the influence of developing skeletal discrepancy on the upper airway volume and shape.

9.2.3 Pharyngeal Airway in Different Vertical Craniofacial Discrepancy

Vertical craniofacial discrepancy might develop during pubertal growth because of several etiologic factors, which may include abnormal growth of the maxilla and mandible, dentoalveolar development, and function of the tongue [78]. According to Schudy [79] and Isaacson et al. [80], backward mandibular rotation and bite opening occur when vertical growth of condyles is less than that of the craniofacial sutures and alveolar process. It has been suggested that Class I subjects with vertical growth patterns may exhibit narrower airway passages than subjects with horizontal growth patterns.

Ucar et al. [81] conducted a lateral cephalometric analysis and identified significant differences between vertical skeletal patterns and airway dimensions in Class I subjects. When 31 low-angle subjects (mean age, 14.0 years), 40 high-angle subjects (mean age, 12.7 years), and 33 normal-angle subjects (mean age, 13.9 years) with Class I malocclusion were examined, significant differences were found between the low-angle and high-angle groups at the level of the nasopharyngeal airway space, the palatal tongue space, the upper posterior airway space (PAS), and tongue gap. Furthermore, the authors reported that the nasopharyngeal airway space and upper PAS decreased from low angle to normal to high angle, and the tongue gap distance was greater in high-angle subjects vs. normal- and low-angle subjects. These findings were confirmed by CBCT study comparing the pharyngeal airway volume among different vertical skeletal patterns of 100 healthy Turkish young adult patients (aged 18–30 years) with normal sagittal skeletal pattern [82]. Accordingly, total airway volume, as well as individual nasopharyngeal and oropharyngeal volumes, was lowest in the high-angle group (mean age 23.9 years) and highest in the low-angle group (mean age 24.3 years).

Park et al. [83] investigated morphometric growth changes of the nasopharyngeal space in association with the development of the adenoids in different vertical craniofacial features. The authors assessed a longitudinal sample of Caucasian children (4–13 years of age) using lateral cephalometric radiography (◘ Fig. 9.17). They reported that the hyperdivergent types started with greater nasopharyngeal airway areas than did the hypodivergent types, but this relationship reversed with time. Even though the distance from the most superior point of adenoid tissue to the posterior nasal spine (PNS) was consistently greater for the hyperdivergent types across all age groups (supposedly due to the forward and downward movement of the PNS), the nasopharyngeal airway area became smaller than that in the hypodivergent group after 8 years of age. It was posited that this was attributable to more pronounced adenoid enlargement that lasted longer in the hyperdivergent types. However, one limitation of this study was that conclusive evidence was lacking to establish a causal relationship.

On the contrary, Grauer et al. [70] showed in their study that there was no significant difference in pharyngeal airway volumes among long, normal, and short facial height groups. One limitation was the fact that vertical grouping was performed simply by dividing the sample by face height although each subject belonged to both a sagittal group and a vertical group. Many sub-

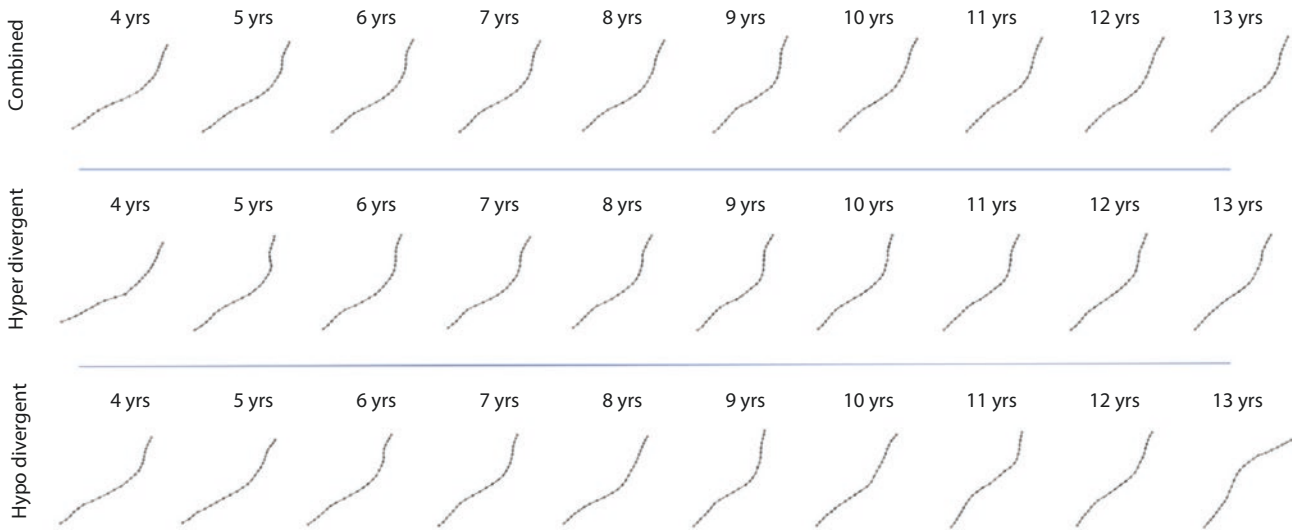


Fig. 9.17 Mean shapes of the adenoids according to chronologic age for the hyperdivergent and hypodivergent subjects are shown together and separately. (Park et al. [83])

jects with longer faces also were classified as skeletal Class II or Class III, whereas those with shorter faces tended to be classified as skeletal Class I. Bias from this source may be responsible for false differences in airway volumes between the vertical groups or to conceal the real differences.

9.2.4 Upper Airway in Transverse Craniofacial Discrepancy

Underdevelopment of the nasomaxillary complex in a transverse dimension (especially underdevelopment of that nasal cavity, maxilla, and hard palate) has not been clearly described as an etiologic factor of airflow reduction and OSA. Guilleminault et al. [84] studied the relationship between maxillary constriction and the etiology of OSA, suggesting a familial tendency of narrow high palates in the relatives of OSA patients. Cistulli and Sullivan [85] showed a high prevalence of OSA with elevated nasal airway resistance in patients with Marfan syndrome who have characteristic constricted maxilla and high-arched palate. Zhao et al. [86] reported that their CBCT analysis of oropharyngeal airway volume showed that it was significantly smaller in growing patients with maxillary constriction than in those without constriction. On the other hand, Johal et al. [87] investigated the role of maxilla in the etiology of OSA and found no significant maxillary morphological differences between OSA groups and normal subjects, except for the significant difference of the palatal angle (ANS-PNS-uvula).

It is not possible to determine from the literature whether transverse skeletal discrepancies are etiologic factors for airway constriction and pediatric sleep-disordered breathing. It remains questionable as to whether maxillary constriction can be a primary etiological factor or a resultant factor in OSA and, more importantly, whether or not treatment directed at maxillary expansion is supported by sufficient evidence.

In summary, the effect of anteroposterior or transverse skeletal relationships on the upper airway dimension is not completely understood. Not all children with a retruded mandible or constricted maxilla will have airway problems; however, the upper airway of the children with retruded jaws with a hyperdivergent growth pattern may need special attention and, potentially, early intervention.

9.3 Craniofacial Alteration by Abnormal Respiratory Function

While some researchers have found no association between airway function and dentofacial morphology [88–92], there seems to be consensus that healthy nasal breathing with adequate pharyngeal patency plays a role in the favorable development of the dentofacial complex. Any obstruction in nasal breathing will affect various fundamental orofacial functions during early development causing SDB and can be a risk factor of craniofacial deformation beyond the inherited skeletal pattern.

9.3.1 Prevalence of Craniofacial Deformation in SDB Children

Ameli et al. [93] orthodontically evaluated a cohort of 118 suspected pediatric OSA patients prior to prearranged adenotonsillectomy and found that 65% of them

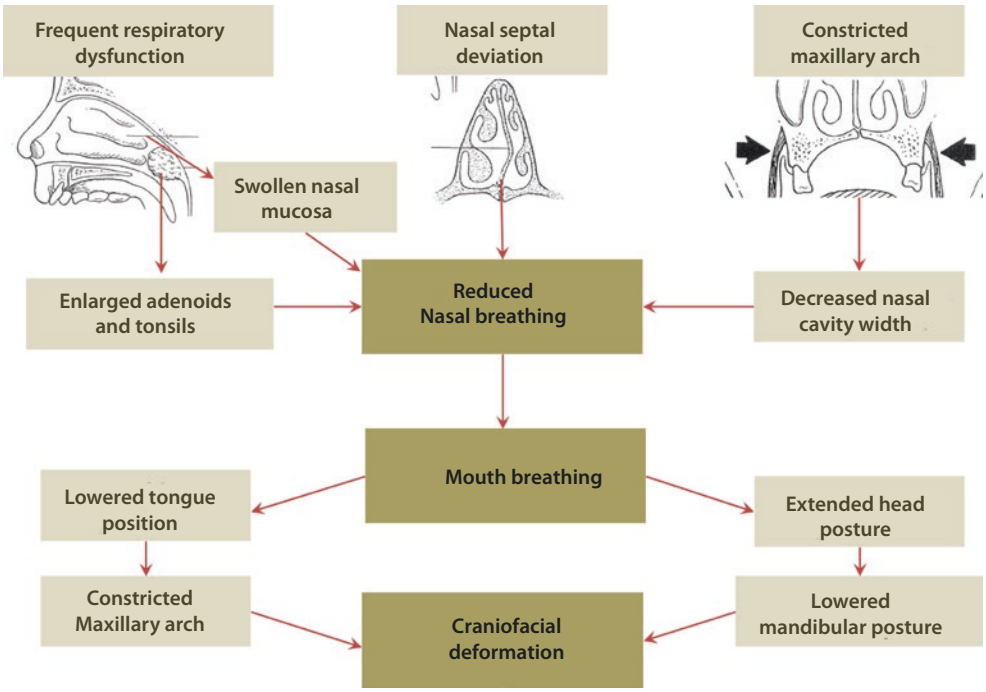


Fig. 9.18 Influence of sleep-disordered breathing on orofacial growth. (Guilleminault et al. [101])

had malocclusion. According to Kim and Guilleminault [94], 93.3% of 400 nonobese children (between 2 and 17 years of age) with diagnosed SDB had craniofacial features considered to be risk factors for SDB, including small retruded mandible and high and narrow hard palate associated with narrow nasomaxillary complex. Craniofacial dysmorphosis in the nonobese or slightly obese patients with OSA is commonly recognized, which may be associated with a genetic or epigenetic predisposition or a growth disorder during childhood [95]. On the other hand, severely obese children with OSA (mean body mass index of 48 kg/m^2) were observed to have no significant craniofacial abnormalities [96]. Instead, dysfunction seemed to be related to the changes around the upper airway soft tissues, head posture, and hyoid bone position induced by obesity [97, 98]. The prevalence of craniofacial deformation in OSA patients may therefore vary depending on the obesity, severity of OSA, age, gender, and race of the study sample.

9.3.2 Craniofacial Alteration by Physical Upper Airway Obstruction in Children

It was previously assumed that enlarged adenoids and palatine tonsils were the major cause of SDB in early childhood. Long-standing influence of nasopharyngeal obstruction by adenotonsillar hypertrophy on the facial morphology is described as "adenoid face" or "long-face syndrome" [99]. Physical upper airway obstruction gives rise to mouth breathing and environmental impairment of orofacial muscle activity, particularly in the geniohyoid, the genioglossal muscles of the tongue, the suprahyoid dorsal tongue fibers, the upper lip elevators, and the digastric muscles. Impaired nasal breathing had an impact on the development of nasomaxillary complex in related to the extended head posture, and secondarily on the mandibular position. The position of the condyle in the articulation changes transferring cartilaginous production more posteriorly [100]. This will alter the incline at which bone grows, causing a posterior mandibular rotation narrowing the upper airway conversely. This leads to craniofacial deformation such as vertical maxillary excess, constricted maxilla, high palatal vault, retrognathic mandible, long face with hyperdivergent vertical pattern, and excessive lower facial height, which deteriorates the airway dimension and mouth breathing, particularly during sleep (Fig. 9.18) [101]. McNamara [102] insisted that there was a potential interaction between the respiratory obstruction and vertical growth patterns based on the comparison of clinical cases between patients with untreated nasopharyngeal obstruction and patients who chose adenotonsillectomy (Fig. 9.19). Even with a normal sagittal skeletal relationship, the mouth breather exhibited the increase in anterior facial height and relative posterior displacement of the maxillary complex, causing the face to become more retrognathic (Fig. 9.20).

In terms of hyoid bone position, it was found that the children with enlarged tonsils impinging on the oropharynx had a more inferiorly positioned hyoid bone in reference to the mandibular plane (hyoid to

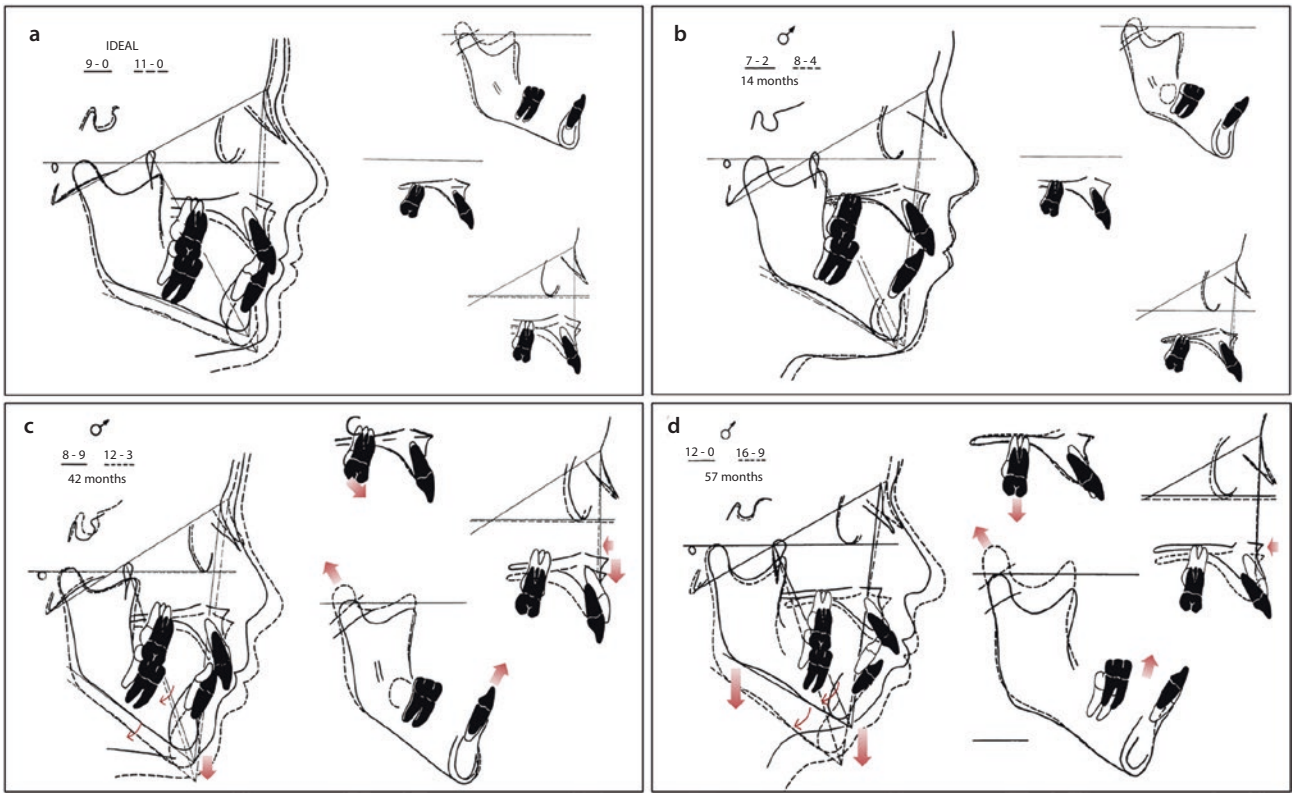


Fig. 9.19 A, Ideal face growth from 9 to 11 years of age: The maxilla descended vertically with nasion and point A retaining the same anteroposterior relationship. The mandible was displaced forward with condylar growth, and the mandibular plane angle was slightly decreased. B, Growth in children who underwent adenotonsillectomy from 7 years 2 months to 8 years 4 months of age: Dramatic closure of the mandibular plane angle and reduction of vertical growth pattern occurred without any orthodontic intervention. C, Growth in children with untreated nasopharyngeal obstruction from 8 years 9 months to 12 years 3 months of age: Vertical facial growth pattern with increased anterior facial height was noted and posterior maxillomandibular rotational displacement caused the face more retrognathic. D, Growth in children with complete pharyngeal airway obstruction by surgical intervention of submucous cleft of soft palate from 12 years to 16 years 9 months of age: Severe distortion of facial pattern was progressed presumably due to pharyngeal flap surgery which might cause the alterations in neuromuscular function and the subsequent adaptation necessary for maintaining oral respiration

mandibular plane, H-MP) [103]. However, there were no significant differences observed between the enlarged tonsils groups and the normal children when the sagittal position of the hyoid bone was compared in relation to either the cervical spine or the gnathion (the most anterior and inferior point of the symphysis). Nelson et al. [104] reported that snorers showed a lower position of the hyoid bone with respect to the mandibular plane (greater H-MP distance) during the prepubertal and pubertal period. Even in adults, snorers displayed lower position of the hyoid compared to nonsnorers.

Regarding the mechanism of craniofacial alteration, adenoids and tonsils influence function due to their relative size compared to the available space in the pharynx and not because of their absolute size [105]. Because of their entrapment in a relatively small space due to adenotonsillar hypertrophy, pharyngeal soft tissues will also become functionally impaired. If this impairment is counteracted by an increase in tongue and facial muscle activity, there may be no soft-tissue functional deficits. However, patients with inadequate tongue and facial muscle strengthening to counteract soft-tissue impairment will not return to normal breathing even after treatment like adenotonsillectomy or nasal allergy. Harvold [106] speculated that the nature of the structural alterations produced by nasal obstruction in experimental monkeys depended on their unique, individual ability to achieve neuromuscular adaptation. If there is a direct form-and-function relationship between nasal obstruction and the course of craniofacial alteration, it can be assumed that removal of the causative obstruction would initiate a reversal of functional changes, leading to a gradual corrective change in the previously altered craniofacial configuration. However, this does not always happen. The understanding of these relationships between function and growth of the craniofacial structures gave rise to the necessity of myofunctional therapy to correct the deficits caused by abnormal growth patterns.

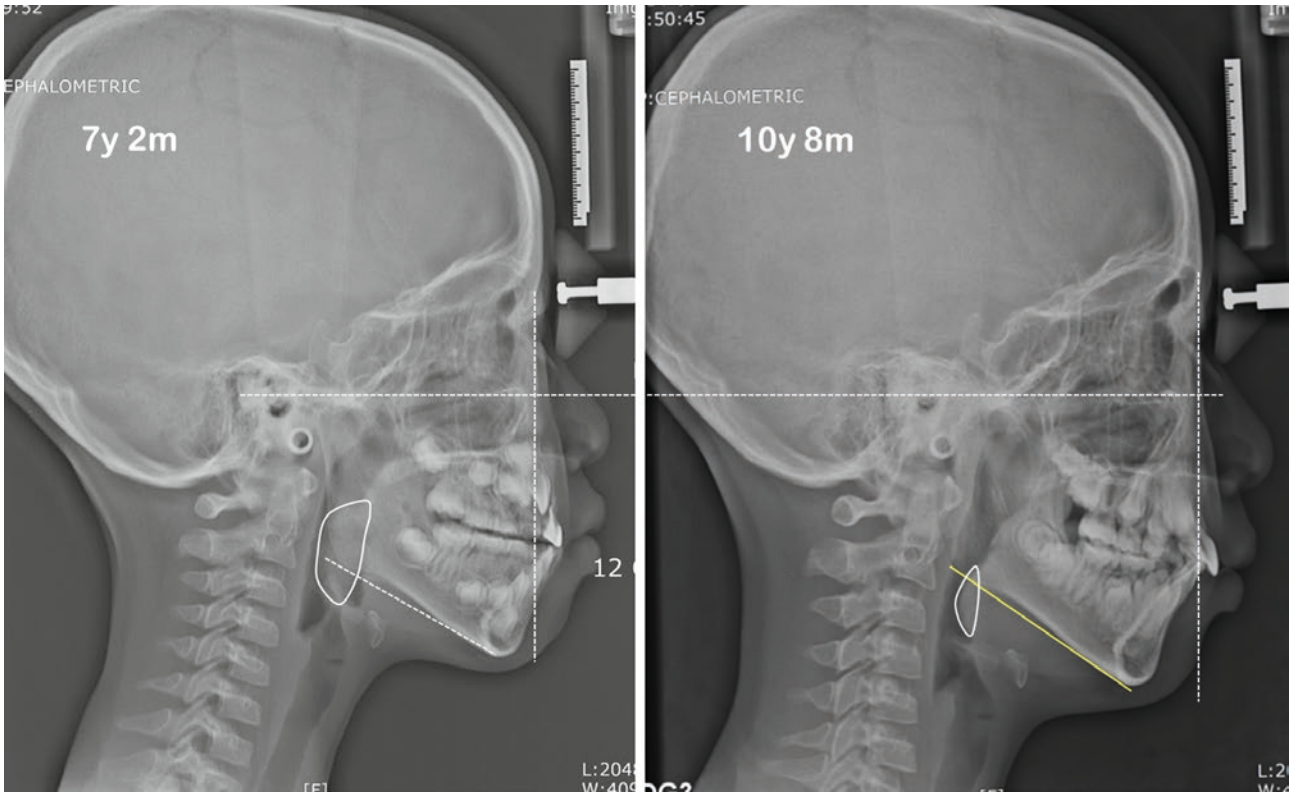


Fig. 9.20 A patient who showed craniofacial alteration of increased vertical dimension and retruded chin at 3.5 years after tonsillectomy. Persistent mouth breathing with low tongue posture probably in relation to narrow retroglossal airway with residual lingual tonsils might aggravate Class II hyperdivergent skeletal pattern

Macari et al. [107] performed a study whose results have great clinical significance to help determine the optimal timing of early adenoidectomy in consideration of the critical age of irreversible dysmorphologic growth change. They evaluated the relationship between adenoid hypertrophy and facial morphology in children (mean age: 6 years) dividing groups younger than 6 years and older than 6 years. Facial dysmorphology developed, starting with the maxilla, tilted posteroinferiorly as measured by reverse inclination of palatal plane (around −8°), at mean age of 4.37 years in the younger group (<6 years) with the smallest distance between the adenoid and soft palate. This study implies that early clearance of the nasal passage is required to arrest or reverse facial alteration in the most severely affected children.

Again, however, this is not always the case. Studies of various clinical populations have indicated that mouth breathing by respiratory obstruction existed in patients with a variety of sagittal and vertical skeletal types [94, 106, 108]. Feres et al. [109] insisted that there was no difference between obstructive and nonobstructive patients concerning all cephalometric skeletal variables. Furthermore, correlations between skeletal parameters and the percentage of adenoid obstruction were reported to be low or insignificant (Fig. 9.21).

9.3.3 Craniofacial Characteristics of Pediatric SDB Patients

Previous studies in nonsyndromic children with SDB symptoms have shown a positive association with craniofacial disharmony [110–112]. The evidence from case series and some excluded trials have suggested that mouth-breathing children with OSA largely present with a retrognathic mandible, micrognathia, excessive lower anterior face height with increased mandibular plane angle, and narrow maxilla with a high palate [113–116].

On the other hand, Huynh et al. [110] demonstrated that SDB symptoms in the pediatric cohort were primarily associated with the long face with increased vertical dimension and the narrow face with transverse deficiency, whereas the anteroposterior skeletal deficiency like retrognathism was not significantly related to the SDB symptoms. These findings were obtained from

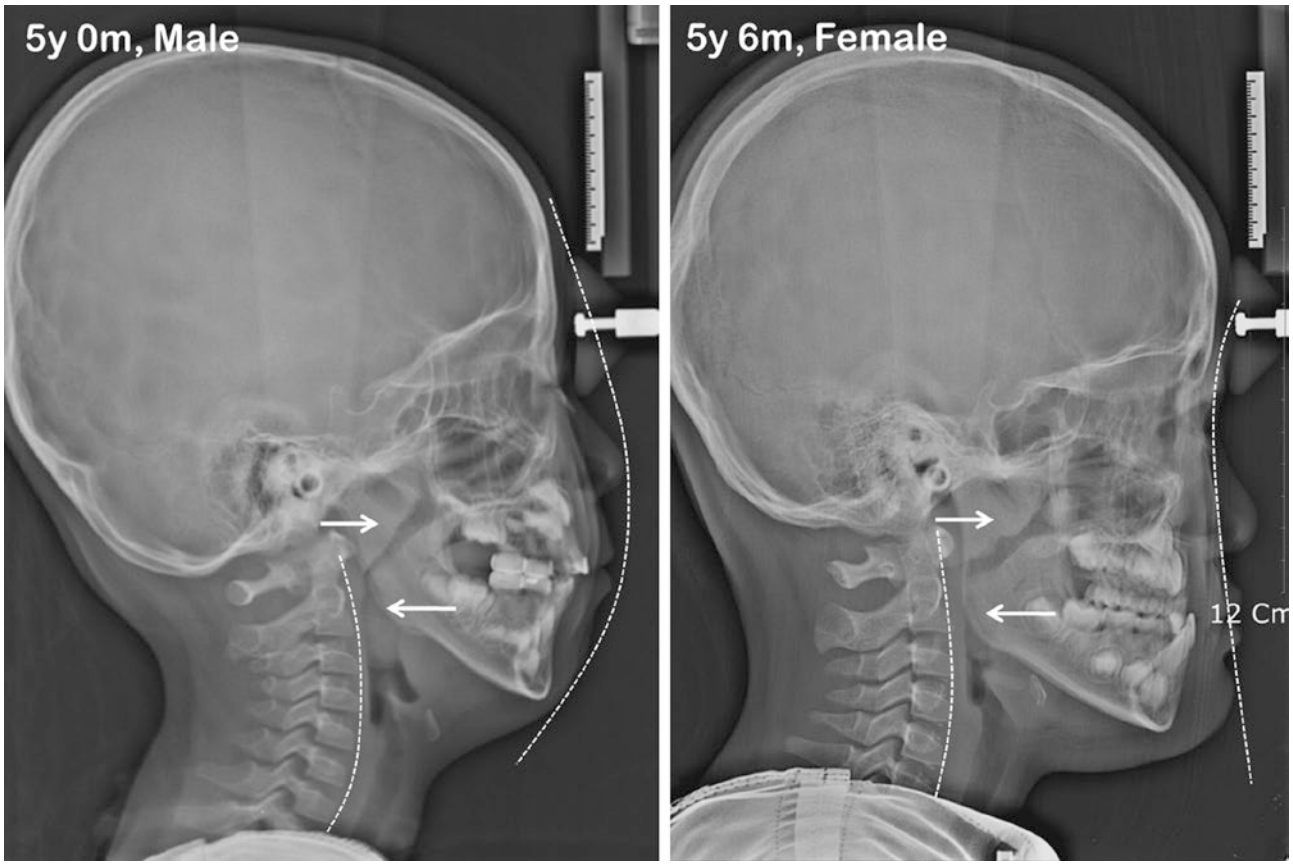


Fig. 9.21 Different facial growth patterns of young children with adenotonsillar hypertrophy. The patient at left showed a Class II hyperdivergent pattern with extended head posture, whereas the patient at right showed a Class III hypodivergent pattern. (Feres et al. [109])

604 Canadian orthodontic population (mean age 13.01 years; range 7–17 years).

Kim et al. [117] discovered that some Korean children aged 9–11 years who had impaired respiration exhibited protrusive growth of the maxilla and mandible in puberty. The authors explained that the active compensational changes of facial growth for the narrow airway might result in a Class III skeletal relationship. Anderson et al. [118] categorized the craniofacial patterns of 236 Korean children with chronic snoring into three characteristic clusters according to different age groups (Fig. 9.22): cluster 1 included younger children 5–8 years of age with increased vertical discrepancy and without definite sagittal skeletal discrepancy; cluster 2 included older children 9–12 years of age with progressed sagittal and vertical discrepancy representing a skeletal Class II, hyperdivergent pattern; and cluster 3 included children aged 7–8 years of age with a skeletal Class III, hyperdivergent pattern. The authors agreed that adenotonsillar hypertrophy-related snoring might cause earlier influence on vertical growth pattern, showing no typical sagittal pattern of craniofacial alteration, a finding supported by a more recent study of 30 Caucasian children with nasal obstruction-related primary snoring [119].

Other contradictory studies did not report such associations [120, 121]. A recent systematic review by Katyal et al. [122] concluded that evidence of a direct causal effect between craniofacial structure and pediatric SDB was lacking, even though there was strong support for reduced upper airway dimension in children with OSA (between 0 and 18 years of age). Children with SDB had an increased ANB angle but by less than 2 degrees compared to controls, which could be regarded as having marginal clinical significance. The mandibular plane angle showed a trend toward hyperdivergence, but with significant heterogeneity across the studies. This finding is in contrast to prior studies, showing increased lower anterior face height and mandibular plane hyperdivergence in adults with OSA [123, 124].

In summary, inherent craniofacial growth might be altered by functional problems of respiratory obstruction and SDB despite present controversies arising from limitations of methodologic inconsistency across studies (Table 9.1). This suggests that early intervention to treat pediatric SDB improving nasal breathing, such as adenotonsillectomy and nasal allergic treatment, should be considered to prevent irreversible craniofacial alteration, especially in the vertical direction.

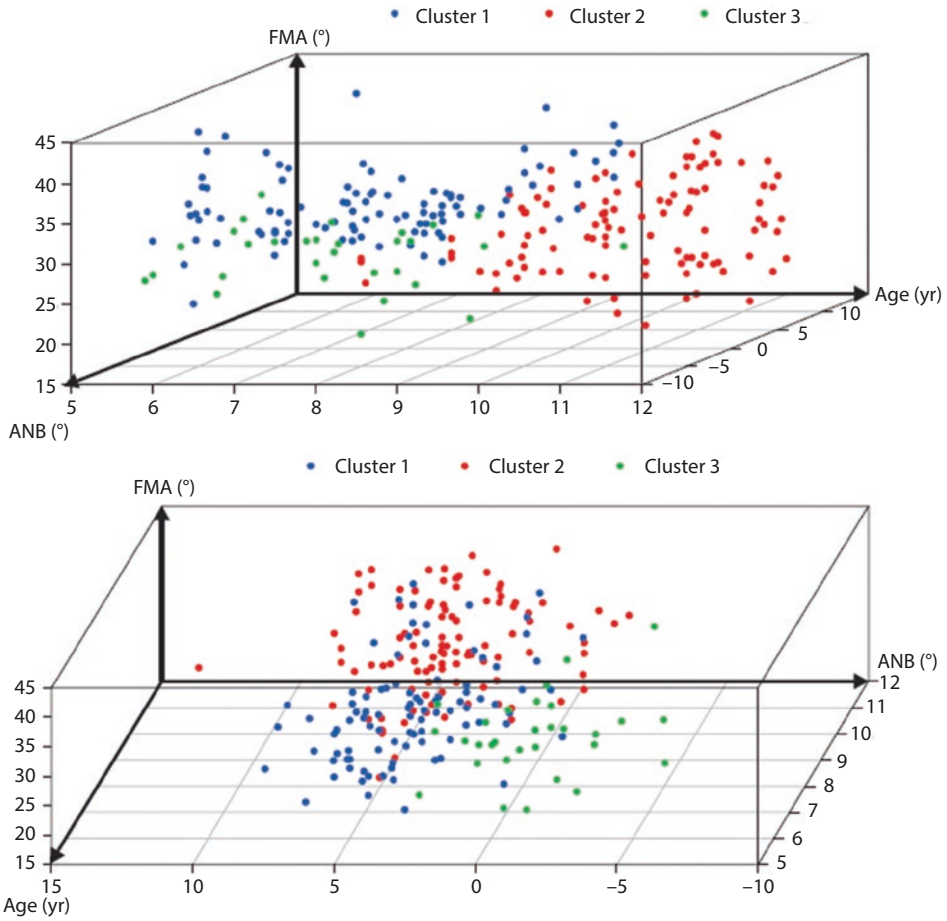


Fig. 9.22 Simplified three-dimensional scatter plots describing the result of a cluster analysis. **a** A scatter plot constructed using the factors ANB (X-axis), age (Y-axis), and FMA (Z-axis). **b** A scatter plot constructed using the factors age (X-axis), ANB (Y-axis), and FMA (Z-axis). Three clusters can be identified in the three dimensions, and clusters 1, 2, and 3 are indicated by blue, red, and green dots, respectively. (Anderson et al. [118])

Table 9.1 Timeline of normal development and craniofacial alteration resulting in abnormal respiratory function

Critical time point		5–6 y	7–9 y	12–15 y	15–18 y
Main growth event		Neural growth ends (~95%): from cranial to facial dynamics 1st molar eruption	Benchmark of midface growth Juvenile mandibular growth spurt	PHV (puberty) permanent dentition	Post-adolescence growth decelerates/ends
Normal growth and development					
Main skeletal growth site	Cranium	1. Basicranial flexion → Dolicho/Brachy pattern → Affects facial depth, width, height 2. Cranial base lengthening → Displaces maxilla forward 3. Mid- cranial fossa development → Displaces glenoid fossa	1. Residual growth of ACB length: frontal sinus development 2. SES ossified → Affects facial height	1 Post-cranial base growth → SOS starts ossified → Increases maxillary depth → Affects mandible position	1. PCB growth ends: SOS closed
	Naso-maxillary complex (NMC)	1. Displacement by ACB growth (*) & circum-maxillary sutural growth 2. Nasal septal cartilage → Nasal cavity development → Push midface forward 3. Additional surface remodeling	1. Forward and downward displacement by sutural growth (*) → Width, depth height growth 2. Downward drift of palate (PNS) → Palatal depth remodeling → Soft palate uprighting → Nasal cavity increase	1. Maxillary width and depth growth decreased 2. Active maxillary height growth and palate remodeling	1. Midpalatal suture ossified 2. Maxillary growth ends
	Mandible and hyoid	1. AP growth > vertical growth → Rate body length > ramus 2. Hyoid triangle established (~4–5 y)	1. Juvenile growth spurt – mandibular internal rotation → Gonial angle decrease → Determines mandibular shape	1. Mandibular growth spurt-vertical growth of condyle and ramus → CCW external rotation and surface remodeling 2. Second puberty of hyoid growth → consistent mandible-hyoid-cervix relationship	1. Residual growth of mandible 2. Stable hyoid-cervical relationship maintaining UA patency
Pharyngeal soft tissue	Adenoid tonsils soft palate	1. Airway dimension is mostly established and substantially maintained afterwards	1. Enlargement → No influence on retropalatal airway width: maxillary forward growth and palatal bone drift → Upright soft palate and increase nasopharyngeal airway → Compensate enlarged A&T	1. Reduction of A&T size	1. Later development of lingual tonsils → Affects retroglossal airway
	Tongue	1. Tongue development ends → Affects mandibular development 2. Backward and downward displacement toward oropharynx	1. Increase m tongue size → Transient decrease of retroglossal airway	1. Tongue moves lower → Upright soft palate → Increases retroglossal airway 2. Increase of retroglossal airway by mandible growth	

Table 9.1 (continued)

Critical time point	5–6 y	7–9 y	12–15 y	15–18 y
Craniofacial alteration affected by abnormal respiratory function				
Critical influencing factor on craniofacial growth	1. Extended head posture → Inhibit cranial flexion → Dolichocephalic pattern → Post. positioning of MC fossa → Protruded maxilla and retrognathic mandible 2. Low posterior tongue posture → Decent hyoid → Impairs mandibular forward G. and affects symphysis growth (by extended suprahyoid m.)	1. Nasal obstruction → Inhibits maxillary forward/lateral G. 2. Abnormal habit causing impaired palatal bone drift → Deep palatal vault, narrow arches, and narrow nasal cavity → Deficient constricted maxilla (CIII) 3. Excessive ATH → Retropalatal obstruction – Mouth breathing → Poor mandibular internal rotation → Increases mandibular divergency 4. Low tongue posture → Retroglossal obstruction → Hyperdivergency, long symphysis	1. Persistent ATH → Retropalatal obstruction mouth breathing → Increases maxillary height, increase of palatal depth, decrease of palatal width and Mandibular CW rotation long face and ant. openbite 2. Sagittal mandible growth is controversial	

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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.

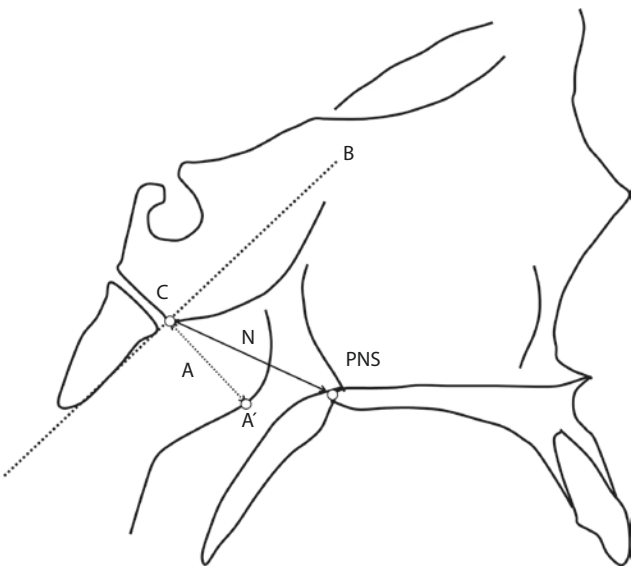
10.1 The Diagnostic Value of Cephalometrics for Airway Evaluation

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

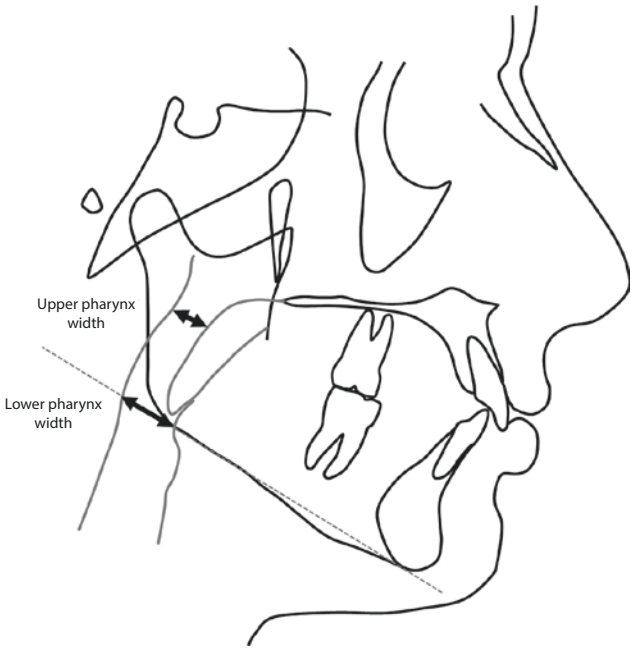


Fig. 10.2 McNamara Analysis. Airway widths according to McNamara analysis, upper pharynx and lower pharynx widths

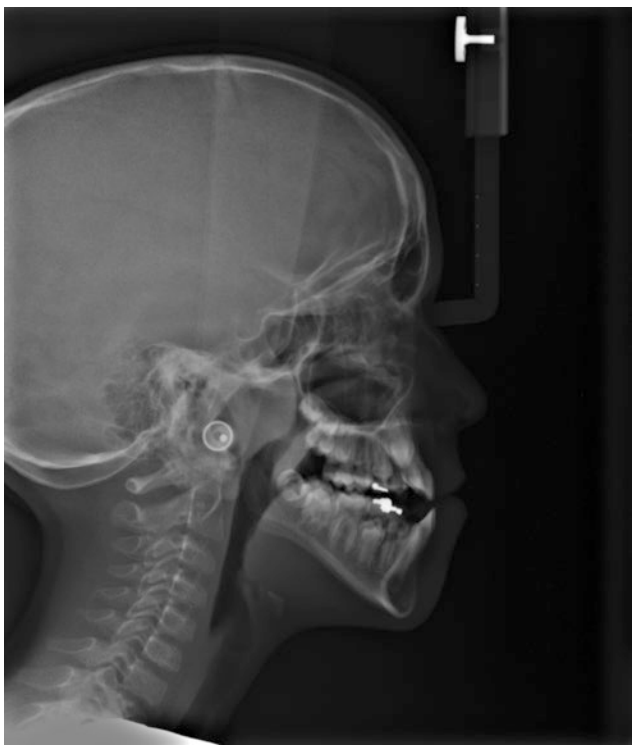


Fig. 10.3 Example of hypertrophic adenoid cephalometrics

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.

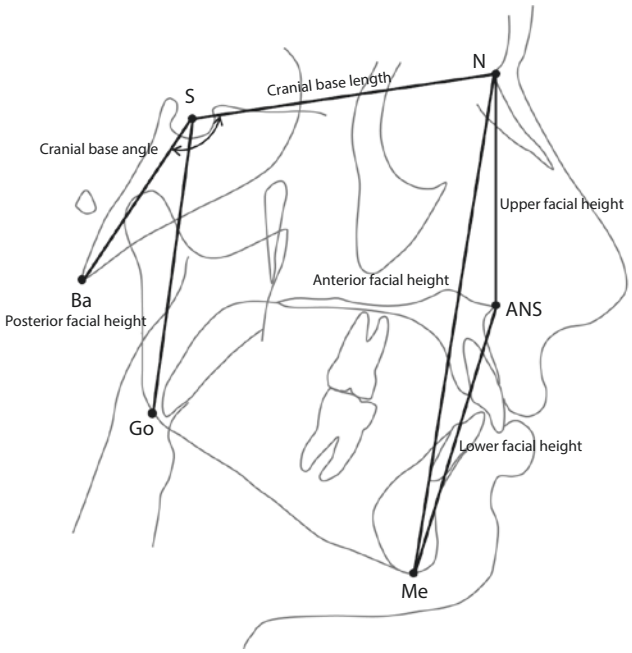


Fig. 10.4 Cranial base and facial height. Cranial base length: N (Nasion) to S (Sella), cranial base angle: N (Nasion) – S (Sella) – Ba (Basion), upper facial height: N (Nasion) to ANS (Anterior nasal spine), lower facial height: ANS (Anterior nasal spine) to Me (Menton), anterior facial height: N (Nasion)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

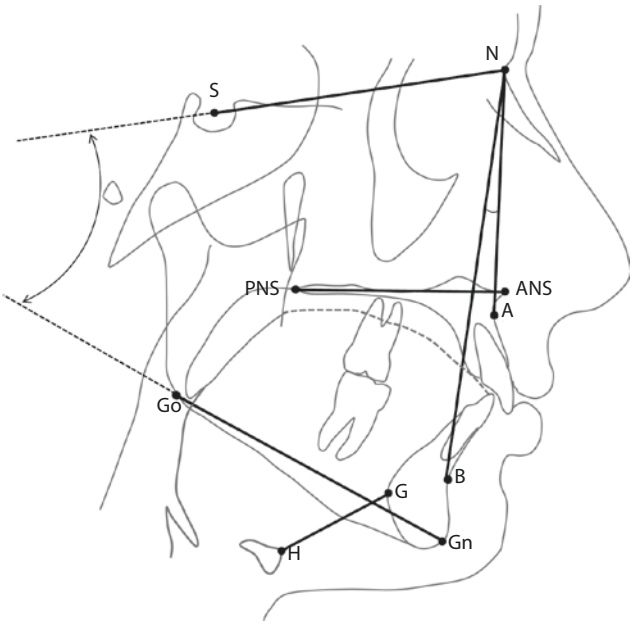


Fig. 10.5 Cephalometric landmarks and measurements. S (Sella), N (Nasion), A (A point), B (B point), SNA, SNB, ANB, PNS (posterior nasal spine), SN-GoGn (Gonion to Gnathion), G (Genial tubercle), H (Hyoid)

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).

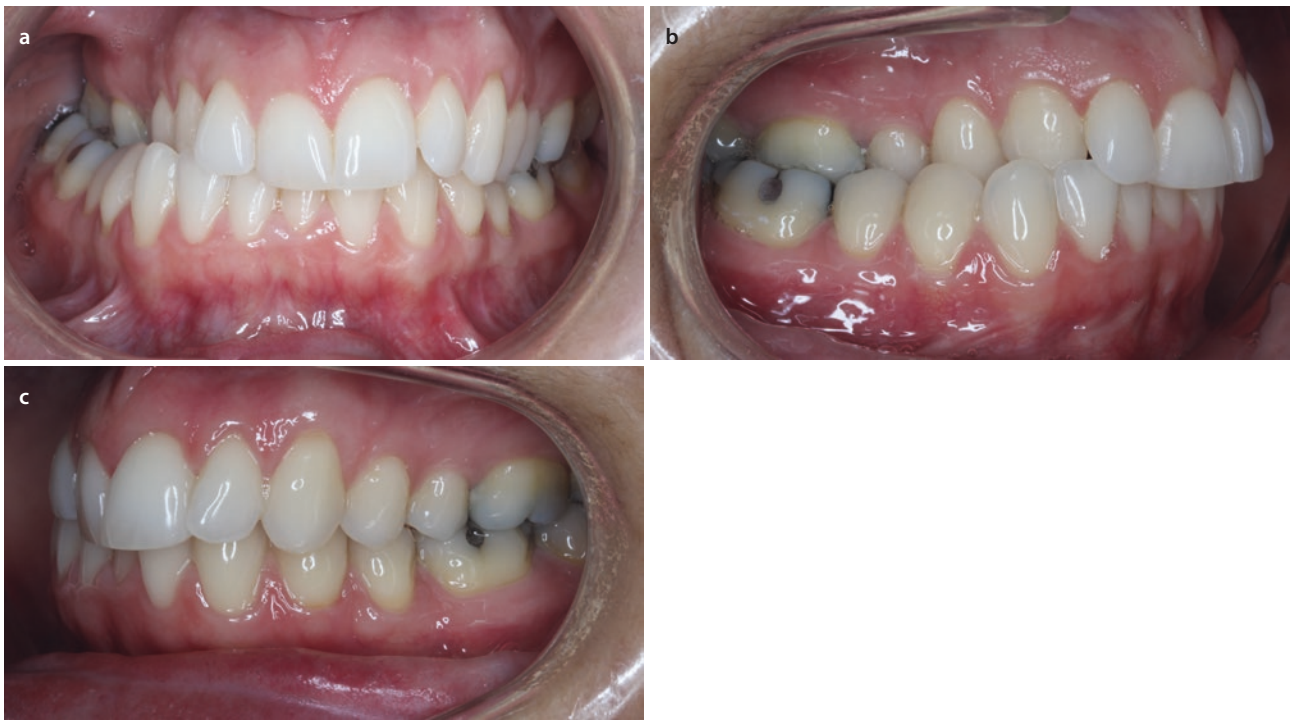
The primary purpose of maxillary expansion is to mechanically open the midpalatal suture with the intent to appreciably widen the maxilla. Therefore, this technique has been mainly used for adolescents whose sutures are not fully integrated. For adults, however, surgically assisted expansion is recommended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

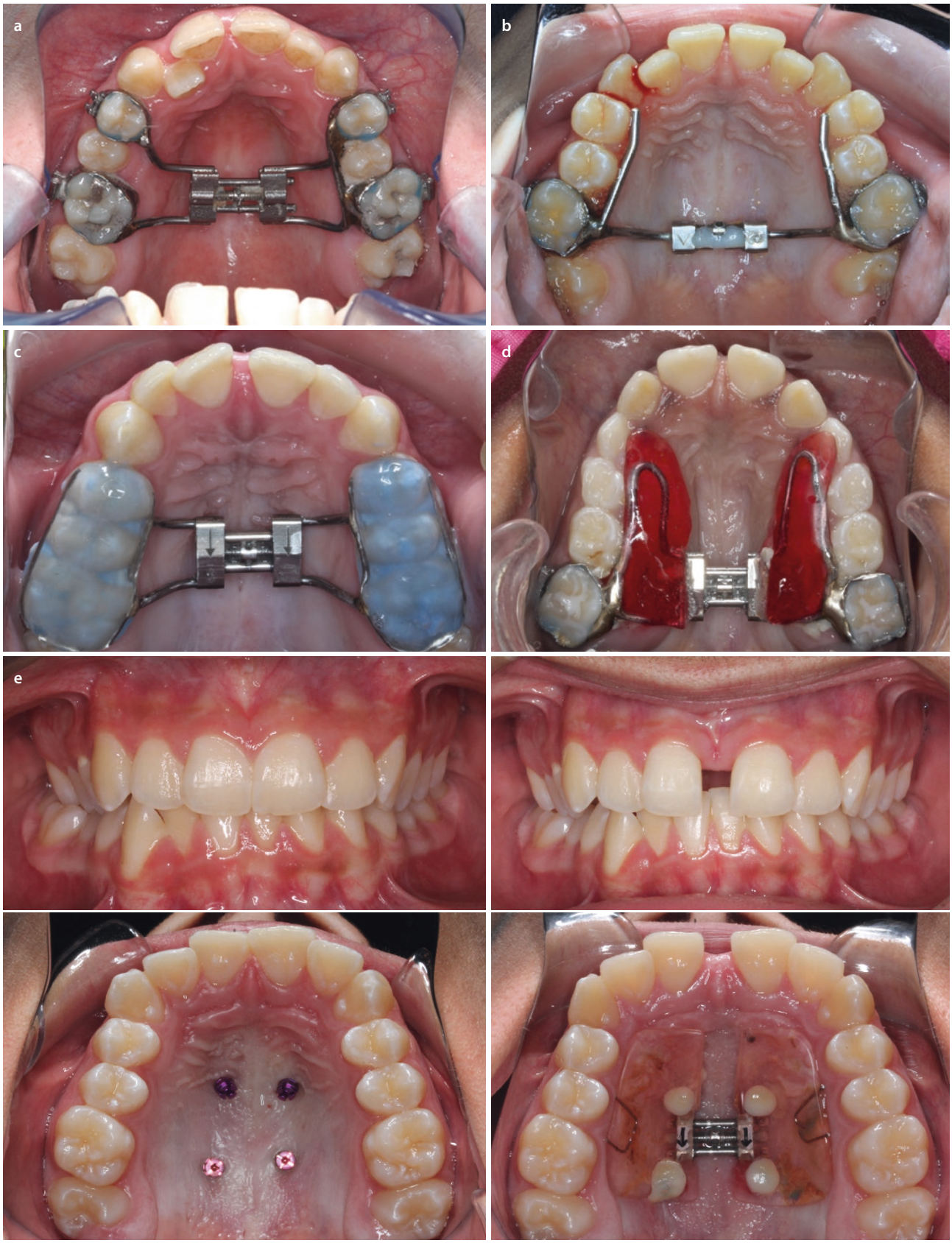
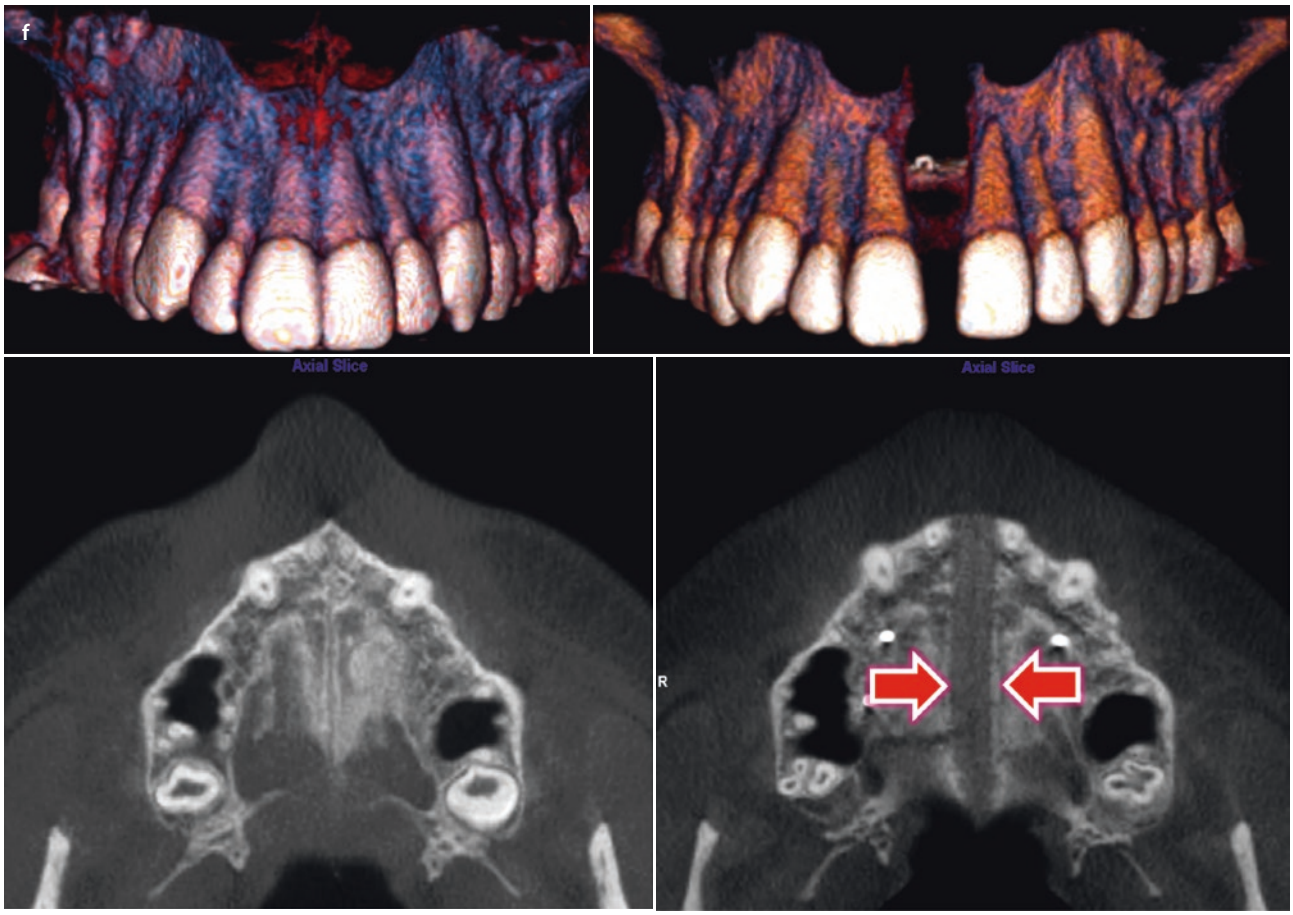


Fig. 10.8 Various expansion devices: **a** Four bands expander, **b** Two bands expander, **c** Bonded expander, **d** Haas expander, **e** Miniscrew-supported expander, **f** Before and after miniscrew-supported expander



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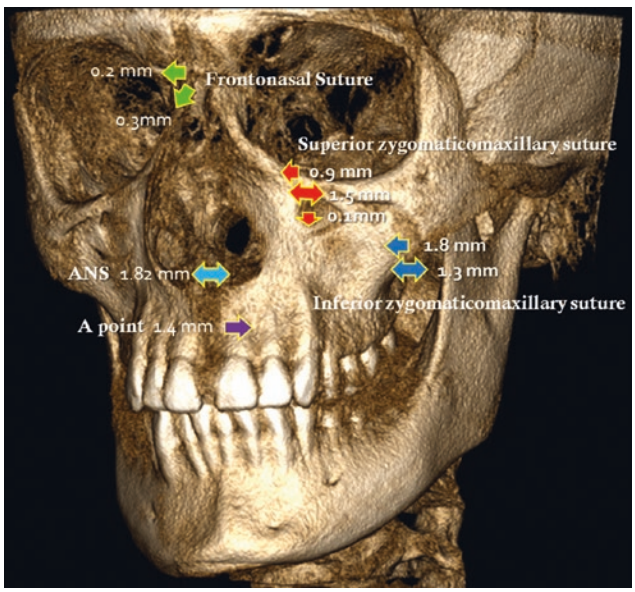


Fig. 10.9 Sutural changes after RME

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

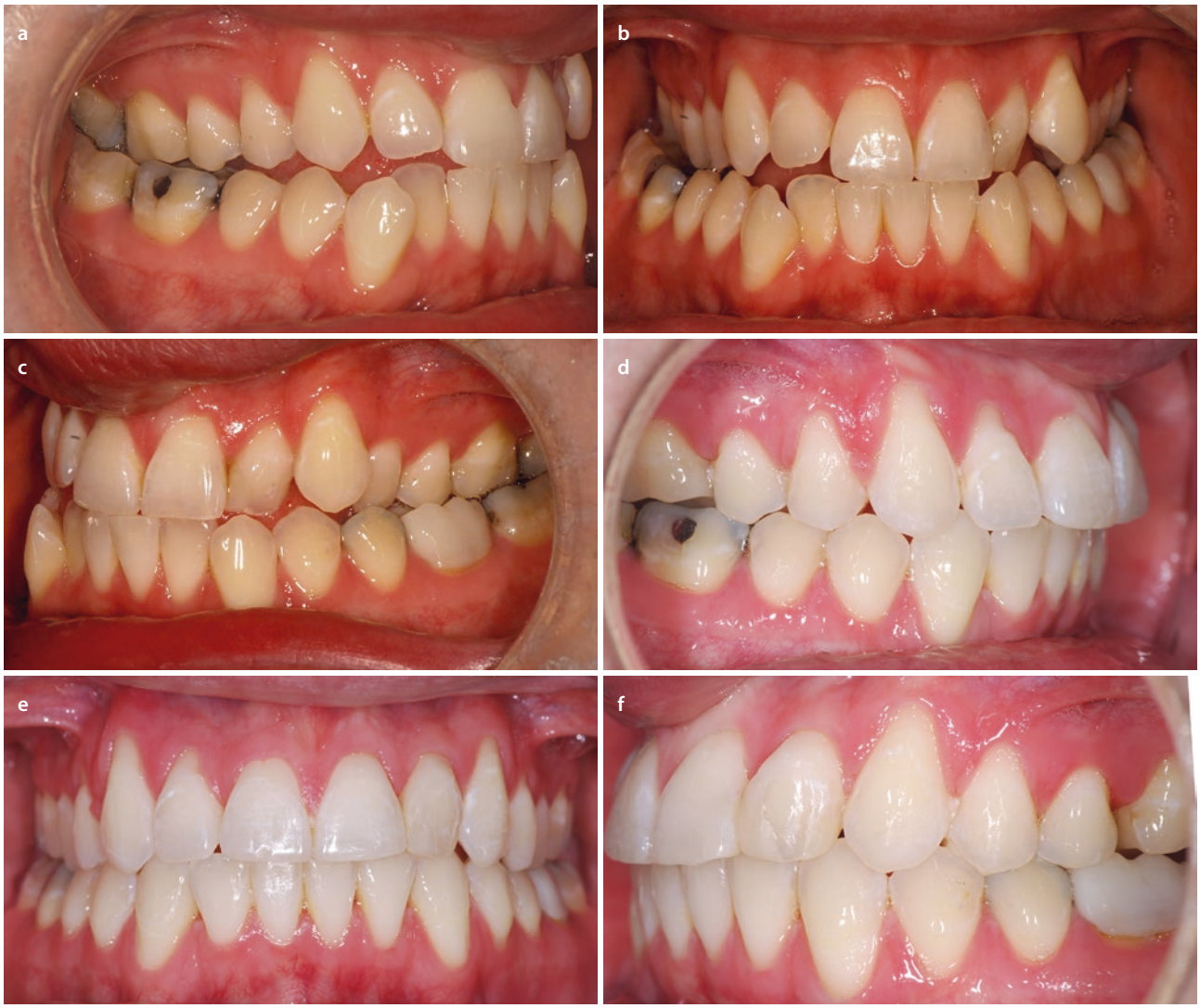
months after the end of the treatment, no significant changes in the AHI were observed. Pirelli et al. studied the long-term efficacy after a 12-year follow-up. All 23 patients were still normal as confirmed by PSG [211]. However, the long-term effect of maxillary width change in the early-treated (pre-pubertal) 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.

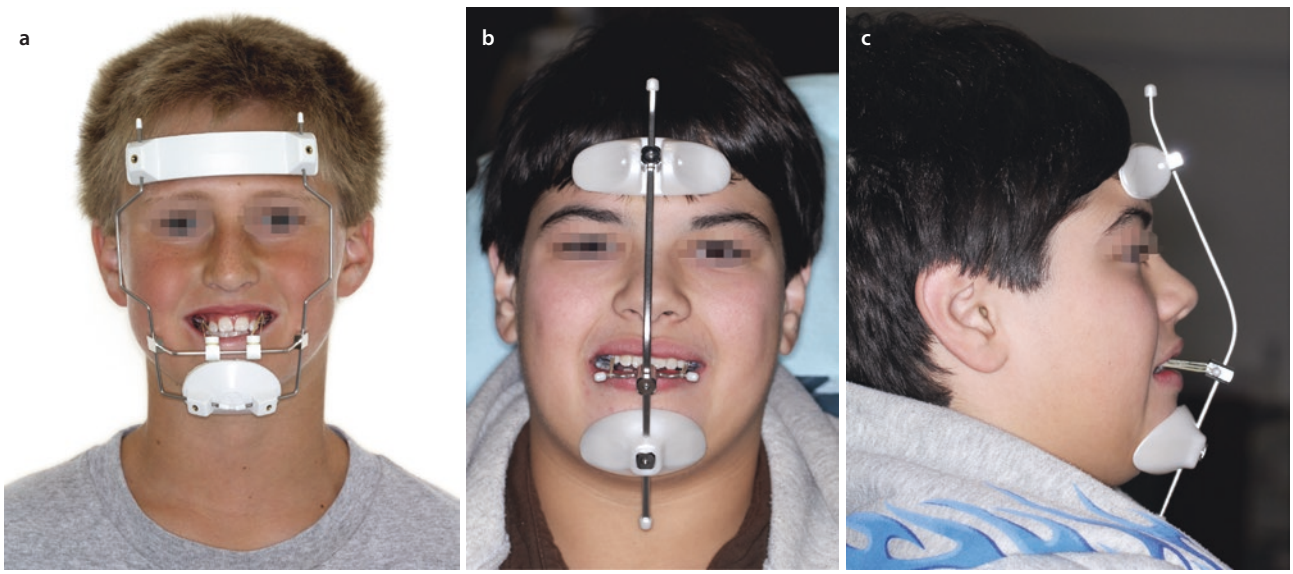


Fig. 10.12 Protraction headgear

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

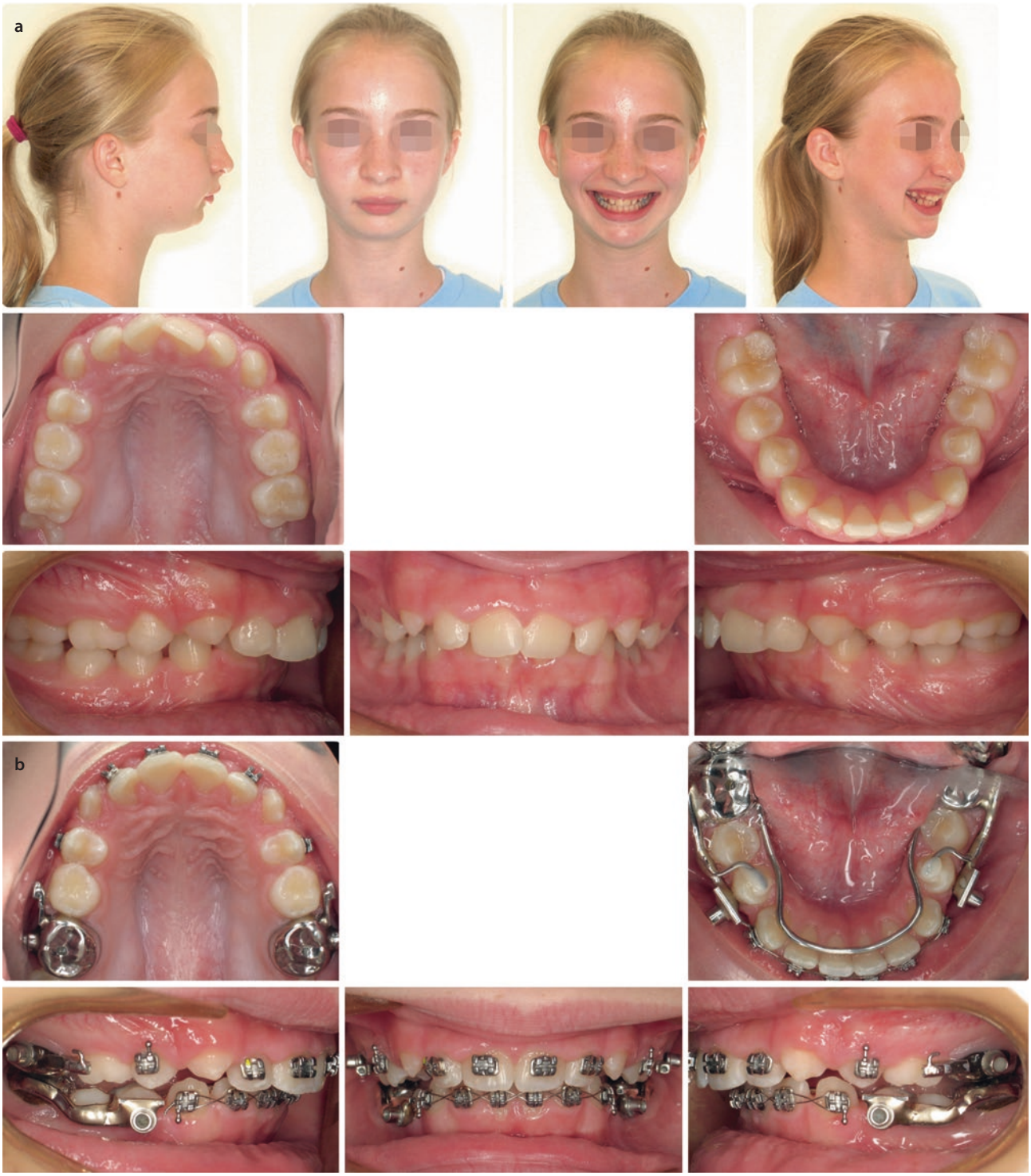
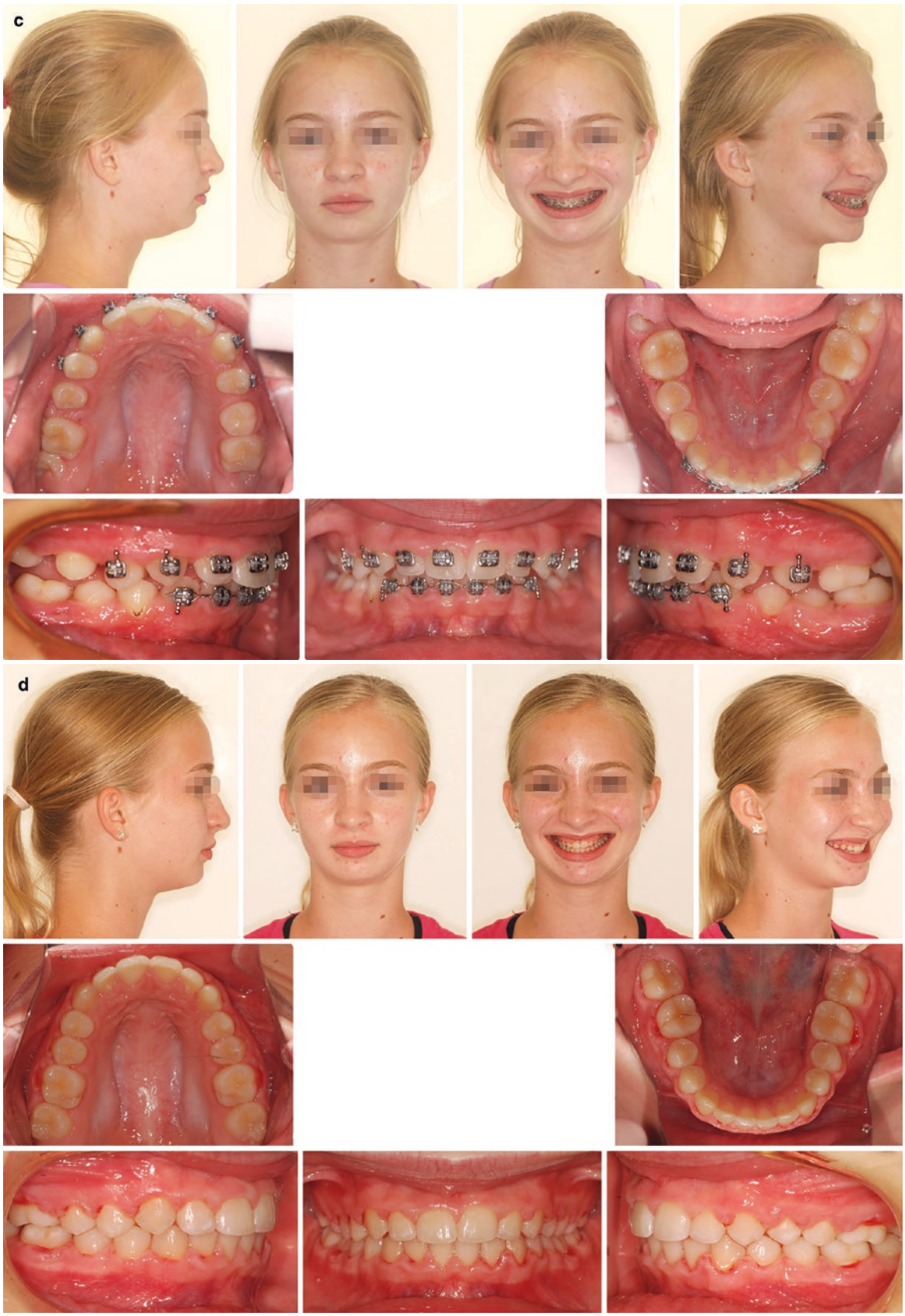


Fig. 10.14 Functional appliances. **a** Initial records, **b** Herbst appliance placed, **c** Herbst appliance removed, **d** Final records, **e** Initial cephalogram, f Cephalogram after Herbst appliance removed, **G** Final cephalogram



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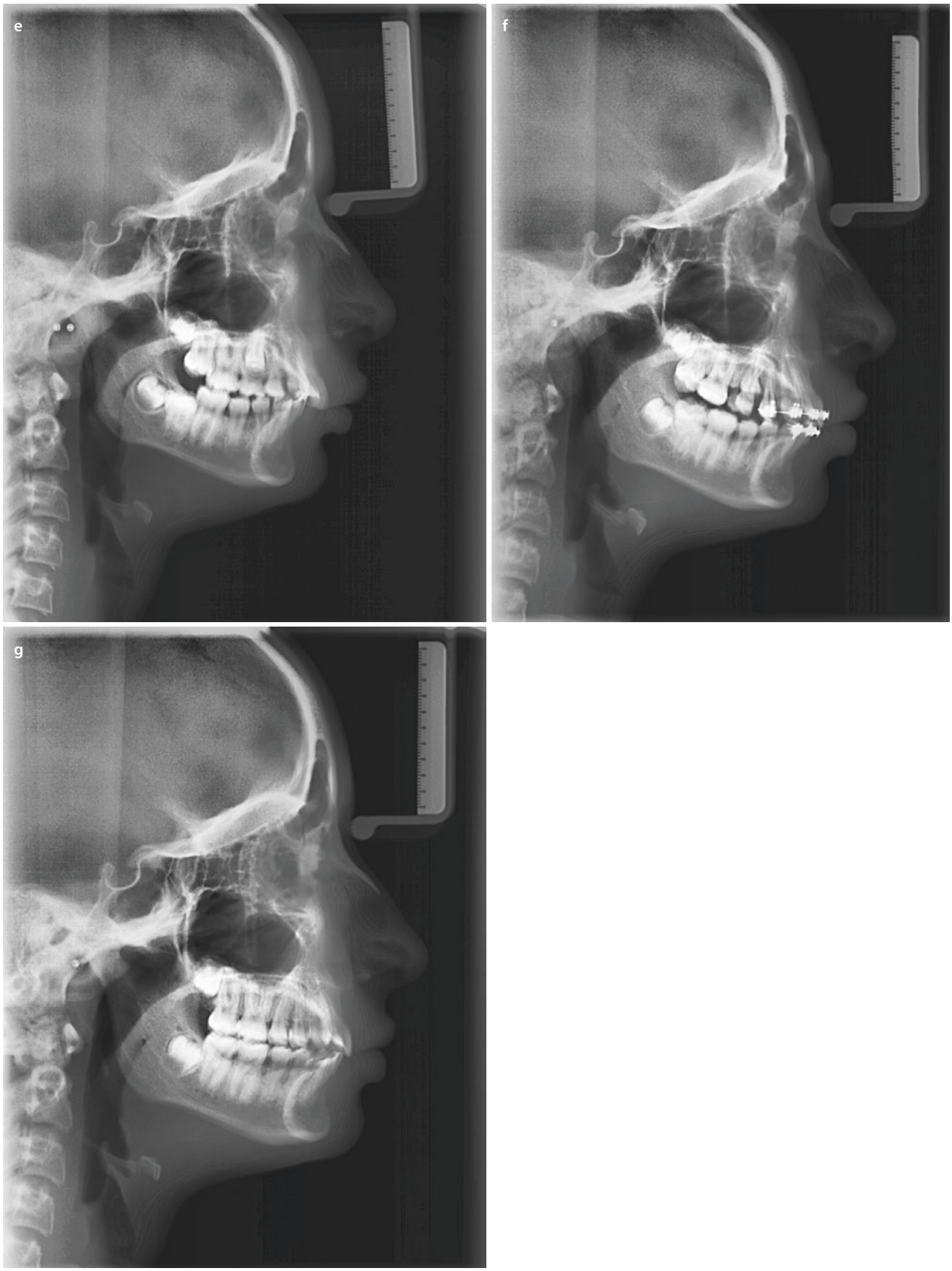

Fig. 10.14 (continued)

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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Obstructive Sleep Apnea in the Setting of Mandibular Condyle Resorption

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Contents

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Background
It is well documented that anatomic abnormalities such as macroglossia [1, 2] and mandibular retrognathia or hypoplasia [3] may contribute to obstruction of the airway. Likewise, acquired defects of the temporomandibular joint and resulting skeletal abnormalities may result in airway compromise. Most typically, this is observed in the setting of resorptive or destructive processes of the mandibular condyle, resulting in clockwise mandibular rotation and secondary decreased posterior airway space. In these cases, anatomic correction may often improve or eliminate airway compromise.

11.1 Pertinent Anatomy and Function

The temporomandibular joints (TMJs) are complex ginglymoarthrodial articulations of the mandible and the base of the skull. Thin bone separates it from the middle ear and middle cranial fossa. It is a synovial joint with an interpositional disc of dense fibrous connective tissue.

While the axis of rotation is in the mandibular condyle and it functions against the temporal bone in the glenoid fossa, the other end of the mandibular "lever" holds teeth that function against the maxillary dentition. It may be considered that the mandible is a sort of tripod, with the joints themselves as two legs and the dentition as the third point of stability. In this way, small changes in TMJ anatomy or physiology can effect large differences in occlusion and dental function. Fortunately, both the joint and the dentition have adaptive capacity, which allows for changes and compensation on either end of the lever over time and in mild disease states.

11.2 Pathophysiology

The TMJ is subject to the same diseases that may afflict other synovial joints, including osteoarthritis and inflammatory arthritis [4, 5]. Other disease states, such as acquired traumatic defects, autoimmune disease such as scleroderma, or idiopathic condylar resorption may affect the TMJ and lead to anatomic alterations giving rise to airway obstruction.

Osteoarthritic change often degrades and foreshortens the condyle [6]. Arthritic pain may or may not accompany these morphologic changes (Fig. 11.1). Degenerative changes to cartilage and underlying bone are the result of inability to adapt to mechanical load. This may be due to increased or unusual load, but may also be found in the setting of a normal load with dysfunctional cartilage. Further, intraarticular inflammation contributes to TMJ osteoarthritis. Finally, hormonal and genetic factors have been suggested as factors leading to TMJ osteoarthritis.

While osteoarthritis is a low-inflammatory arthritis, higher-inflammatory arthropathies, autoimmune, and rheumatologic diseases may affect the temporomandibular joint [7]. These include, but are not limited to, rheumatoid arthritis, juvenile idiopathic arthritis, scleroderma, psoriatic arthritis, and systemic lupus erythematosus (SLE) (references for each of these). The various pathophysiologies of these disease processes are well documented in other texts, and an in-depth discussion of each one is beyond the scope of this chapter. Suffice it to say that the temporomandibular joint may be affected by these conditions, causing symptoms including pain, intraarticular dysfunction, and condylar resorption or destruction. The unique case of scleroderma also results in bony destruction from the constant pressure of an ever-tightening, restrictive soft tissue envelope (Fig. 11.2).

So-called idiopathic condylar resorption (ICR) is another clinical entity that contributes to similar clinical findings, albeit with a less-clear pathophysiology [8]. Often painless, ICR is usually bilateral, affecting females between 15 and 35 years old. Etiology is unclear, but it may be initiated by autoimmune disease, hormonal imbalance, trauma, or jaw surgery in a susceptible individual (Fig. 11.3).

Condylar resorption in all of these conditions may progress until either the entire condyle is gone down to the mandibular ramus or the underlying disease process is controlled. This means that the resorption and resulting progressive retrognathia may eventually "burn out." It may also be stabilized and halted once there is adequate medical management of the underlying cause.

A last cause of acquired condylar resorption or similar anatomic change is trauma. Facial trauma resulting in injury to the mandibular condyles may eventuate in the same clockwise rotation of the mandible and anterior open bite in one of two ways. First, injury to the joint may initiate condylar resorption in the susceptible patient. This may be observed in gross facial trauma or potentially secondary to orthognathic surgery. Second, condylar or subcondylar fracture may result in foreshortening of the ramus-condyle unit if there is malunion, nonunion, failed fixation, or a failure to adapt through physiotherapy (Fig. 11.4).

11.3 Clinical Exam

Typical findings in an individual with condylar resorption severe enough to result in obstructive sleep apnea include:

- Clockwise rotation of the mandible
- Mandibular retrognathia/class II malocclusion
- Anterior open bite
- Decreased posterior face height
- Radiographic narrowing of the upper airway space

Fig. 11.1 Osteoarthritis-related degenerative joint disease resulted in a malocclusion and severe airway compromise over 2 years (A is prior to change and B is upon clinical presentation). AHI was 41

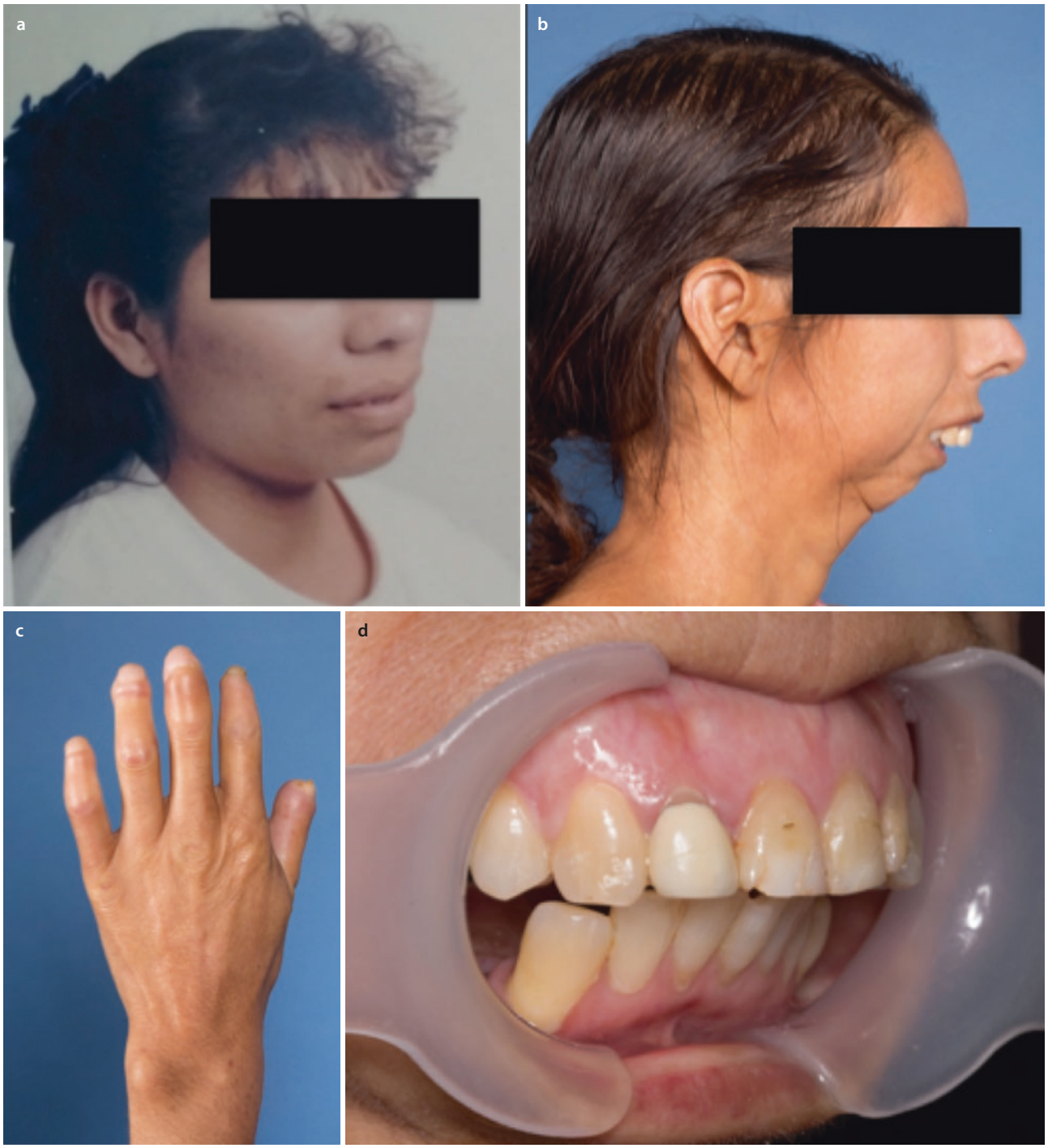

Fig. 11.2 Scleroderma-associated condylar resorption. The patient 15 years before presentation for sleep-disordered breathing and facial changes **a**. The same patient with severe retrognathia and skin tightening with retraction of the upper lip over the incisors **b**. Scleroderma-associated changes to the hands in the same patient **c**. Progressive malocclusion with mandibular retrognathia and associated dental compensation **d**

11.4 Diagnosis and Diagnostic Testing

In cases of suspected condylar resorption, exploration of etiology is imperative as is assessment of the extent of resorption [5]. In all cases, a thorough medical history is critical to help establish an accurate diagnosis, making note of systemic symptoms that may indicate a rheumatic autoimmune disorder such as inflammatory joint symptoms, photosensitive skin rashes such as a malar or discoid rash, Raynaud's with features of digital ulcers or ischemia,

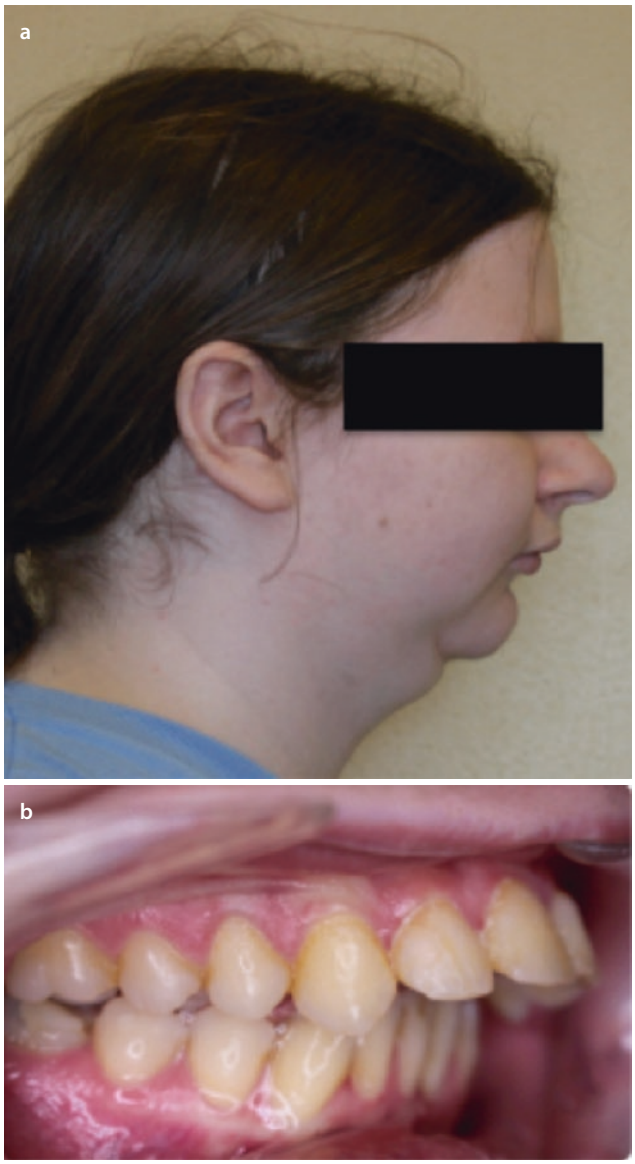

Fig. 11.3 A 21-year-old female with 3 years of progressive idiopathic condylar resorption. All laboratory studies were negative for a detectable systemic cause. Presentation included class II skeletal **a** and dental **b** malocclusion minimal intraarticular symptoms and AHI of 28

pleurisy, pericarditis, history of renal insufficiency of unclear etiology, cytopenias, or other similar findings.

Radiographically, multiple studies may be indicated. Lateral cephalometric films, particularly obtained serially over time, are easily obtained and help track progress of disease or treatment thereof (Fig. 11.5a). Lateral cephalometrics offers both measurement of the condylar height and the pharyngeal airway spaces (which are typically narrow in the OSA patient with condylar resorption). In the office, a screening panoramic radiograph may show shortened condyles and an overall short ramus/condyle unit (Fig. 11.5b).

Computed tomography (CT) with bony windows is likely to show more clearly the extent and dimensions of any condylar destruction as well as other important features important when considering surgical intervention (fossa anatomy, osteophytes or other loose bodies within the joint, EAC or middle ear abnormalities, or other craniomaxillofacial findings that may require evaluation or treatment) [9]. CT is particularly helpful in cases where alloplastic joint replacement is planned, but would be important for any major surgical intervention. Cone beam computed tomography (CBCT) is frequently used as an in-office modality of obtaining cross-sectional imaging for this purpose (Fig. 11.6). It is the author's opinion that CBCT may be helpful for screening and in some cases for following progression of disease, but that medical-grade CT is more useful for planning of major surgery (i.e., joint replacement or distraction). CT may also be used to print models for diagnosis and planning purposes (Fig. 11.7).

Nuclear medicine studies may indicate levels of bone metabolism in the temporomandibular joint. For example, in a patient who has some resorption but has been undergoing medical therapy aimed at arresting the process, a technetium-99 study may show if there is still high metabolic activity (possible therapeutic or diagnostic failure) or reduction/resolution of metabolic activity (therapeutic success or "burn out").

In many cases, it may already be known that a patient has inflammatory arthritis or some other condition. However, it is common that an underlying disease process has not been explored or diagnosed on initial presentation. Many simple blood tests may be considered in the evaluation of the patient with condylar resorption. Consultation and coordination of care with a rheumatologist is important to assure appropriate medical diagnosis and management. Authors commonly advocate for many of the following, among others [10–13]:

- Anticyclic citrullinated peptide (anti-CCP) antibodies
- C-reactive protein
- Erythrocyte sedimentation rate
- Antinuclear antibody
- HLA-B27
- Vitamin D
- Estrogen

11.5 Treatment

In some cases, medical management of the underlying cause for condylar resorption may be possible [5]. For example, use of a disease-modifying antirheumatic drug may arrest resorption and improve both pain and function of the temporomandibular joint. However, if resorption has progressed enough to contribute to air-

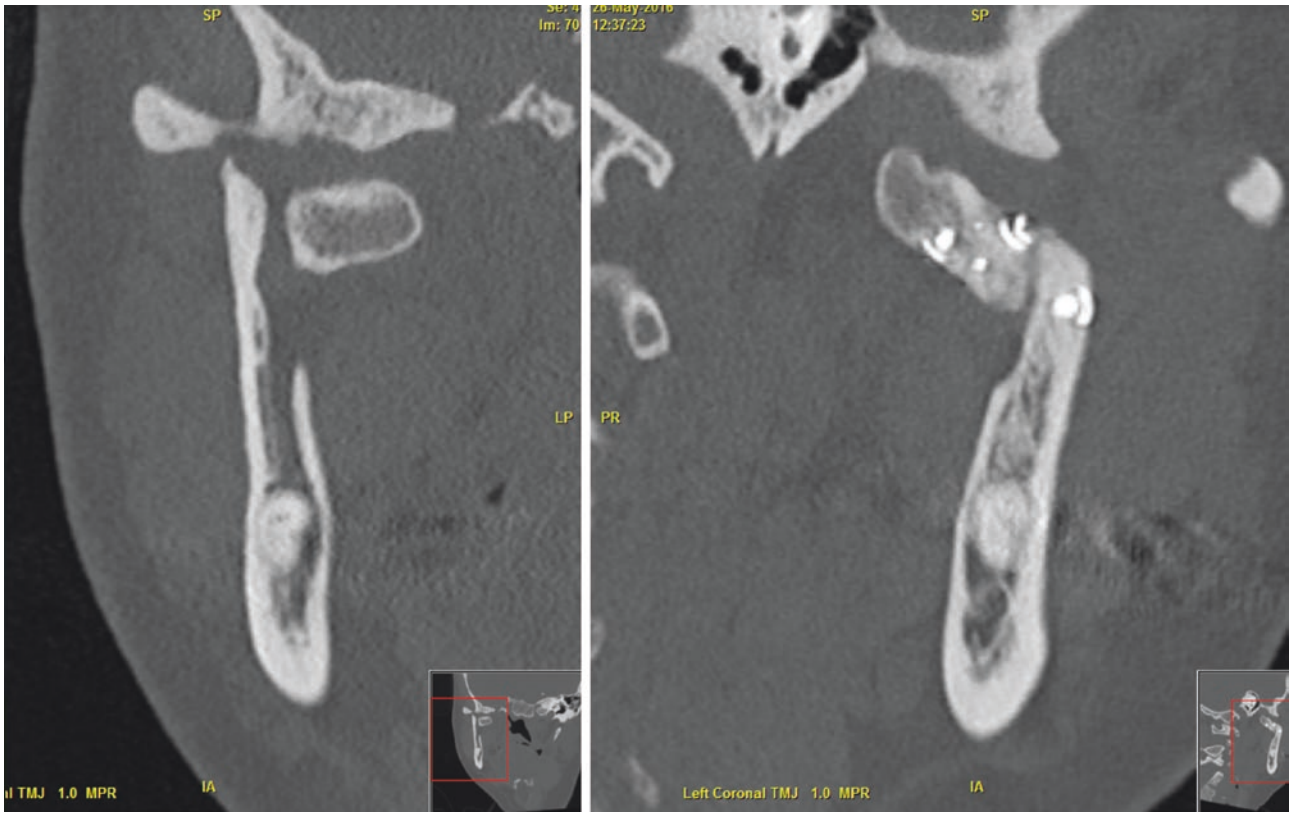

Fig. 11.4 Trauma-related clockwise rotation of the mandible in a patient who had a nonunion of the right condylar fracture and a malunion of the left condyle. The patient presented to our clinic with mandibular retrognathia, TMJ arthralgia, limited range of motion, and an AHI of 31

way obstruction, treatment of etiology must still give way to surgical correction in order to restore proper form. Advocates abound for multiple treatment modalities. The most common include:

- Orthognathic surgery
- Distraction osteogenesis
- Autologous reconstruction of the condyle
- Alloplastic reconstruction of the condyle (total temporomandibular joint arthroplasty and reconstruction)
- Tracheostomy

Traditional orthognathic surgery may be performed when the condylar resorption has ceased progression [5]. Most often, this will involve a counterclockwise rotation of the maxillomandibular complex with closure of the anterior open bite with in the mandibular portion of the surgery. Both bilateral sagittal split osteotomy and inverted-L osteotomies may accomplish this closure, depending on the amount of advancement and rotation required. Osseous genioplasty will also often assist in re-establishing appropriate facial proportions and lip competence. It is much easier to perform orthognathic surgery when there is still some remaining condyle to seat, but it is still possible after the condyles have completely resorbed. In addition, there is risk of additional condylar resorption following orthognathic surgery. Some authors feel this may be mitigated with medical treatments before and after surgery [5]. Others also advocate for combined intraarticular surgery (such as disc repositioning) and orthognathic surgery.

Distraction osteogenesis is a technique advocated for correction of malocclusion in patients with condylar resorption [14]. There is some evidence from animal studies that distraction techniques were less traumatic to the condyle than mandibular orthognathic surgery. In addition, there is other evidence suggesting long-term stability without relapse. Mandibular distraction may be accompanied by traditional Le Fort osteotomy as well in order to optimize jaw position and airway space. Distraction is a technique that may be considered when the condyle–fossa relationship is stable and without dysfunction. In cases where there is progressive deterioration of the condyles or significant TMJ dysfunction, it may be preferred to consider joint reconstruction.

Condylectomy with costochondral grafting is another method of restoring posterior face height and restoring a normal jaw relationship in the setting of con-

dylar resorption [15]. Both open and endoscopically assisted approaches have been described with some success, albeit with relatively short-term follow-up and not always in the setting of concurrent airway obstruction. This autogenous graft technique involves donor site morbidity, but outcomes are stable occlusally and show good TMJ function. This technique does not rely on

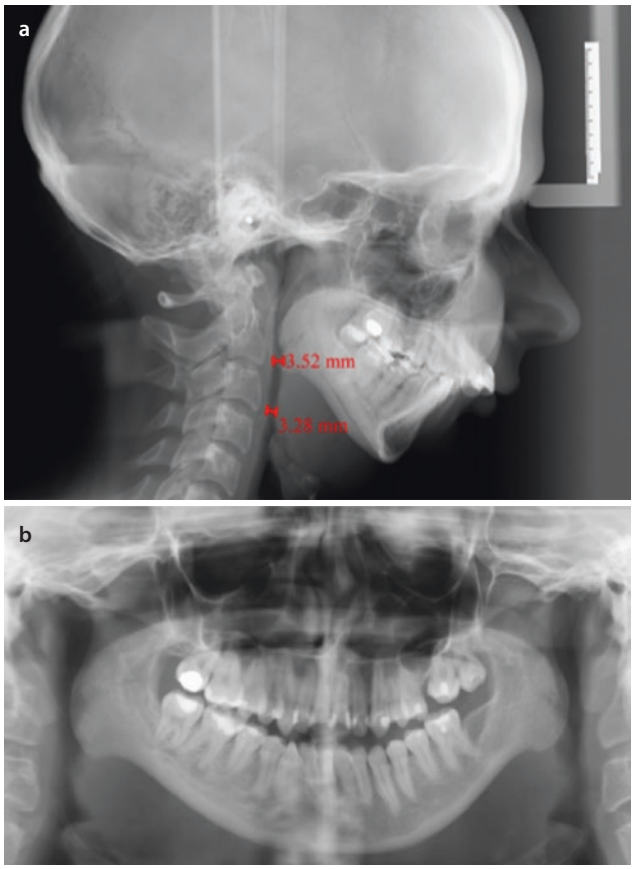

Fig. 11.5 Radiographic exam from the patient in Fig. 11.3. Lateral cephalometric film showing reduced posterior airway space, shortened posterior face height, and clockwise mandibular rotation **a**. Panoramic radiograph illustrating resorption of the mandibular condyles **b**

Fig. 11.7 Three-dimensional model printed for the patient in Fig. 11.2 with scleroderma. Note the complete absence of the condyle bilaterally and resorption of the nasal bones

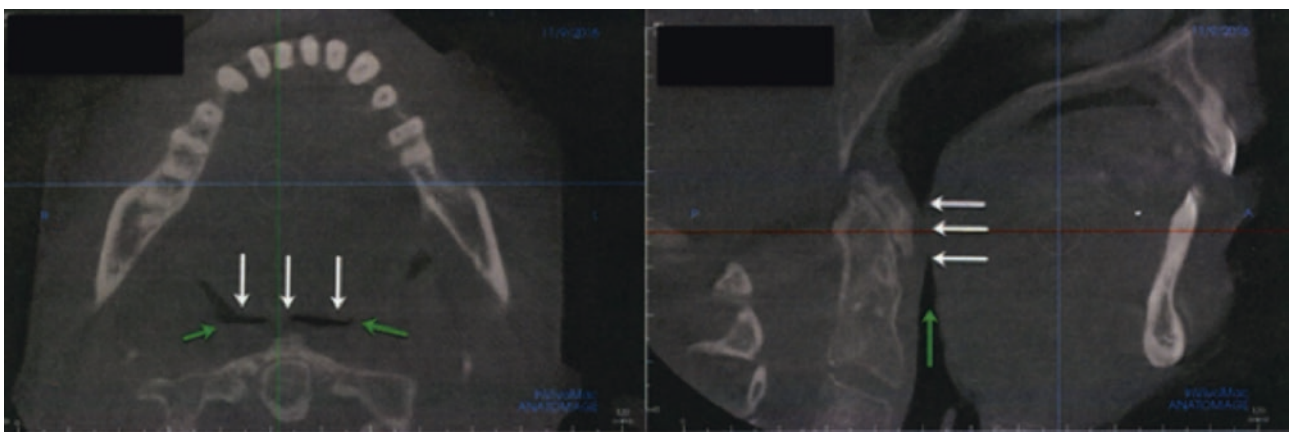

Fig. 11.6 Cone Beam Computed Tomography (CBCT) from the patient in Fig. 11.1. Axial and sagittal views clearly show diminished airway dimensions and may be utilized for volumetric analysis as well

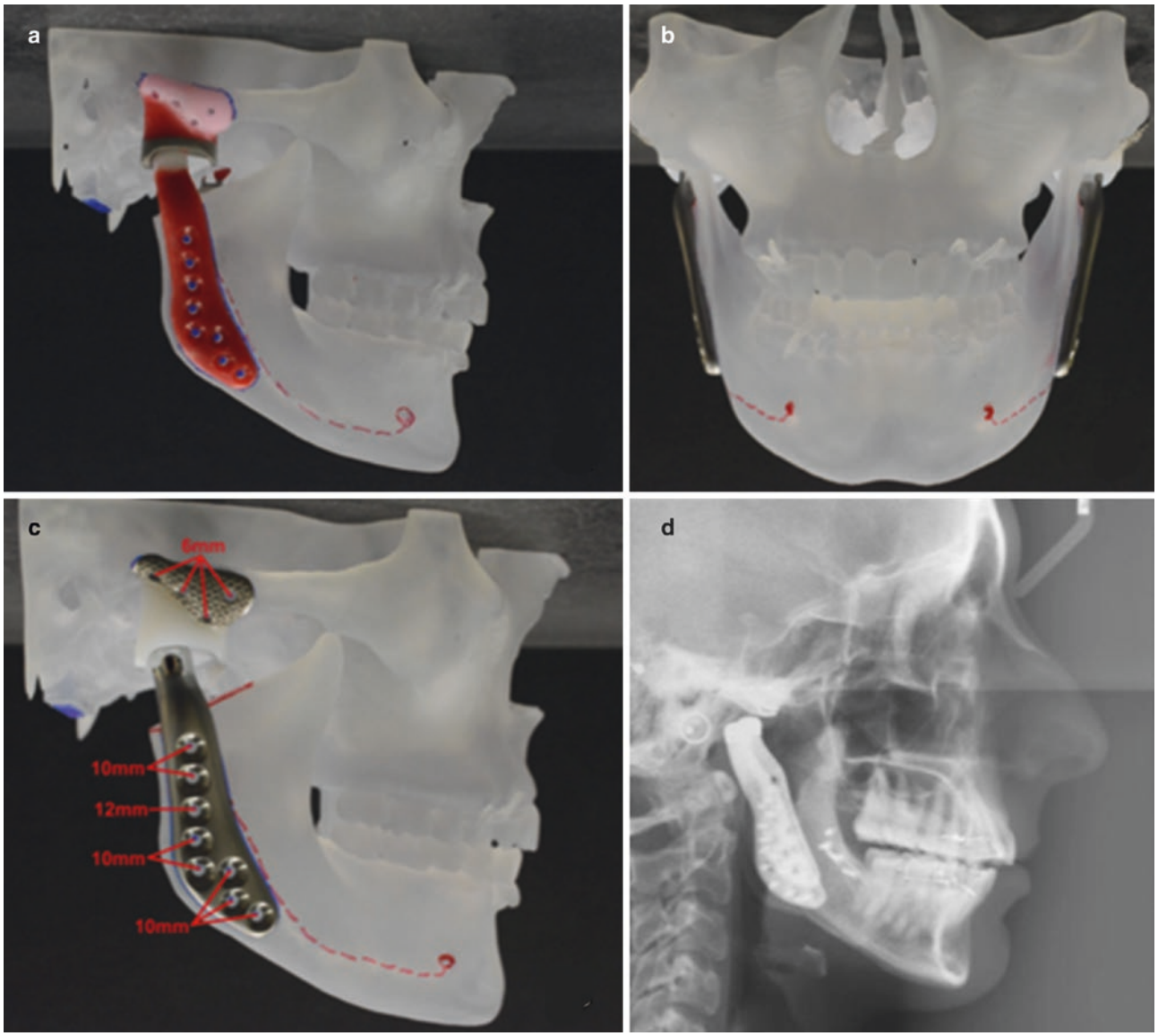

Fig. 11.8 Patient-fitted (custom) alloplastic total temporomandibular joint reconstruction. This can be used to advance the mandible and correct mandibular rotation and retrusion and may be employed in conjunction with orthognathic surgery. Prosthesis in the planning stage **a**. Coronal **b** and sagittal **c** views of the final prosthesis. Lateral cephalometric film of a patient treated for condylar resorption and subsequent retrognathia and OSA after orthognathic surgery in the form of sagittal split osteotomy **d**

resolution or stabilization of condylar resorption prior to or after surgery, and it may also be employed in the absence of a condyle as well.

Alloplastic total joint reconstruction involves replacement of both the condyle and fossa components of the temporomandibular joint (Fig. 11.8). These may be both patient-fitted (custom) and stock implants. Alloplastic joint replacement eliminates donor-site morbidity and relapse. Closure of an anterior open bite and advancement of the mandible to open the airway can also be combined with maxillary orthognathic surgery to achieve optimal jaw positioning and airway patency [16]. In addition, the alloplastic joint reconstruction is versatile, in that the surgeon may not be limited to treatment of idiopathic condylar resorption; destructive diseases of the joint including osteoarthritis and inflammatory arthropathies are readily addressed [17]. In most cases, this is the author's preferred method of reconstruction and restoration of anatomy in patients with condylar resorption and OSA together. Drawbacks to alloplastic reconstruction include risk of facial nerve injury, prosthesis infection, prosthesis failure, or sensitivity to materials. All of these are very uncommon, and the prostheses have proven quite durable over long-term studies [18–20].

Tracheostomy is a means of establishing a definitive, secure airway in patients who may be poor candidates because of failed previous surgery or medical comorbidities. It is, thankfully, rarely required for the patient with OSA and is usually avoidable in the patient with condylar resorption when mainstream reconstructive techniques are utilized.

11.6 Summary

In summary, resorption or destruction of the mandibular condyles may result from a number of different processes. These include various arthridities, trauma, and idiopathic condylar resorption. If sufficient clockwise rotation of the mandible and retrognathia combine with unfavorable soft tissue fullness or laxity in the tongue or pharynx, obstructive sleep apnea may result. Treatment of the underlying cause in these cases of OSA may involve medical management and will typically involve surgical correction. The goals of surgery are to re-establish posterior face height, establish a normal occlusion, ensure a functional temporomandibular joint, and sufficiently advance or position the maxillomandibular complex to alleviate obstructive anatomy and physiology. This may be accomplished through multiple means, including orthognathic surgery, distraction osteogenesis, condylectomy with costochondral grafting, or alloplastic joint reconstruction.

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Non-surgical Management of OSA

Contents

Positive Airway Pressure for the Treatment of Obstructive Sleep Apnea

Shalini Paruthi

Contents

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12.1 Introduction

Positive Airway Pressure (PAP) therapy is the first-line and most effective treatment for obstructive sleep apnea in adults [1]. Continuous positive airway pressure (CPAP) has been used for treatment of obstructive sleep apnea since 1981 [2]. PAP works as a pneumatic splint that maintains the patency of the upper airway during sleep. It can also be used to treat other sleep-disordered breathing such as central sleep apnea, hypoventilation, and treatment emergent central sleep apnea. PAP therapy comes in a variety of forms: continuous positive airway pressure (CPAP), auto-titrating positive airway pressure (APAP), Bilevel positive airway pressure(BPAP), and adaptive servoventilation (ASV).

CPAP machines deliver a constant pressure regardless of whether the person is awake, asleep, inhaling, or exhaling, and resting in any position. BPAP gives a set pressure during inhalation, but decreases the pressure delivered during exhalation. BPAP is typically used in persons with obstructive sleep apnea in whom CPAP is ineffective. CPAP may be ineffective in patients due to the patient requiring higher pressures (CPAP max =20cmH20, BPAP max 30 cm H20/30cmH20) or when a person is intolerant to CPAP, i.e., having difficulty exhaling. It may also be used to treat other sleep-disordered breathing such as sleep-related hypoventilation by helping create an inspiratory: expiratory gradient to help ventilate [1].

ASV is typically indicated in patients who have treatment-emergent central sleep apnea, not resolved by CPAP, BPAP, BPAP with a backup rate, or in patients with other forms of central sleep apnea such as periodic breathing.

12.2 Indications for Positive Airway Pressure

Recent guidelines from the American Academy of Sleep Medicine (AASM) recommend that treatment with PAP be based upon a diagnosis of obstructive sleep apnea established using objective testing. PAP is recommended to treat obstructive sleep apnea with patients with excessive daytime sleepiness, impaired sleep-related quality of life, and comorbid hypertension [1]. Other guidelines, including many payer policies, suggest PAP treatment for moderate to severe obstructive sleep apnea (AHI > 15) or mild sleep apnea (AHI 5–15) with symptoms of excessive daytime sleepiness, nonrestorative sleep, insomnia, neurocognitive dysfunction, or a history of mood disorder, hypertension, or cardiovascular disease. The clinical guidelines for the manual titration of PAP will not be covered in this chapter but can be found in reference [5]. Initiation of PAP can be through either home APAP trial or an in-lab PAP titration per the recent AASM Clinical Practice Guideline. Educational interventions, troubleshooting, and follow-up in person or through telemedicine are also recommended to improve adherence [1].

12.3 PAP Adherence and Compliance

Though PAP is very effective in treating obstructive sleep apnea, the main barrier is adherence to therapy. Adherence with PAP has varying definitions, but one commonly used (though arbitrary) cutoff is use of PAP therapy for over 4 hours per a night, 70% of nights. Objective adherence rates using this definition can range from 40% to 80% depending on a variety of factors [3]. One factor that predicts improved adherence includes the patient having subjective sleepiness before therapy. If patients notice improvement in sleep quality, they are more likely to use PAP long term as therapy for their obstructive sleep apnea. One important point, subjective reports of adherence have been shown to be much higher than the objectively reported usage that is available on most PAP machines [4].

12.4 Difference Between CPAP and APAP?

Within CPAP therapy, two modalities exist: fixed pressure CPAP or auto-adjusting PAP (APAP). For patients who are titrated in the lab, several pressure settings can be evaluated to determine which pressure setting best treats the patient's sleep disordered breathing and was tested in a variety of positions and stages of sleep, including supine-REM sleep. The pressure setting that best treats the sleep-disordered breathing (SDB) can then be used to set the pressure on the CPAP machine [5]. Some clinicians will choose to order an APAP machine and set the machine to a narrow range of pressures when the pressure setting that appears to work the best is known. For example, if the PSG identified a pressure setting of 8cmH20 best treated the patient's SDB in supine-REM sleep and 6cmH20 best treated SDB in lateral sleep, the CPAP machine may be set from 6 to 8cmH20. Other providers may use a wider range of settings, such as 4–12cmH20 or rarely 4–20cmH20, to allow the machine to adjust to a higher setting if needed, since patients may sleep differently at home than in the lab, or may have other factors (weight, congestion) that would create a need for a higher pressure setting in the future.

Therefore, CPAP has a fixed pressure setting and the machine will always deliver the same constant

amount of air, whether inhaling or exhaling and only at one pressure setting. On the other hand, APAP is an auto-adjusting CPAP machine, and the machine will automatically adjust (based on flow of air through the mask/tubing) within a range of pressures chosen by the clinician. Research studies show similar adherence rates and effectiveness between APAP and CPAP [1, 6]. AASM Clinical Practice Guidelines also state that either is appropriate as therapy for obstructive sleep apnea [1].

Due to some payer policies, some patients may be diagnosed and treated for sleep apnea, without ever setting foot in a sleep lab. In this case, after the home sleep apnea test confirms the presence of hypopneas and apneas, the patient is prescribed an auto-adjusting PAP machine. Care must be taken to thoroughly review the adherence report that can be downloaded from the APAP machine within the first 12 weeks or sooner if needed due to problems, to determine median pressure [1].

Examples to illustrate the importance of close follow-up: If an APAP machine is set to 4–20 cm, and the adherence report shows the median pressure is typically 6 cm of water, with the maximum pressure recorded is 20 cm, and the patient is complaining of dry mouth or leak, the pressure can be reset to a lower range of 4–8cmH20, which will likely reduce the patient opening their mouth, which was causing oral leak and dry mouth. Conversely, for a patient with an APAP machine set to 4–20, who consistently has a median pressure of 16 cm, this patient may be spending an inordinate amount of time at suboptimal pressures while the machine is following its algorithm to get up to the median pressure; thus, the patient may complain of lack of benefit or not getting enough air pressure, i.e., "air hunger" and difficulty tolerating the PAP. This machine could be reset to 14-18cm so the patient may receive a therapeutic pressure soon after turning on the machine.

Depending on the brand of machine and the model, some machines have smart phone applications (apps) that can be downloaded and used by patients. The apps use Bluetooth technology to gather data from the machine and send to a person's smartphone or tablet. During clinical visits, the adherence report generated by the PAP machine is reviewed by the patient's medical providers. Adherence Reports (ARs) provide a substantial amount of information that can be used to improve the efficacy of CPAP for a person and improve the user experience. This information is also now able to be accessed via the internet by the clinician and the durable medical equipment company. Settings may also be changed through the same technology.

Adherence reports provide information on adherence, as many insurance providers require users to wear CPAP for >4 hours for >70% of nights during a consecutive 30-night period, sometime during the initial 90-day period after receiving PAP therapy, in order for the insurance provider to continue payment for the equipment and refill of supplies. Adherence reports also provide data on machine settings, such as pressure or pressure range, ramp, pressure relief (different name per each brand), and leak. The reports can be viewed as 1-, 2-, or 7-page (or longer!) reports, and each presents a different level of detail.

A significant amount of details are found in the 7-night extended review, which can provide the clinician an opportunity to see night-to-night variability and where problems are occurring, such as if it appears the person's mouth is opening, causing intermittent leak, or the pressure range is so high that the person must use the ramp feature multiple times a night.

Adherence reports also generate a machine-based respiratory disturbance index (RDI) or apnea-hypopnea index, or how many times the machine notes changes in air flow, which would represent a person having partial or total blockage of the airway. Clinical consensus is that ideally, the machine-generated RDI goal is <5 events per hour, although if the patient clinically reports improvement and the machine-generated RDI is <10, it may be reasonable to make no further changes [5]. Additionally, each brand of machine may have slightly different algorithms to help determine this RDI as no arousal or desaturations are measured. Some reports will further describe what proportion of respiratory events are likely obstructive, central, or mixed type. This can be helpful to determine if a person struggling with CPAP or having lack of benefit may need a change in pressure or needs to be brought in for an in-lab overnight attended titration or re-titration study to determine if the problem is with the mask, tubing, leak, pressure setting, or treatment-emergent central sleep apnea, which requires treatment with a different PAP modality altogether.

12.5 Mask Options

The mask can "make or break" a patient's CPAP or APAP experience! When patients dislike their CPAP, they are often referring to their mask experience. Studies suggest the best chance for success is to use a nasal or nasal pillow-type mask initially. Nasal interfaces also seem to have better data with regard to effectiveness versus full facemasks [5]. It is believed that full facemasks can actually put pressure on the lower jaw, causing the jaw/tongue to move backward through the night, reducing airway dimensions and increasing airway resistance [5]. However, some people are unable to keep their mouth closed despite a chin-

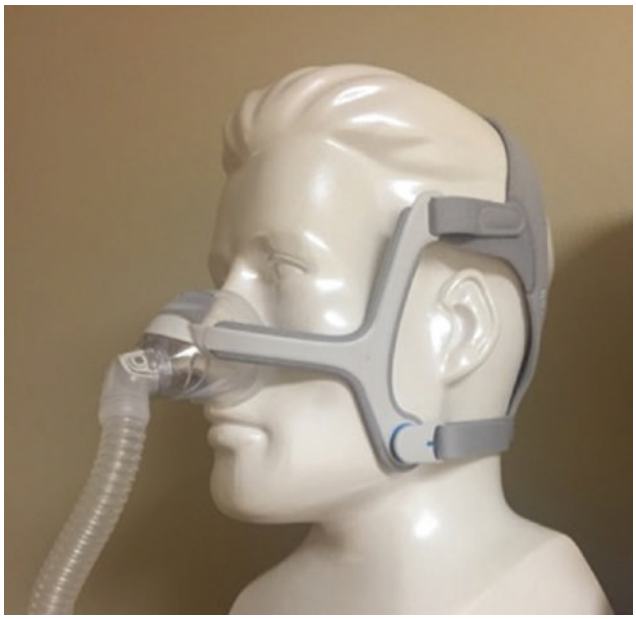

Fig. 12.1 Example of a nasal mask

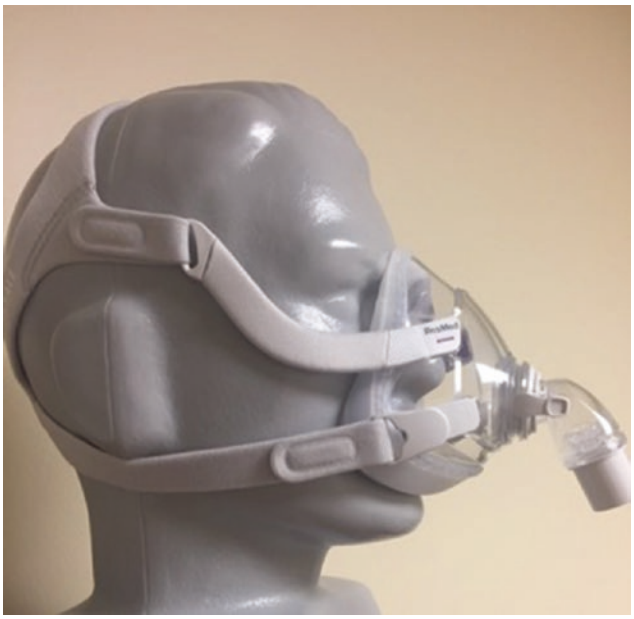

Fig. 12.2 Example of a full face or oronasal mask

strap or cannot become comfortable with the nasal or nasal pillow-type mask. Thus, there are a variety of full facemasks available as well. Masks should not leave red marks or indentations on the skin for longer than a few minutes after being removed in the mornings. Recently, manufacturers have placed a warning that masks with magnetic clips should not be used in persons with pacemakers or that the magnetic clip must stay at least 2 inches away from the pacemaker [7]. Most masks are made of silicone material so they are hypoallergenic. It is common for patients to exchange masks or get refit with masks as they are getting used to positive airway pressure. There are several different styles of masks; however, some unique styles include cloth masks or memory foam masks. See Figs. 12.1, 12.2, 12.3, and 12.4.

12.6 Possible Side Effects from PAP Therapy

12.6.1 Dry Mouth

For people who experience dry mouth by morning time, it is likely they are opening their mouth at some point during the night, experiencing oral leak. *A chinstrap* will help keep the jaw closed, reduce the ability of the mouth to open, and thus reduce leak and dry mouth discomfort. Other products to try include mouth moisturizers (artificial saliva) or xylitol tablets.

Fig. 12.3 Example of a nasal pillow-style mask

Fig. 12.4 Close-up view of the nasal pillow-style mask

It is also important to treat any nasal symptoms, such as runny nose or nasal congestion, which may have caused mouth breathing.

12.6.2 Tangled Tubing

The tubing comes in various lengths, heated or non-heated. Tubing can be ordered in lengths of 6, 8, and 10 feet. PAP tubing holders, either store-bought or home-made, can prevent tangling. For example, some people run their tubing over their headboard, while others run their tubing up above their pillow. There are also masks which the tubing connects on the superior portion of the mask headgear, thus keeping the tubing away from the face and body altogether.

12.6.3 Condensation

Heated tubing may help decrease condensation, also commonly referred to as "rain-out", within the tubing when there is significant mismatch in the bedroom air temperature (much cooler) and the temperature of the air inside the tubing (much warmer). Tubing sleeves or tubing covers are also available. See Fig. 12.5.

Fig. 12.5 Example of tubing sleeve

12.6.4 Headgear Problems

Headgear comes in a variety of sizes and is typically designed just for the mask it comes with. However, sometimes the headgear can be mixed and matched with masks other than the mask it came with. Most masks are standard with straps and clips to secure the mask in place all night despite the person's sleeping position or position changes. Some unique styles of headgear include the Bella Loops headgear, which is compatible with the Swift FX mask, and includes two straps that loop around the ears; there are no straps that go behind the head. Another unique headgear is the soft cap headgear, made mostly of spandex, which covers nearly all of the posterior surface of the head and snuggly secures the mask overnight. There are some headgears that have an extra loop built in, either on the side or the top of the headgear to help secure the tubing, again to decrease the person becoming entangled. Patients are requested to hand-wash and air-dry the headgear every 2–4 weeks to help the headgear maintain its shape and elasticity.

12.6.5 Humidifier Problems

Humidifiers have become standard on most machines. They can be adjusted from OFF (or zero) up to the highest setting offered by each brand. There is a hot metal plate on the bottom of the water container that heats up proportional to the setting selected in the settings menu. It is recommended that for regular use, dis-

tilled water is used nightly and the excess dumped out each morning, with the humidifier container allowed to air dry between uses. The benefits of distilled water include that it is mineral-free and microorganism-free due to the process of distilling water. Water is boiled, and only the evaporation is collected to create distilled water. Due to presence of microorganisms, it is not advised to use tap water or well water on a daily basis, as the air in the PAP machines pick up moisture from the humidifier chamber.

12.6.6 Ramp

The ramp feature is a patient-comfort feature. When the machine is started, or anytime during the night, with the push of a button, the air pressure can be "reset" and restarted at a previously lower set pressure. For example, for a person on an APAP machine with a range of 8–10cmH20 and a ramp start pressure of 4cmH20, each time the person desires, s/he can push the ramp button and restart the pressure at 4cmH20. This setting allows the pressure to slowly increase to the goal pressure setting, over a prespecified time period. Ramp time periods can be set from 5 to 45 minutes on most machines.

12.6.7 Cleaning Equipment

12.6.8 Skin Irritation

Skin care is an important consideration for PAP users. The mask fit should be snug, not too tight, and not too loose. Masks should fit comfortably, without leaving lasting red marks or indentations after 7 hours of continuous use. It is normal to have some indentations for a few minutes after removing the mask. For patients who get red sores or skin irritation, petroleum jelly or zinc oxide preparations can help soothe and heal the skin.

Sometimes the mask needs to be exchanged for a different style altogether. Sometimes people alternate different mask styles so that the face does not have constant pressure in the same place every night. Wraps are small cloths with Velcro designed to wrap around the PAP straps to cushion the skin to decrease the appearance of indentations. For patient who use the nasal pillow-style masks and have chafing to the skin between their nostrils, they may need to consider switching masks.

12.6.9 Nasal Congestion

Some patients will experience nasal congestion, while others will experience clearing of their nasal congestion after starting PAP therapy. Over-the-counter saline-based solutions are available to help with nasal congestion or dryness, and can be applied inside the nostril prior to using PAP. An alternative is petroleum jelly.

Additionally for persons with significant "runny" nose, prescription ipratropium bromide spray can be sprayed inside the nostrils. For people with "stuffy" nose, a nasal corticosteroid can be considered. Nasal saline rinse or squeeze bottles may be helpful. Treatment for seasonal allergies is also recommended with antihistamines or leukotriene inhibitors.

12.6.10 Aerophagia

Some patients will describe increased belching, burping, or passing gas after waking up after starting PAP therapy due to swallowing air. Treatment of this aerophagia is to decrease the machine pressure setting or pressure range until this no longer occurs, yet maintains a low RDI.

12.7 Cleaning Equipment

Cleaning the equipment is vital to equipment longevity.

The mask cushion, which can be dissembled from the headgear, should be washed with a gentle dish soap, wiped, or sanitized daily. Baby wipes or CPAP-designated wipes can ease the burden of daily cleaning to remove the natural oils, sweat, and nasal drainage that can accumulate on the mask cushion. Alcohol-containing or bleach-containing wipes should not be used. The headgear and other plastic parts of the mask should be hand-washed at least weekly. The tubing and humidifier chamber should be cleaned weekly with soap and water. After a night of use, any water remaining in the humidifier container should be emptied daily in the morning and the water container allowed to air-dry. Filters should be changed as specified in the user manual.

Additionally, for intermittent, more thorough antimicrobial cleaning, the equipment may be soaked in a vinegar mixture, typically 1 part vinegar to 5 parts water for about 15 minutes. This can be consid-

ered every few months or particularly after a person has used their PAP equipment while also suffering from an upper respiratory infection. Several chemical sanitizers or ultraviolet light sanitizer devices are available on the market for purchase to clean the machines; however, there are no data to suggest that these are more beneficial over traditional soap and water cleaning.

12.7.1 Travel Options

Travel CPAP and APAP machines are available from durable medical equipment suppliers. The machines can be used plugged in at night or used with an additional battery attachment. Optional humidifiers or humidifying mechanisms may be available. However, not every mask fits with every travel machine. The tubing is typically less wide and less heavy, so it pulls on the mask less. Heated tubing is currently not available. Currently, some travel machines can provide adherence reports.

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Oral Appliance Therapy

Marie Marklund

Contents

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13.1 Introduction

Oral appliances (OAs) aim to increase the upper airway size, facilitate breathing during the night, and reduce obstructive sleep apnea (OSA) and snoring (Fig. 13.1a, b). There are two subgroups of OAs: the mandibular advancement device (OA_M) (Fig. 13.2) and the tongue-retaining device (OA_T) (Fig. 13.3). OA_M is attached to the teeth and holds the mandible forward during sleep. This treatment has the highest level of evidence among non-PAP therapies [1–4]. OA_T aims to hold the tongue forward into an anterior bulb by suction and can be used irrespective of the presence of teeth. Both devices reduce sleep apneas, but OA_M is better tolerated and has been much more studied.

This chapter primarily describes OA_M treatment; the effects, the side effects, and the longer term outcomes. The efficacy of OA is more variable than that of PAP, since its mechanism of action depends on a number of factors such as the degree of mandibular or tongue advancement and OSA endotype [5]. OA_M can be used solely or in combination with other sleep apnea treatments such as positive airway pressure (PAP) or positional therapy, when the efficacy of OA alone is insufficient. The adverse effects of OAs are generally mild, but in the longer term, there are risks of tooth

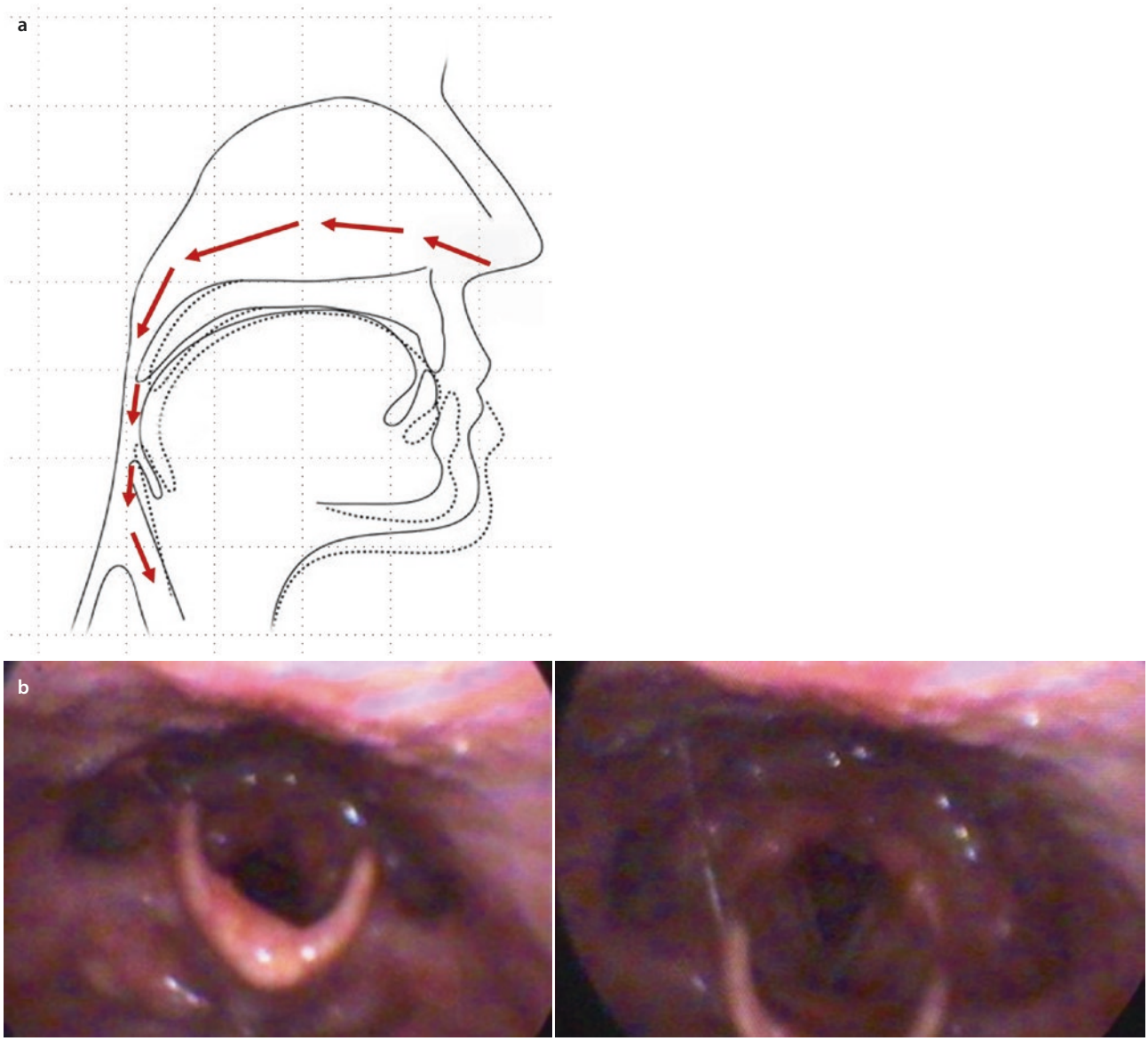

Fig. 13.1 **a** Illustration of the mechanism of the oral appliance. **b** Photo without (left) and with (right) the oral appliance showing the widening of the upper airway with the appliance in place compared to without it

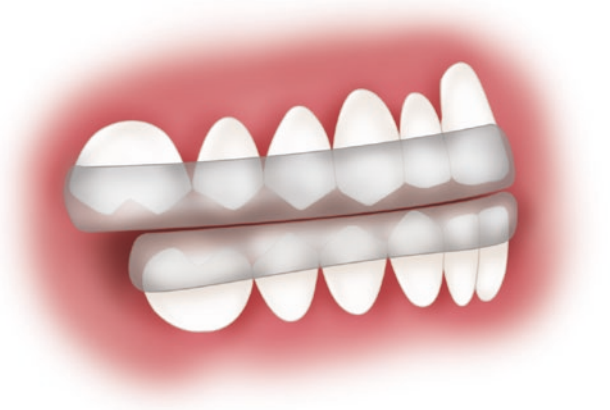

Fig. 13.2 Schematic illustration of the OA_M

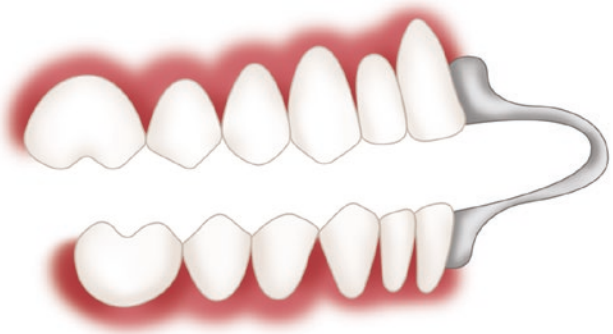

Fig. 13.3 Schematic illustration of the OA_T

movements from the forces of these devices. OA treatment must be continuously followed-up regarding efficacy, side effects, and adherence.

13.2 Terminology

OA is a generic term for devices that are inserted into the mouth in order to modify the position of the tongue and other structures to reduce snoring and sleep apnea [6]. The oral appliance that holds the mandible anteriorly, the OA_M is also termed "mandibular advancement device (MAD)," "mandibular advancement splint (MAS)," "mandibular repositioning appliance (MRA)," "unterkieferprotusionsschiene (UPS)," and "orthèses d'avancée mandibulaire (OAM)." A device that holds the tongue forward, OA_T, is named "tongue-retaining device (TRD)" or "tongue-stabilizing device (TSD)." There are combinations of OA_M and OA_T including both mechanisms. Most commonly, the term OA is used synonymously for OA_M, since this type of OA has become overwhelmingly most common.

13.3 Devices That Hold the Mandible Anteriorly—OA_M

13.3.1 Mechanism of Action

OA_M repositions the lower jaw anteriorly and slightly opened in order to increase the upper airway volume and reduce the pharyngeal collapsibility (Fig. 13.1a) [7–10]. The upper airway is enlarged, particularly in its lateral dimension at the velopharyngeal level (Fig. 13.1b), and the tongue is displaced anteriorly [8, 9, 11, 12]. The mechanism of action of OA_M is variably efficient depending on a number of factors such as OSA pathogenesis and OA design. To various degrees, OSA patients may have some degree of anatomical compromise in their upper airway, a reduced dilator muscular activity, an increased pharyngeal collapsibility, an overly sensitive ventilatory control system (high loop gain), or a low arousal threshold [13]. OA_M compensates for individual deficiencies in upper airway anatomy. Non-anatomical traits such as a high loop gain or a low arousal threshold are unchanged by OA_M [14]. Patients with mild OSA are generally better suited for the mechanism of OA_M compared with patients with more severe OSA, since mild OSA patients often have less collapsible airways [13, 14]. Pharynx has also been found to widen more, along its whole length in mild OSA patients, compared with patients with more severe disease [15].

In summary, the mechanism of action of OA_M is less effective compared with PAP that increases the airflow in the upper airway in relation to the needs of each patient. This means that the indications of OA_M are more difficult to assess and that the treatment has to be more rigorously controlled and followed up compared with PAP.

13.3.2 Device Designs

There are many various designs of OA_M (Fig. 13.4a–h), and they may be subdivided in various ways. There are custom-made devices and prefabricated ones. The evidence for the efficacy of OA_M therapy is primarily based on the results of custom-made devices. The knowledge about custom-made devices will therefore constitute the major part of this chapter.

OA_{Ms} have various types of adjustment mechanisms between the jaws in order to facilitate changes in jaw positioning and improve the efficacy and tolerability of the device [16–19]. The adjustment mechanism can be located either laterally on each side of the jaws (Fig. 13.4a–d) or in the midline (Fig. 13.4e–f). These mechanisms are primarily intended for anterior-posterior adjustments of the lower jaw during the titration procedure when the optimal jaw position is

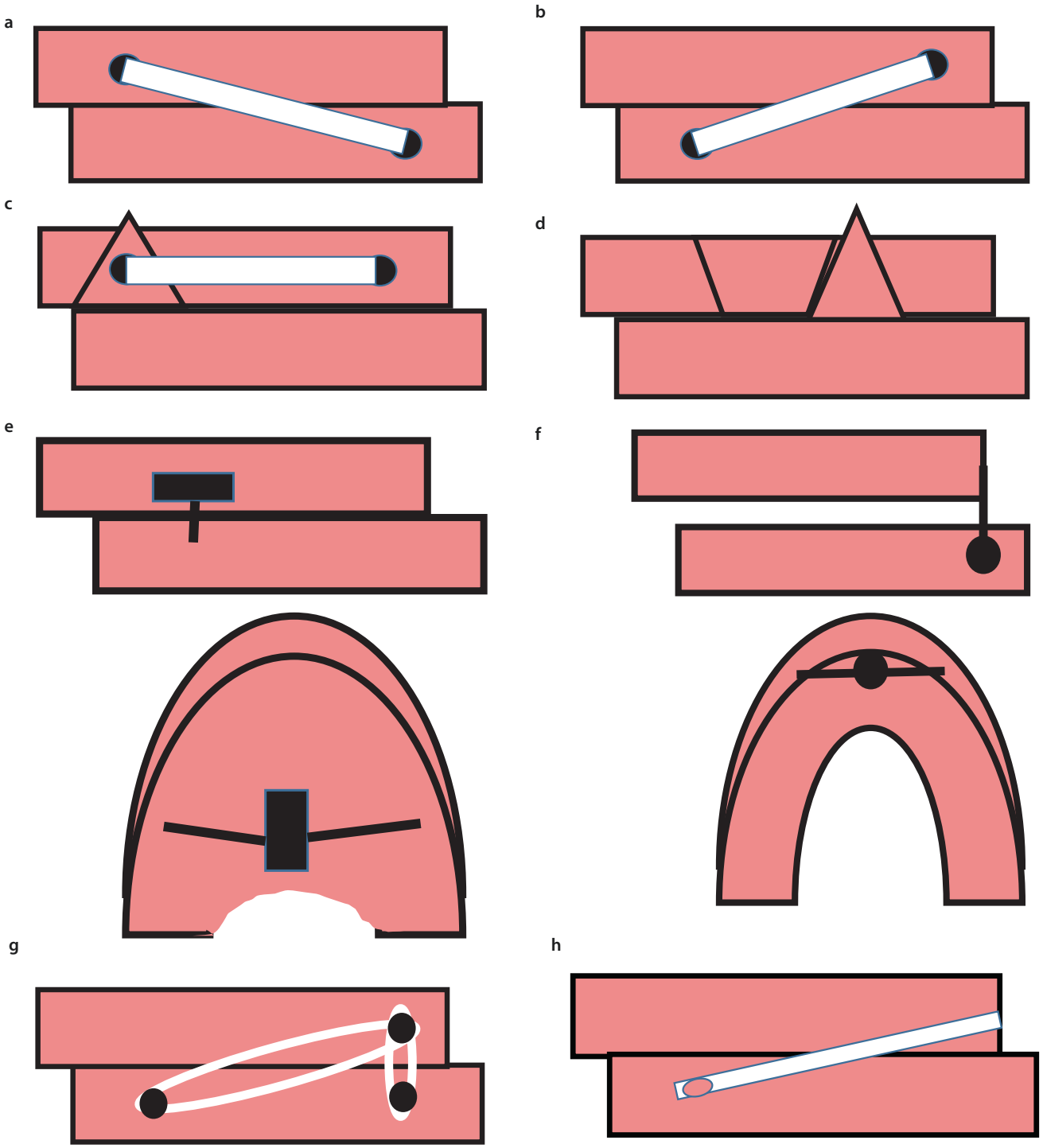

Fig. 13.4 **a**–**g** Various designs of adjustable OA_M. The adjustment mechanism of the device may be located laterally **a**–**d** or in the midline **e**–**f**. Some appliances allow mouth opening **a**–**e**. Elastic bands can be applied in different ways in order to hold the jaws together and certify the degree of advancement **g**. A new device introduces a combination of lateral and midline mechanism **h**

determined. During use, the jaws can be fixed firmly together by the mechanism (Fig. 13.4f). More or less mouth opening can also be allowed with other types of mechanisms (Fig. 13.4a–e). This mouth opening can be prevented by the use of elastic bands (Fig. 13.4g).

Lateral adjustment or some movement in the lateral dimension is possible in several designs. The earliest types of OA_M were made in one piece and lacked this adjustment mechanism. Titration of mandibular positioning was more difficult, since it required new con-

struction bites and help from a dental technician to remake the device with the lower jaw in a new position.

An opening within the appliance to allow mouth breathing might be important for many patients, since nocturnal nasal obstruction is reported by one third of sleep apnea patients [20]. Still, it is essential that the appliance allows the lips to close in order to primarily promote nasal breathing.

In summary, custom-made oral appliances that allow titration of the lower jaw position are recommended. These appliances are most commonly used in clinical practice, since the adjustment mechanism is considered to be important for the efficacy and tolerance of the device.

13.4 Methodology

Good oral health is essential for treatment success with OA_M and it is therefore important with a complete odontological investigation including dental occlusion, jaw movements, and the temporomandibular joint. Patients with poor oral health should be treated for these conditions before OA_M therapy is initiated. Existing odontological diseases will increase the risk of further impaired oral health, side effects, and a poor treatment outcome. If the teeth are unsuited to hold the lower jaw forward, the mechanism of the device is also jeopardized.

OA_M therapy starts with impressions or intraoral scanning of the teeth. Thereafter, a bite registration in an advanced mandibular position is taken in wax or other material. This registration is advised to be taken with the mandible advanced straight forward and approximately 50% forward of maximum protrusive capacity or 4–6 mm compared with centric relation. In patients with a poor protrusive capacity, some increase in protrusive capacity might be expected during the first months' of treatment [21]. A bite fork or a steel sliding caliper can be used to help the patient find this position. After fabrication and adaptation of the device to the teeth of the individual patient, a test period starts with acclimatization and titration of the lower jaw forward. This means that the lower jaw is continuously advanced in steps of in between 0.1 and 1 mm until an effective mandibular positioning is identified. A renewed sleep apnea recording has to be performed in order to verify the treatment outcome of OA_M on breathing stops and oxygenation. Such recordings must be repeated until an effective mandibular position is found, particularly in patients with moderate to severe disease or comorbidities. Intermediate testing during the titration procedure can be performed at the dentist's office, depending on the health-care system in each country. The final decision about the efficacy of the device in relation to the individual patient's health is made by the referring sleep physician. In the future, this titration procedure may be simplified by an overnight testing of the optimal mandibular positioning at home [22] or by the use of auto adapting devices [23].

In summary, OA_M therapy requires good oral health, a time for adaptation and titration of an optimal jaw positioning, and confirmation of the efficacy of the treatment in renewed sleep apnea recordings.

13.5 Short-Term Effects on AHI

OA_M reduces AHI effectively compared with placebo interventions or untreated controls [24–37] (Fig. 13.5). PAP is more effective than OA_M in reducing AHI, according to studies comprising patients with varying disease severity [24, 25, 32, 38–45] (Fig. 13.6). Among the mildest OSA patients, this difference becomes smaller or is levelled out [3, 24, 42, 46]. The nightly oxygenation is improved with OA_M [2, 3], but PAP restores the nightly oxygenation even further [2].

13.6 Definitions of Treatment Success

There are several definitions of treatment success for OA_M therapy [47]. These are based either on strict cut-off points, such as an AHI of below 5 or 10 or a percentage cut-off, usually a 50% or more reduction in AHI. Combinations of criteria are also used, such as an AHI <5 + the resolution of symptoms or AHI <5 or 10 + 50% or more reduction in AHI. The last criterion assures a sufficient AHI reduction also among the mildest cases.

Complete responders are defined by an AHI <5, often with an additional requirement of 50% reduction or more in AHI, non-responders have less than 50% reduction in AHI and a treated AHI of above a specific level, such as 20, and partial responders lie in between [28, 29, 41–43].

The proportion of patients who receive complete success with an AHI <5 sometimes with an additional requirement of ≥50% reduction in AHI varies in between 10% and 57% in randomized controlled trials including patients of varying disease severity [26–29, 34–36, 38, 41–43]. The reason for this large variability in response to OA_M therapy depends on factors such as disease severity, patients' phenotype, definitions of sleep-disordered breathing events, and methodology including device design.

In summary, there is a high level of evidence of a satisfactory AHI reduction by OA_M. The variability in treatment response means that PAP will represent a more efficient alternative, particularly among the more severe OSA patients.

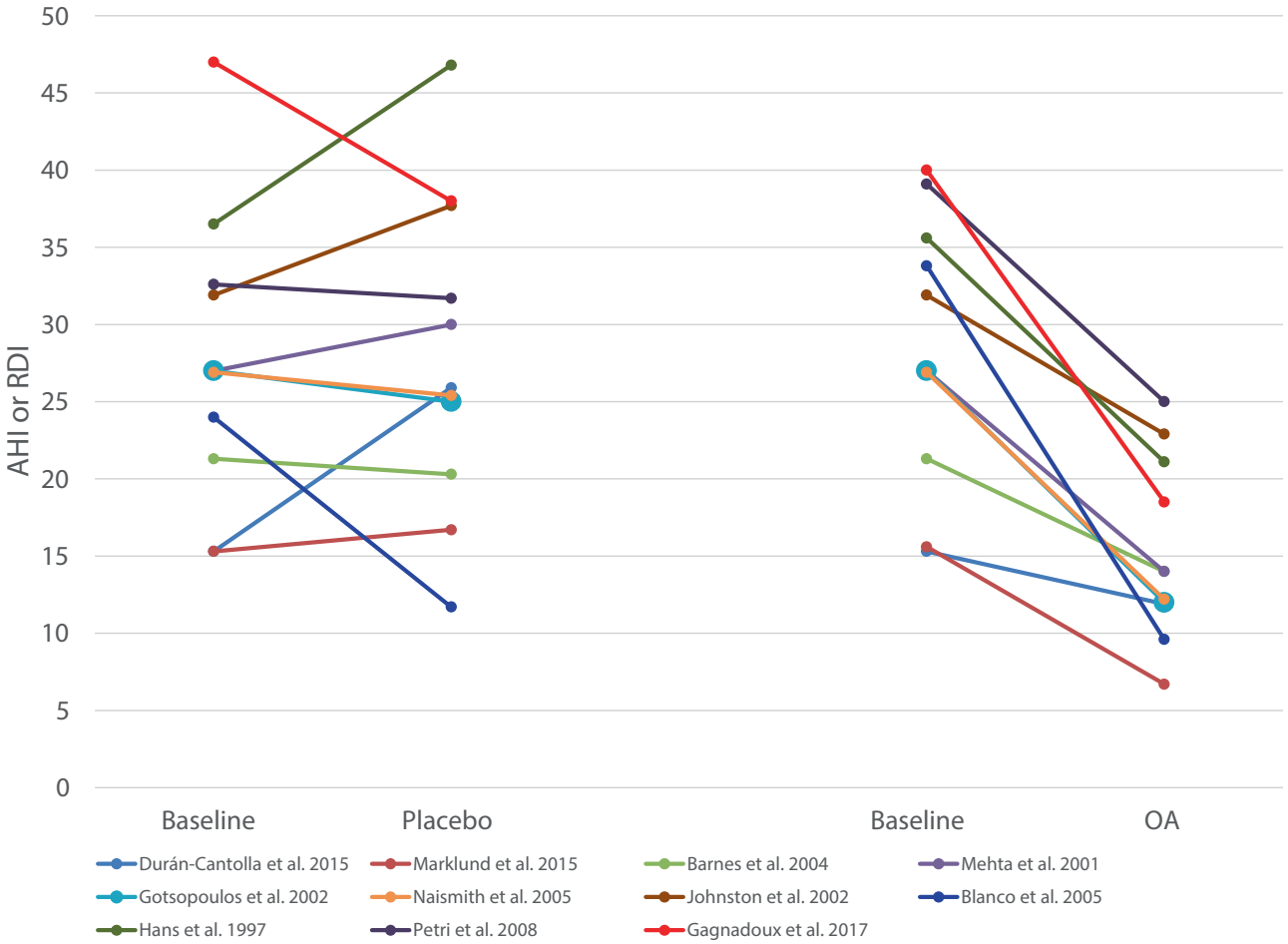

Fig. 13.5 Effect of OA_M compared with baseline and placebo

13.7 Factors Related to the Efficacy of OA_M

13.7.1 Importance of Mandibular Repositioning

A larger advancement of the mandible will generally produce a higher efficacy of OA_M [7, 48], but there is no exact linear relationship between mandibular advancement and treatment success [49, 50]. In addition, the needed advancement might be influenced by disease severity or other individual factors [51]. Despite this, an insufficient capacity of the patient to move the mandible forward to some degree will limit the possibilities to receive an optimal treatment outcome with OA_M therapy. A device that is produced with the mandible extensively opened is likely to produce a poorer result [52] compared with a device that is constructed within a more limited range of mandibular opening [53, 54]. Too wide openings over a centimeter have also been related to discomfort for the patient [54].

13.7.2 Device Design

Comparison between different devices regarding their efficacy in reducing AHI have been made in 13 randomized controlled trials [37, 54–65] and four non-randomized studies [66–69]. Nine of the 13 randomized studies compared various custom-made designs [54–58, 60–62, 64, 65] and showed no or only small differences. Two studies compared a custom-made device versus a prefabricated one [59, 63] and both favored the custom-made designs. Complete treatment response was found in 49% [63] and 64% [59] of the patients when they used a custom-made design and in only 17% and 24%, respectively, when they were treated with a prefabricated device. This result is further supported by results of a non-randomized study [66]. The prefabricated device was also unreliable as test device to find responders to OA_M according to one study [63]. In addition, the results from a large study including various degrees of customized devices showed fairly small AHI reductions

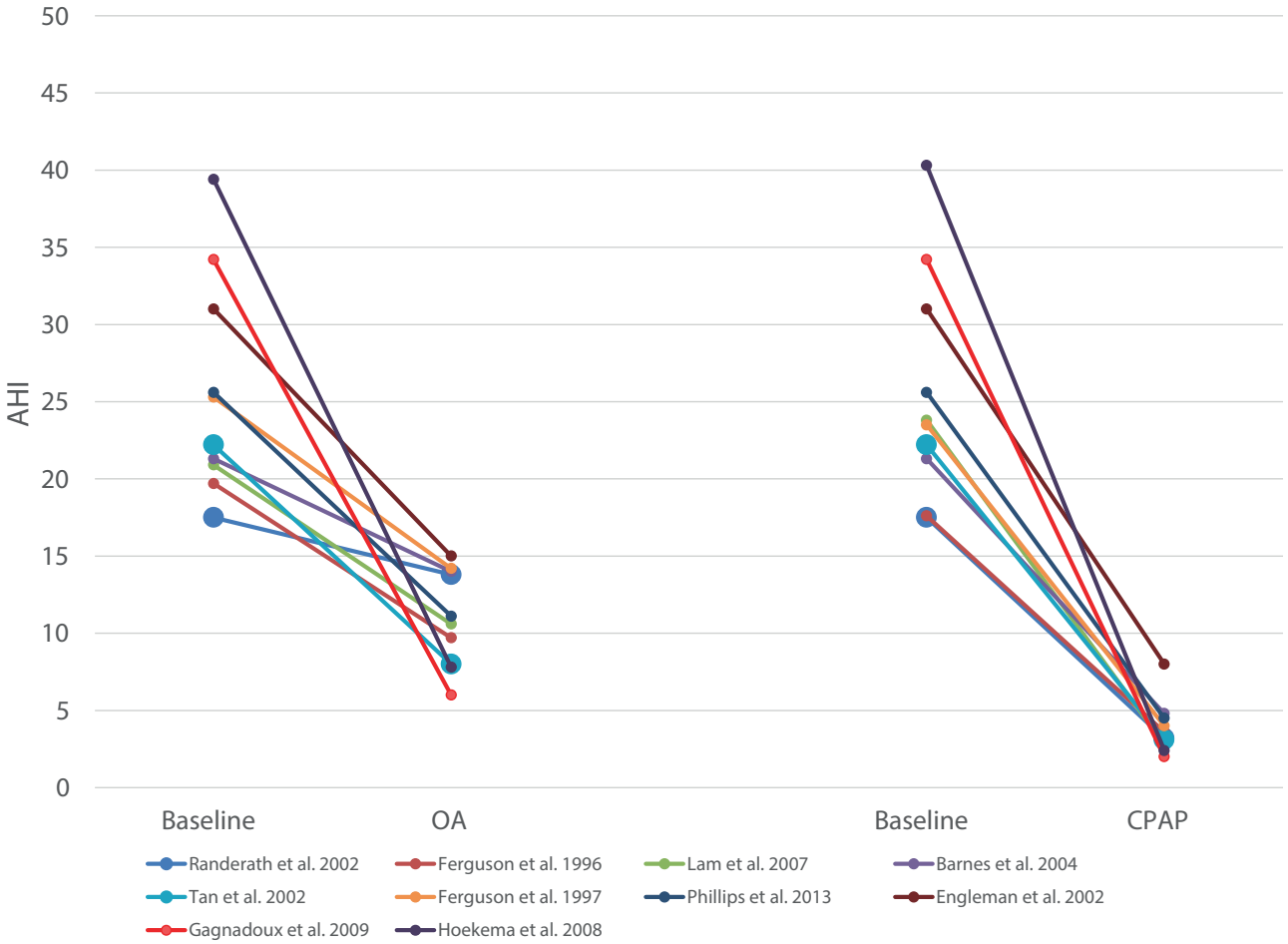

Fig. 13.6 Effect of OA_M compared with baseline and PAP

by non-customized designs, although they did not differ significantly [37]. The retention of non-customized designs might be one explanation to their poor efficacy [70], although the properties of the materials in these devices are improving [69, 71].

Adjustable devices where one of them fixate the lower jaw to the upper jaw and the other one allowed mouth opening were compared in seven randomized studies [55–58, 62, 64, 65]. Six of them favored the fixed design, four significantly [55, 58, 62, 64]. Further support for these findings is presented in one non-randomized study [67] and two studies comparing the influence of elastic bands that hold the jaws together in devices that allows mouth opening [61, 68]. Based on these studies as well as some observational ones [18, 19, 67, 72], adjustable, custom-made devices that fixate the lower jaw to the upper are most effective.

13.7.3 Determining the Optimal Mandibular Position and Find Responders to OA_M Therapy

New technology can be used to find the optimal mandibular position and predict which patients that are likely to respond to the treatment. A feedback-controlled mandibular positioner has been developed and tested during un-attended in-home conditions [22]. This method is a further step forward and a cheaper alternative compared with the previous system using a remotely controlled mandibuilar advancement device during an attended in-hospital testing night [73–75]. The developed testing method produced a high potential of predicting treatment success defined as an oxygen desaturation index (ODI) of less than 10 with a sensitivity 85% and a speci-

ficity of 93% [22]. The predicted degree of mandibular advancement was effective in 86% of the cases according to a later prospective test in a new sample [22]. Another study described a novel auto-adjusting OA_M, which could be used for predictions as well as to continuously optimize jaw positioning in order to secure an effective apnea reduction [23]. These two new methods will be of great help to find the responders to OA_M treatment as well as reduce the time and costs for titration of mandibular positioning, provided that the equipment becomes clinically available and has a reasonable price.

13.7.4 Pharyngeal Anatomy and Physiology

Treatment success with OA_M is more likely in patients who have a verified widening of the upper airway during a manipulated mandibular advancement procedure [76]. Drug-induced sleep endoscopy (DISE) can be used to visualize such effects. In one study, a simulation bite in a maximal comfortable advanced mandibular position was used [77]. One hundred and thirty-five patients with a mean AHI of 21 who were referred for OA_M therapy were studied. Those who had complete resolution of pharyngeal obstruction with the simulation bite were more likely to get treatment success with OA_M with an odds ratio of 5, compared with patients who only had partial or no effect on their pharyngeal collapse with this bite registration. In another study using DISE, 28 patients with mild to moderate OSA were studied [78]. The anesthesiologist moved the mandible 4–5 mm forward and observed the effects on snoring and apneas. Prediction was based on observations that the obstructive events were less frequent or were eliminated together with an improved upper airway patency of at least 50% at one or more sites during 3 minutes. This method predicted success in 71% of the patients, defined as an AHI <5 or an AHI reduction of ≥50%.

Endoscopy during wakefulness has also been used to test if the pharyngeal response to mandibular advancement could be used for prediction purposes [12, 79]. A study, including 36 severe OSA patients, reports that patients with a verified widening at the velopharyngeal level in supine position predicted success with OA_M, irrespective if the expansion of the airway occurred in the antero-posterior direction or the lateral direction [12]. These findings were later confirmed in a larger sample of 61 patients with moderate to severe OSA, where a widening at the velopharyngeal level was an independent predictor of success together with a low AHI in a model controlling for age and body mass index (BMI = kg/m^2) [79]. Several criteria for treatment success with OA_M were tested, and the ORs varied in between 1.4 and 7.3 for this predictor. A more recent study finds that it is primarily the effect of OA_M on pharyngeal collapsibility, which may be predicted during awake endoscopy [80].

A new way of analyzing airflow curves in order to localize the site of collapse into tongue-related, isolated palatal, lateral walls, or epiglottis has been developed [81, 82]. This technique can be of help to predict which patients will respond to OA_M therapy, since a tongue-related collapse has been associated with OA_M treatment success [83–85], and persistent collapse at velopharynx and epiglottis and deeper events have been related to failure [86, 87].

13.7.5 Non-anatomical Traits

Patients with milder OSA have less collapsible upper airways. They might, however, differ in the occurrence of non-anatomical traits that may cause upper airway collapse [5, 13]. Such factors can therefore be used to predict treatment response with OA_M therapy. Patients with less collapsible upper airways and a less sensitive ventilatory control system are therefore more likely to benefit from OA_M therapy [13, 14, 88]. In contrast, those with an overly sensitive ventilator control system (high loop gain) and a low arousal threshold, which are factors that are uninfluenced by OA_M therapy, are less likely to be responders.

13.7.6 Disease Severity

Patients with milder OSA are generally considered to have a greater chance to get a sufficient AHI reduction with OA_M therapy compared with patients with more severe disease. The success rate is fairly equal between OA and PAP [3, 42, 46] in these patients with mild OSA. The exact indications and large night-to-night variability in AHI represent uncertainties in this group of patients [89–91]. Patients might have a more severe disease than the sleep apnea recording indicates and risk a suboptimal treatment or no treatment.

Some patients with severe OSA can be successfully treated with OA_M [42], but PAP is more effective [3]. OAs are therefore regarded a second-line treatment in PAP-intolerant patients [3, 92].

13.7.7 Supine Dependency

A change from supine to lateral sleep position will result in markedly improved upper airway dimensions when the tongue base and the larynx relocate to more favorable positions [93]. The collapsibility of the upper airway is reduced, and breathing stops occur less frequently in the lateral sleep position compared with the supine sleep position [94]. Some patients are highly supine position dependent [95]. These patients with primarily supine

sleep apneas have been found to have more normal upper airway anatomy, with a wide airway in the lateral dimension compared with patients with co-existing non-supine sleep apneas who have lateral pharyngeal narrowing [96]. The position dependency is mainly regulated by a tendency of the lateral walls to collapse [93], but may also arise from a posteriorly located tongue [97].

There are several definitions of supine dependency such as a high AHI supine and normal value non-supine, a doubled frequency supine versus non-supine or definitions that are specifically designed to be of help for treatment decisions [98]. Depending on the exact definition, the prevalence of supine dependency varies in between 20% and 60% among OSA patients [99].

Supine dependency has been identified as a predictor of success for OA_M therapy [100–103] with similar efficacy as for PAP [102], although the results vary [99, 104]. The conflicting results regarding these predictors of success can be explained by device design. A non-randomized study report almost four times increased responder rate controlled for baseline characteristics with the use of elastic bands in a device that allowed mouth opening in patients with positional OSA [68]. The efficacy of an OA_M that allows mouth opening during sleep is also lower than that of a fixed device [55–58, 62, 64, 67] (Fig. 13.4). In experiments on rabbits, prevention of mouth opening in supine position improves the effect of mandibular advancement on upper airway resistance [105]. The fixation of the lower jaw to the upper jaw to guarantee the intended forward repositioning is probably important, particularly in the supine position in order to optimize the efficacy of the device [61]. Prediction of the efficacy of devices that allows mouth opening might therefore be unreliable compared with prediction of the efficacy of appliances that fixate the lower jaw during sleep.

13.7.8 Anthropometric Variables from the Clinical Examinations

Younger and leaner patients or females have been identified to have a higher chance to receive treatment success with OA_M therapy [18, 46, 101, 106, 107], although with variable strength. Such predictors are generally less useful in clinical practice, since the majority of OSA patients are older and men and many are obese [108, 109].

A combination of variables reflecting increased oropharyngeal crowding with the use of the Mallampati score and BMI has been related to a large risk of failure with OA_M [110]. Moreover, the patients should be informed about the risks with weight increase during OA_M treatment, since this might reduce the efficacy of the device [101]. Weight gain has also been related to increased severity of obstructive events, which might further risk the patients' health [111].

Evaluation of craniofacial morphology in cephalometric measurements has shown that isolated variables or combinations of such measurements are inconsistent predictors of treatment success for OA_M [112].

Few studies have specifically evaluated the influence of age on the treatment outcome. This might be particularly important in the large geriatric population. One retrospective study indicates that elderly people may be satisfactorily treated with OA_M [113], but there is also some skepticism [114]. Adherence might be low according to one of these studies [114].

Patients who have had a trial with PAP can be assessed if they are suitable for OA_M therapy based on the PAP pressures required in order to successfully improve their airway patency. In this way, a low PAP pressure has been found to predict treatment success with OA_M [115–117].

In summary, many different factors will influence the chance of treatment success with OA_M, where the degree of mandibular advancement of the device and patient characteristics are important factors. Overnight titration procedures represent the most promising prediction alternative for the future. A verified widening of the upper airway dimension can also be used to calculate the chance of treatment success. In the future, airflow curves might be useful to predict the type of pharyngeal collapse and treatment success. A custom-made, adjustable OA_M that prevents mouth opening is primarily recommended. Prefabricated devices often have poor retention. More knowledge is needed about predictors of success and the influence of OA_M design on the outcome on OSA.

13.8 Effects of OAs on Snoring and the Upper Airway Resistance Syndrome

Reduced or eliminated snoring is an important outcome for many patients. Many studies evaluate subjective reports and find that snoring is reduced by OA_M [2, 33, 118]. Objective measurement confirm that snoring is reduced, but usually not eliminated by OA_M [29, 34]. A few studies that compare device designs have included measurements of snoring. Two studies report more snoring with devices that allow mouth opening compared with fixed devices [57, 58]. OA_M is more effective in reducing snoring than an intraoral placebo device [24, 29, 33, 34], while PAP is more effective than OA_M [39–41]. Persistent snoring during OA_M treatment has been related to insufficient apnea control and poor adherence to treatment [40, 119].

OA_M can also be used in patients who suffer from the upper airway resistance syndrome, defined by daytime

sleepiness with increased respiratory effort, but without increased AHI [120]. The respiratory disturbance index, arousal index, and severity of depression symptoms decreased and sleep quality improved with OA_M in this group of patients.

In summary, less research exists on treatment effects of OAs on snoring compared with effects on sleep apneas, despite that many patients are primarily interested in how to best eliminate snoring and its negative consequences on family life [121]. Research about why and how we sleep together, which is of importance in terms of snoring treatments, is emerging [122]. The limited research interest in snoring might be explained by difficulty measuring sounds and that this symptom has been less related to longer term negative health outcomes [108].

13.9 Symptomatic Effects of OAs

Excessive daytime sleepiness affects a fraction of sleep apnea patients in the population [108]. Still, sleepiness is a common reason for many patients to seek treatment for their snoring problem. Excessive daytime sleepiness is usually assessed by the ESS score where patients report the likelihood of falling asleep in eight different situations using a scale from 0 to 3 [123]. The total score varies in between 0 and 24, where a score of more than 10 is defined as excessive daytime sleepiness. The degree of excessive daytime sleepiness defined in this way correlates poorly with AHI [108, 124–126]. A variety of symptoms have been related to OSA according to the International Classification of Sleep Disorders (ICSD-3) [127]. The inclusion of more symptoms than daytime sleepiness in the sleep apnea diagnosis highly increases the prevalence of obstructive sleep apnea syndrome [128]. Examples of such symptoms include nonrestorative sleep, fatigue, insomnia symptoms, nightly choking, as well as concomitant sleep apnea–related diseases such as hypertension and coronary artery disease.

Symptomatic effects of OSA treatments have mostly focused on daytime sleepiness measured by the ESS score. In subjects within the mildest spectrum of disease severity, effects from OSA treatments on daytime sleepiness are uncertain [3, 27, 33, 129, 130]. Patients often report lower ESS scores with an OA compared with untreated conditions [2, 3], but some of these can be explained by placebo effects [24, 28, 29, 33, 36]. Only one cross-over study [29] of nine randomized controlled trials found a significant difference in the ESS score between OA_M and a placebo device [24, 26–28, 30, 31, 33, 36]. None of the six parallel RCT studies reported a significantly lower ESS score with OA_M compared with a placebo device [24, 26, 28, 30, 33, 36]. Milder OSA patients, that is, those with primarily indications for OA_M therapy were included in two of these studies with no effect on the ESS score compared with placebo devices [27, 33]. One of these two studies found no effect on prospective reports of daytime sleepiness measured by the Karolinska Sleepiness Scale, objective tests of sleepiness by the Osler test or quality of life [33]. A meta-analysis reports no effect on the ESS score from OA_M compared with placebo and no difference between OA_M and PAP in patients with moderate sleep apnea [3]. There were no published randomized controlled studies of mild OSA patients when that meta-analysis was published. Most likely, a number of other causes to daytime sleepiness exist in patients with milder OSA. This might explain why as much as one third of patients who are treated with either PAP or OA_M are still sleepy, defined as an ESS score of above 10, despite a successful sleep apnea reduction [131, 132]. In patients with more severe disease, OA_M reduces daytime sleepiness [3], although to a lesser degree than PAP does. Other measures than the ESS score might also be used in future studies to evaluate subjective complaints, such as fatigue, which gave interesting results in one study [133].

Few other OSA-related symptoms than daytime sleepiness have been systematically evaluated regarding treatment effect from OA_M. Positive effect on symptoms of restless legs by OA_M treatment versus placebo interventions has been found in two studies [33, 134]. Some other OSA-related symptoms, such as headaches, nasal congestion, and insomnia, were improved with OA_M treatment compared with untreated conditions in an RCT, although there was no difference to a placebo device [33]. Many symptoms, such as insomnia, daytime sleepiness, headaches, and restless legs, may coexist with OSA and might require individualized treatment approaches [135–137]. The difference in expression of symptoms between OSA patients is exemplified in a study that subdivided the results of the polysomnographic sleep recordings. OSA patients who were registered to have primarily sleep arousals more often continued with OA_M treatment compared with those who had primarily desaturations [138]. More studies of various phenotypes of obstructive sleep apnea patients are therefore needed in order to better understand the symptomatic effects from various treatments of this multiclausal and multifaceted disease. A good approach in patients with mild symptomatic OSA is to test if treatment with PAP reduces daytime symptoms before treatment with OA_M is initiated [90].

In summary, the effect of OA_M on daytime sleepiness is uncertain in patients with mild to moderate OSA, that is, the group of patients for whom this type of therapy is primarily recommended. Mild to moderate OSA probably causes less pronounced daytime sleepiness than previously assumed, while there are a number of other plausible causes to their daytime sleepiness. Patients with severe disease are likely to become less sleepy with

OA_M treatment, although PAP is more effective. Future studies are needed of various symptomatic effects of OA_M treatment in relation to phenotype and maybe unknown outcomes on sleep of OSA treatment, such as on the glymphatic system [139].

13.10 Cardiovascular Effects

Blood pressure is reduced from OA_M treatment compared with placebo interventions according to meta-analyses [2, 140–142]. The effect is similar between OA_M and PAP in the studied samples [2, 140]. One meta-analysis that subdivides between APAP, CPAP, OA_M and control favored CPAP in terms of blood pressure effects [141]. The results of OA_M on blood pressure derive from randomized controlled trials comparing with placebo [25, 28, 143–146], PAP [25, 43, 144, 147, 148], untreated controls [37, 147], or in a comparison between OA_M designs [55]. In between 12 to 108 mild to severe OSA patients completed these studies that lasted in between 1 and 4 months. All but one study included patients irrespective of initial blood pressure, while one study required baseline hypertension [143]. In addition, there are six descriptive studies of blood pressure effects from OA_M [149–153]. The reduction in blood pressure by OA_M was significantly associated with a reduction in AHI [143, 151, 152]. Several of the studies show that the blood pressure effects are particularly evident in hypertensive patients [43, 143, 150–152]. One study subdivided women and men and found effects on blood pressure only in women and at night [146]. Further studies are needed on possible differences between women and men regarding effects from sleep apnea treatments, since previous samples have included a majority of men, 80% in average.

OA_M has also been found to normalize the nightly dips in blood pressure [25] or give a better outcome than CPAP [144] in the studied samples. Beneficial effect on endothelial reactivity was found in one small study [148], but the result was not confirmed in a larger randomized controlled trial in severe OSA patients [28]. A small, descriptive study reports similar mortality rate in patients treated with OA_M or PAP as in healthy controls, while untreated severe OSA patients had a higher mortality rate than the other groups [154].

A higher adherence to OA_M treatment than with PAP may, to some extent, explain the fairly similar outcomes on blood pressure from these treatments [43]. It is also possible, that patients included in studies regarding OA_M treatment, are healthier than patients treated with PAP, particularly those with more severe OSA. A recent RCT comparing OA_M with a placebo device in patients with severe OSA, did not, as a secondary aim show any effect on blood-pressure despite good compliance and a substantial reduction in AHI [28]. This study highlights the complexity of OSA and possibilities to reduce cardiovascular risks with interventions. On the positive side, two studies report beneficial long-term effects of OA treatment on blood pressure after 3 years or more [149, 155].

In summary, some positive effects on cardiovascular health has been found as a result of OA_M therapy. More research is, however, needed on treatment outcomes in this complex area of multiple illnesses.

13.11 Side Effects

The introduction of a foreign body into the mouth that aims to stabilize the lower jaw forward during sleep may cause discomfort with pressure on the teeth, tenderness in the temporomandibular joint, and temporary changes in dental occlusion. These adverse effects may prolong the acclimatization to the device or even cause the discontinuation of treatment. With time, the initial discomfort disappears, while more permanent bite changes become more obvious. These side effects are well known. Their general management relies on clinical experience rather than scientific evidence, since there are few evaluated methods that describe how to manage side effects [156].

13.11.1 Forces from the Device

The nightly repositioning of the lower jaw to an anterior position will create forces on the teeth. Distally directed forces will arise on the upper jaw and teeth and anteriorly directed ones will appear on the lower jaw and teeth. Already in the beginning of the treatment, the patient may experience slight discomfort in terms of a temporary changed bite with fewer occlusal contact areas some hours after appliance removal [157, 158]. The muscle force is weaker, but returns to normal levels [157, 158]. Dose-dependent but individually variable forces from the appliance will influence tooth movement and the changed dental occlusion [159]. A cut-off of 60% advancement for significantly increased forces on the teeth has been identified [160]. Patients with poor oral health including an insufficient bony support of the teeth will be more at risk for negative effects from the forces of an OA_M as well as suffer from impaired oral health by wearing a device on the teeth at night.

13.11.2 Short-Term Side Effects

From treatment start, the patients may experience salivation problems, tenderness in teeth, temporomandibular joint symptoms, or bite changes [2]. Some side effects are appliance related, since devices that are less indi-

vidualized or non-adjustable ones give more adaptation problems [17, 63, 66, 69] and are also less efficient [59, 63, 66]. The repositioning in itself influences the magnitude of discomfort, since a device that advances and opens the jaw more causes more side effects compared with a control device or a device with less repositioning [33, 48, 54]. An unforeseen aspect of the risk of side effects is visualized in a study that found more muscular pain in the initial part of OA_M treatment in patients on statin medication than in controls [161].

PAP causes similar amount of side effects as OA_M [2], although these differ in character between the treatments [24, 32, 38, 39, 41, 44]. The PAP mask produces pressure in different areas of the face and may cause skin irritation, while OA_M produces pressure on the teeth and jaws that may cause tenderness or pain. PAP more often causes dryness in the throat or nose problems, while OA_M may cause either excessive salivation or a dry mouth. Both treatments may disturb sleep [38]. Adherence problems are more common with PAP than with OA_M treatment, and side effects tend to more often cause the discontinuation of treatment with PAP than with OA_M treatment [2].

13.11.3 Methods to Avoid Initial Side Effects

Prevention of muscle tenderness and TMJ pain has been the main topic in studying methods to decrease short-term side effects. A group of patients with diagnosed TMD experienced less pain after performing mandibular exercises compared with a similar group of patients who had been randomized to placebo training [162]. Similar exercises were also tested in patients without previous TMD, and no patient experienced TMD symptoms after 1 month of OA_M treatment [163].

Prevention of occlusal changes in the initial phase of treatment has been studied using either jig exercises or stretching in the morning, and both methods increased the number of occlusal contacts [164].

13.11.4 Longer Term Side Effects

Salivation problems and tooth discomfort usually decline or are less disturbing in the longer term [165–170]. Temporomandibular joint symptoms decrease in patients who continue treatment [168, 171, 172] and do seldom limit OA_M use [171, 173]. Instead, tooth movement and bite changes become more and more prevalent with time. Almost all patients will receive more or less marked bite changes after 5 years' treatment [174]. Only a minor part of the patients is, however, disturbed about these bite changes [169, 172, 175–177].

13.11.5 Changes in Dental Occlusion

There is a continuous progressive mesial shift in dental occlusion with molar positions changing into a more mesial relationship (Angle Class III) and reductions in overjet and overbite during OA_M treatment according to measurements on dental casts and cephalograms [166–170, 174–176, 178–189] (Fig. 13.7). There are also slight skeletal changes, primarily of the lower jaw, in the same direction as those of the teeth [166, 167, 170, 178, 180, 182, 184, 187, 190, 191].

The changes in overjet and overbite are noticed early during the first years of treatment and thereafter continue gradually [186] (Fig. 13.8a, b). After 5 years' treatment, around one third of the patients may be expected to have >1 mm change in overjet or overbite [185]. After one decade of treatment, around 2 mm decrease in average in overjet and overbite has

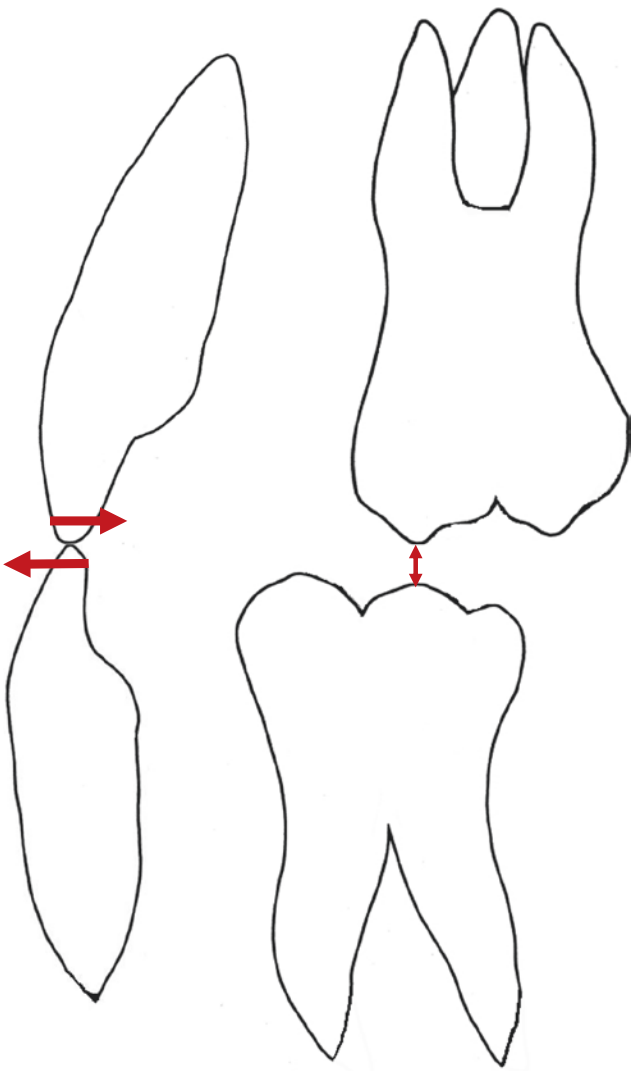

Fig. 13.7 Expected bite changes during longer use of OA_M

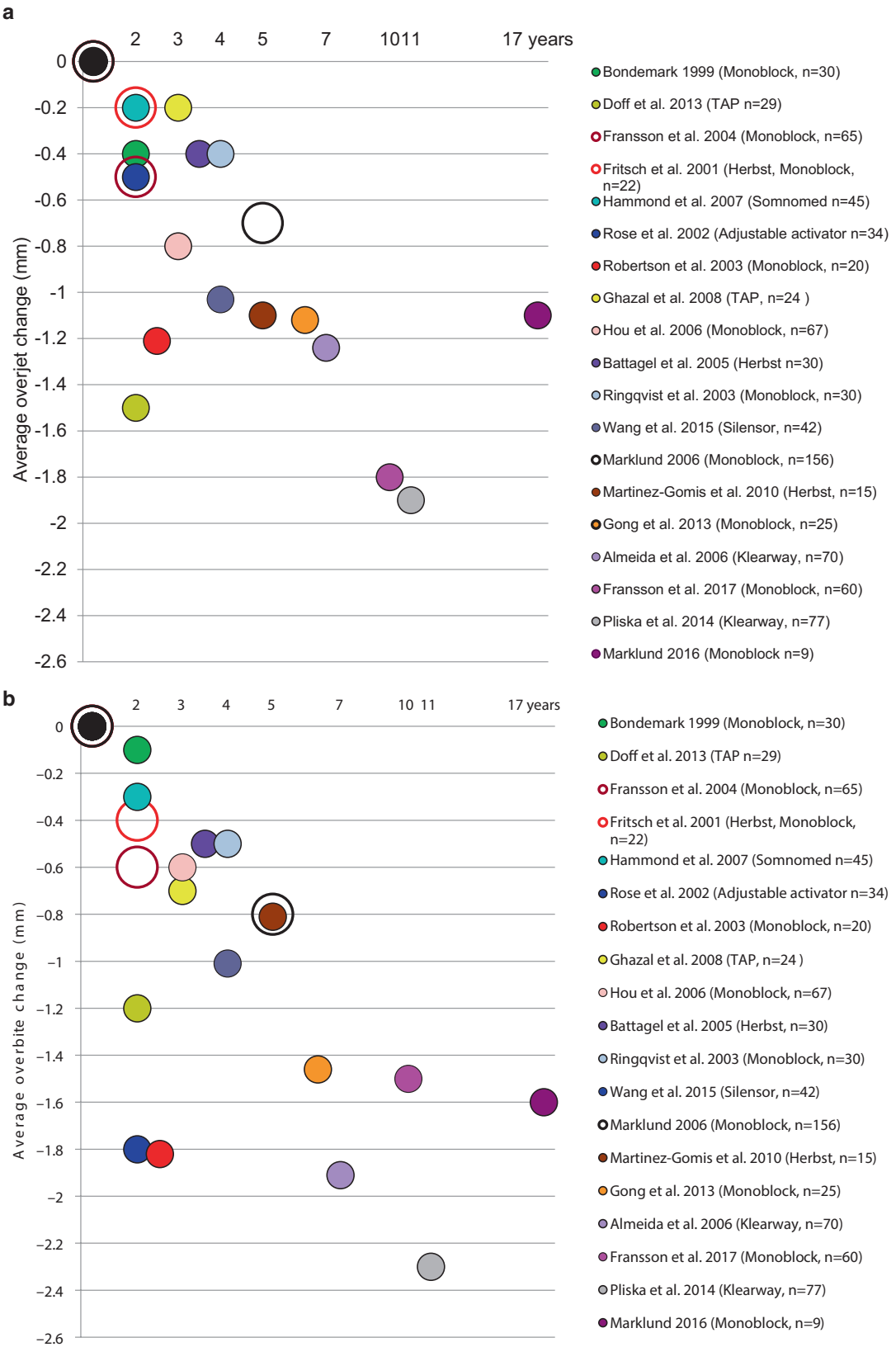

Fig. 13.8 **a** Average overjet change during longer term use of OA_M. **b** Average overbite change during longer term use of OA_M

been reported [186, 189]. In a small group of patients that were evaluated after 17 years, the median changes in overjet and overbite were 1–2 mm [192]. Two patients had more extreme reductions of 4–5 mm in overjet or overbite during those years. Younger and older subjects are affected by bite changes to a similar degree during similar observation times, although this relationship is sparsely evaluated and depends on the individual oral health [113].

In the clinic, some patients complain about problems to chew tough food because they have lost tooth contacts between their posterior teeth. Studies have confirmed that the changed bite will result in fewer contacts between the upper and lower teeth in the posterior parts of the dentition [168, 174, 181, 186, 193, 194]. One fifth to one fourth of the patients have posterior open bites after 1–2 years' treatment with OA_M [172, 188]. It must be noticed that the occlusal contacts vary largely over time and may improve [168]. Patients also complain about food impaction during longer term treatment, possibly because of lost contact between teeth where the appliance is attached [177].

The teeth might also undergo other types of positional changes in terms of crowding of teeth or the development of interproximal open spaces [186, 188, 195, 196], but such changes are not observed in all studies [174]. The upper posterior teeth may tip distally and the lower molars can incline mesially [178].

13.11.6 Bite Changes in Relation to Initial Bite Characteristics

The initial type of bite is associated with the degree of bite changes from OA_M treatment. Patients with normal bite (Angle Class I) or mesial occlusion (Angle Class III) will be more at risk of unfavorable bite changes from OA_M treatment. On the other hand, subjects with distal deep bite and a large overjet (Angle Class II:1) can expect favorable changes with reduced overjet and overbite [174, 185]. Such positive effects might also explain why many patients are unaware of bite changes [177]. An initial deep bite has been associated with a less marked decrease in overjet [174, 185].

13.11.7 Bite Changes in Relation to Mandibular Repositioning

The larger the mandibular advancement by the device, the greater is the risk for reductions in overbite [181] and overjet [176, 185]. In accordance with these findings, a large opening increases the risk for a decrease in overbite [185, 187].

13.11.8 Device Design and Possibilities to Prevent Bite Changes

A few studies have compared devices in terms of dental side effects. A device with an anteriorly positioned adjustment mechanism (Fig. 13.4f) was associated with more pronounced occlusal changes when compared with an OA_M with a lateral adjustment mechanism (Fig. 13.4d) [194]. There was no difference in changes in overjet and overjet between OA_M with a lateral adjustment mechanism and CPAP in that study. Another study used a specially designed OA that lacked material in front of the upper incisors in order to cause less pressure on these teeth backward [191]. In the lower jaw, there was a spring in front of the incisors in order to prevent these teeth from moving anteriorly. No significant change in overjet and overbite was detected after 4 years' treatment. Another observational study compared a soft elastomeric device that covered parts of the alveolar processes and the teeth with a hard acrylic OA_M that was mainly fixed to the teeth and found fewer changes in overjet and overbite with the soft elastomeric device with alveolar extensions [185]. Finally, a rigid full-coverage appliance was found to prevent from incisor crowding that might appear from a flexible device without incisor coverage [196].

One small randomized controlled study aimed to specifically counteract the forces from the device by incorporating counteracting forces on the upper front teeth [197]. The forces were designed to produce a proclination of the upper incisors by relocating them in a plaster cast model, on which the soft elastomeric monoblock appliance was made [197]. This study reports positive effect on overjet changes compared with a control device, but was not developed further because of the need of using adjustable devices which are more difficult to fabricate in soft elastomer.

13.11.9 Comparison of Bite Changes Between PAP and OA_M

PAP treatment may also result in bite changes, primarily the loss of contacts between the upper and lower posterior teeth [181, 194]. This is probably explained by the changed lower jaw posture during PAP use. No change in overjet and overbite was found from PAP [181, 194]. The pressure of the PAP mask on the upper jaw may alter its form [198].

A larger mandibular advancement is generally associated with larger bite changes and a higher efficacy of MAD [48, 199], although there is no linear relationship [49, 50]. These progressive bite changes will successively reduce the mandibular advancement. This will

introduce risk of impaired efficacy of the device, since it is the teeth that move, not the jaws [200]. Higher PAP pressure will produce more side effects in terms of leaks, nasal irritation, and intolerance [201], but not hazard the mechanism of that device.

In conclusion, bite changes during OA_M treatment are progressive in nature, and patients may at some time point be disturbed about the aesthetics or have problems with chewing. Most importantly, these bite changes will influence the mechanism of the device, since a forward shift of the lower teeth compared with the upper ones will result in a successively reduced degree of mandibular advancement by the device. Consequently, patients must be cared for in an individual way, since they will respond differently to a mandibular repositioning during the night. It may be important for the future to study how often the patients have to be followed up in order to assess bite changes in relation to the efficacy of the device and the importance of bite changes for oral health.

13.12 Adherence and Mean Disease Alleviation

13.12.1 Measurement of Adherence

Most studies rely on subjective reports of adherence to OA_M treatment. More recently, compliance monitors for OA_M have been incorporated in some types of appliances and found safe [202]. These monitors have, however, to be more supervised compared with those in the PAP machines [203, 204]. Objective measurement of OA_M adherence has been compared with subjective reports and found to give fairly similar results, with a 30 min overestimation of usage time in the patients' reports [203, 204]. A few studies have now used these monitors, which strengthen their results [28, 119, 205].

13.12.2 Definitions of Adherence

Patients may discontinue treatment or they can adhere to various degrees to a treatment. Adherence to OSA treatment has mainly been identified for PAP and described in various ways [206]. The most common descriptions include the average hours of use per night and the percentage of nights the treatment was used during a specific period. A regular PAP user is identified by: ≥4 hours nightly use at least 70% of the nights [207], and a frequent user is defined even more strictly by Pepin et al. [208] requiring more than 4 hours per night on more than 5 days per week. In general, more CPAP use is related to better outcomes, both on mortality, cardiovascular outcomes, and quality of life [201, 209].

13.12.3 Adherence

Objectively assessed adherence after 3 months' OA_M treatment was 7 hours/night in average in a group of 43 assessed patients [210]. Regular objectively measured OA_M use was found in 85% after 3 months and in 82% after 1 year [203]. More than half of patients continue treatment after 3–4 years according to other studies without compliance monitoring [211–213]. Comparison between OA_M and PAP demonstrates that self-reported adherence is higher for OA_M already at 1 month after acclimatization [43]. OA_M are also used around 1 hour longer each night than PAP [2, 214].

13.12.4 Mean Disease Alleviation

A combined measurement, the mean disease alleviation, summarizes the efficacy and the adherence of the specific therapeutic method [202, 203]. A very effective, but less utilized treatment can in such a comparison be considered fairly equal as another treatment that is less effective, but more often used [4].

13.12.5 Reasons for OA Non-adherence with OA_M

Non-adherence to OA_M treatment is strongly associated with patient worries and experiences about discomfort, adverse effects on the teeth, and lack of efficacy on snoring and apneas [119, 211, 212, 215]. Clinicians are therefore recommended to continuously follow-up the treatment outcome as well as factors such as device quality and new dental restorations that may interfere with OA_M treatment [211, 212, 215].

In summary, the adherence to OA_M therapy is generally higher than that of the more efficient treatment with PAP, which to some extent, may equalize these treatments in some groups of patients. More than half of the patients continue with OA_M after some years, although the adherence is dependent on a number of factors such as oral health, side effects, and disease deterioration: factors that are usually of less concern for PAP treatment.

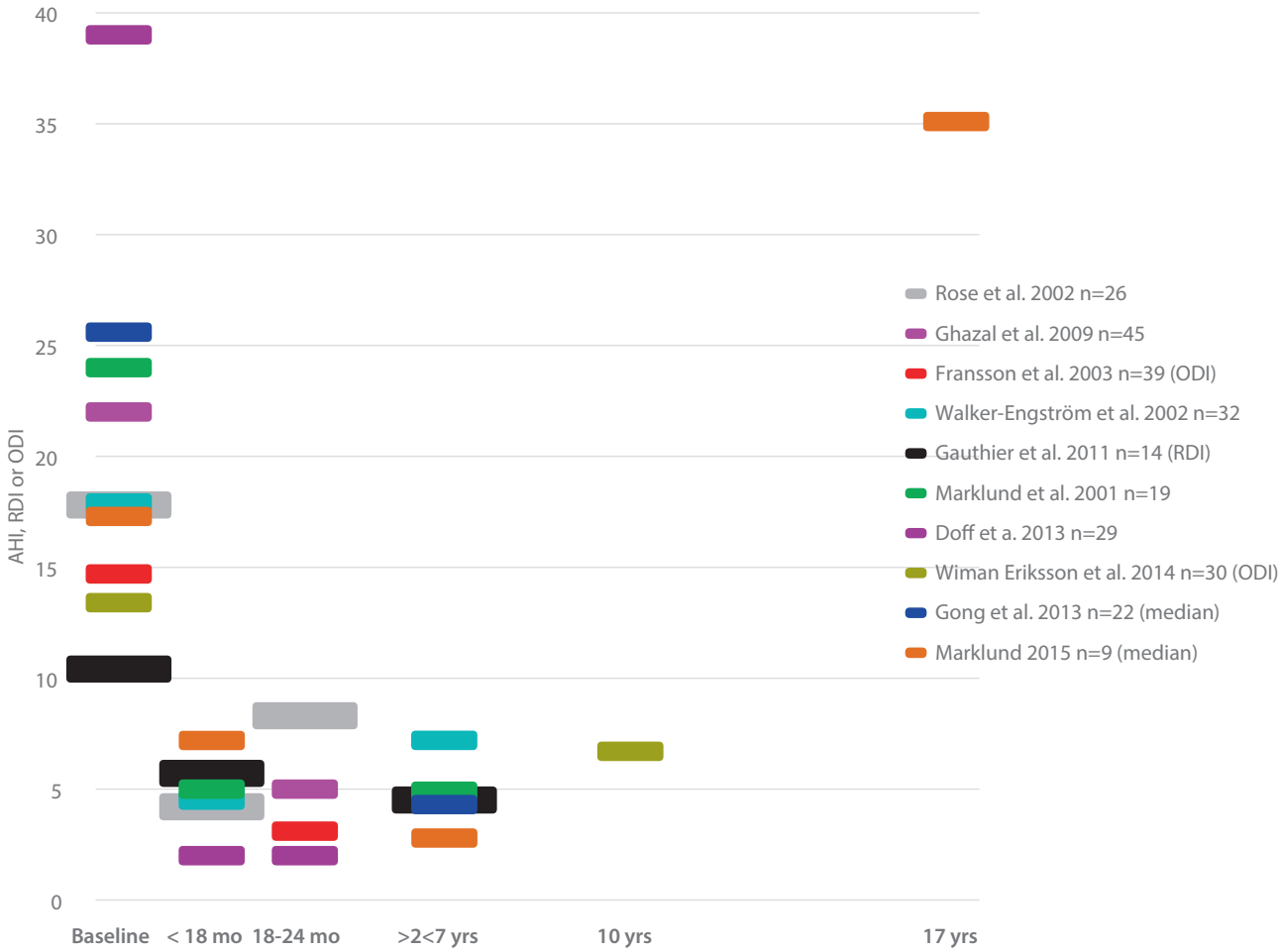

Fig. 13.9 Long-term efficacy of OA_M

13.13 Long-Term Outcomes

A fairly stable effect of OA_M on AHI from 2 up to 10 years has been reported in ten studies of carefully followed-up patients [58, 155, 167, 213, 216–221] (Fig. 13.9). Only one small study has followed OA-treated patients even longer [192]. After 17 years, all the patients had worsened with the device and all but two patients had increased their AHI without it. The patients had not increased in weight, and they were not sleepy at the follow-up. During this long treatment time, the patients had received continuous follow-up of symptomatic effects and side effects. Regularly, the devices had been replaced with new ones with more advancement, if needed, in order to compensate for the mesial shift in dental occlusion. The increase in AHI at follow-up can be explained by the fact that patients were older or had more comorbidities [222]. Studies show that AHI and the durations of apneas, hypopneas, and desaturations may be expected to increase during the time when sleep apnea treatments are ongoing [109, 223]. It must therefore be considered in the original treatment plan that the OSA pathophysiology in the individual patient may change over time. In addition, a mesial movement of the whole dental occlusion will change the intended degree of mandibular advancement, which may reduce the efficacy of the device.

In conclusion, the effectiveness of an OA_M will decrease in the longer term, since some patients discontinue treatment because of poor subjective treatment effects or side effects. Continuing patients may risk a poorer objective treatment effect on sleep apneas. It is not the same patient that is treated after a number of years, because of for instance age, comorbidities, and bite changes. The mechanism of action of OAs is more vulnerable than that of PAP, which makes it necessary

to continuously re-evaluate the treatment outcome. More research is needed about the long-term outcomes of OA_M therapy. In addition, it would be of interest to better study oral health during OSA treatment, that is, whether oral health improves or deteriorates as a result of the treatment, and, in that case, in which patients.

13.14 OA_M in Relation to Other OSA Treatments

13.14.1 OA_M Compared with Positional Therapy

Positional therapy can be used in patients with positional OSA. This method can be subdivided into sleep position training that use some sort of alarm or vibration in order to prevent the subjects to sleep supine and methods that makes it uncomfortable to sleep on the back such as the use of tennis balls or other items. Both techniques effectively reduce the percentage of supine sleep and AHI [224, 225]. The sleep position trainer is preferred by the patients and gives a better sleep quality compared with sleeping with a tennis ball or backpack [225]. Snoring is, however, not satisfactorily reduced by positional therapy [226].

Patients with positional OSA can be diagnosed based on the chance of elimination of sleep apneas with positional therapy. Consequently, a normal value of the non-supine index (non-supine AHI < 5) will give a high chance of complete treatment success [98], but patients may benefit from this treatment also using other definitions.

OA_M therapy and sleep position training have equal short-term efficacy on AHI in patients with position-dependent OSA defined by a doubled AHI or more in the supine sleep compared with the non-supine position [205]. In addition, both treatments remained stable in their effect on AHI after 1 year [227]. Positional therapy must, in accordance with OA_M therapy, continue long term, since there are no indications of a training effect in sleeping less supine after the use of a sleep position trainer [228]. A problem with positional therapy is that supine dependency can vary from night to night and a more than fourfold difference is needed in order to identify supine dependency, and this criterion was only found in men [229].

13.14.2 OA_M Combined with Positional Therapy

The efficacy of OA_M therapy differs in relation to sleep positioning. The lateral-AHI is more often normalized than the supine-AHI by OA_M [99, 101]. Consequently, it is fairly common that apneas may persist in the supine sleep position. The prevalence of supine dependency during OA_M treatment ranges in between 18% and 34% depending on the definition of this condition [99]. These patients with insufficient effect in supine sleep position during OA_M treatment may be subjected to intermittent increased sleep apnea frequencies, depending on the alternatingly preferred sleep position. A good treatment outcome in all sleep positions is important for some of these patients, since severe supine-dependent OSA has been related to an increased risk for cardiovascular events and mortality [230]. A combination of both OA_M and a sleep position trainer that helps the patients avoid supine sleep is therefore a promising new concept in order to increase the success rate of OA_M therapy [231]. The long-term outcome of this therapy is unknown.

13.14.3 OA_M Combined with PAP

Some patients do not tolerate PAP therapy every night, and these patients may benefit from OA_M as an alternate treatment. Sleep apneas will be reduced during periods of non-tolerance to PAP or on journeys [232], which may reduce the risk for negative consequences of untreated OSA.

OA_M and PAP can also be used together in order to reduce the resistance in the upper airway and reduce the PAP pressure [233–235].

13.15 OAs as Second-Line Treatment

Non-compliance with PAP might, to some extent, be explained by differences in OSA phenotypes [236, 237]. For some patients who do not tolerate PAP, OA_M may constitute a less intrusive treatment alternative. Both moderate and severe OSA patients have been found to reduce their AHI to a substantial amount with OA_M treatment, also after 1 to 2 years [92, 238]. In a group of severe OSA patients, over half of them continued with OA_M after 2 years [238]. Half of the patients who had a renewed sleep apnea recording had a treated AHI of less than 15.

13.16 Guidelines

There are many published guidelines and meta-analyses that provide clinical advice about how to conduct the best available care for the patients [1–4, 47, 239–244]. Several guidelines include advice for general practicing dentists to be aware of OSA, since they can be of help to recognize

the signs of the disease in order for the patients to seek medical care [239, 241, 243–245]. One guideline highlights that there might be differences in signs and symptoms of OSA in a geriatric population who still will be at great risk of complications of untreated OSA [242]. In the recognition of OSA, there are some validated methods such as the Berlin Questionnaire and STOP-Bang, but also a validated app for the mobile phone: the NoSAS [246].

The guidelines generally recommend OA therapy for patients with snoring, mild to moderate OSA, or CPAP-intolerance. The need to treat patients within the mildest spectrum of sleep-disordered breathing has, however, been questioned, because of reports that mild OSAS is not significantly associated with comorbidities [90, 247]. New methods do diagnose this disease will probably change these indications [5]. Dentists who are educated in dental sleep medicine can provide treatment with OA therapy for referred patients. The treatment must be proceeded by a thorough odontological investigation regarding dental and periodontal health. Assessment of the temporomandibular joint, and movement capacity of the lower jaw as well as diagnosis of dental occlusion must be performed. Custom-made, titratable oral appliances are recommended, preferably those that have been evaluated in studies. It is emphasized that the treatment should be regularly followed-up and yearly controls of subjective effects, side effects, and device quality are recommended. This regime will probably be facilitated by the novel EC recommendations regarding requirements for drivers with OSA [248]. One guideline [2] highlights the need for a strict terminology and advises to use the term "OA" instead of the many local terms. In addition, well-defined protocols for titration and follow-up should be developed. More research concerning longer term health outcomes during OA_M treatment, avoidance of side effects, and device comparison is needed [2].

13.17 Summary of OA_M

OA_M is the most effective non-CPAP therapy for patients who snore or those with mild to moderate sleep apnea and PAP-intolerant patients. More precise predictors based on the etiology of the disease in the individual patient are under development. This will provide better tools to avoid ineffective OA_M treatments. In patients with mild disease who suffer from daytime sleepiness, a test period with PAP treatment is of help to find if they respond with a symptomatic relief to OSA treatment before OA_M therapy is started. The efficacy of OA_M must be tested in order to verify the objective outcome of the treatment, since symptoms are poorly correlated with sleep-disordered breathing. In the longer term, side effects in terms of bite changes are common and progressively increase. The treatment effect may deteriorate when patients age and sleep-disordered breathing become more severe and change in character. Most likely, OA_M treatment will therefore take place during periods of the patients' lives. When OA_M treatment is initiated earlier in life, it is likely that PAP treatment will be needed after some time. It is important to carefully follow-up OA_M treatment regularly in terms of efficacy, side effects, and the need for other or combined treatment options.

13.18 Devices That Hold the Tongue Forward—OA_T

13.18.1 Effects

OA_T is designed to produce suction of the tongue into an anterior bulb and do not require teeth [249–252]. OA_T produces a similar apnea reduction as OA_M in patients who tolerate both devices [250]. The adherence is generally poor with OA_T, since only around one third of the patients continued after 3 weeks [250]. Only one fifth could be followed up with a renewed sleep apnea recording after some months in another study [252], while half of the patients continued for 5 years in further another study [249]. OA_M are preferred compared with OA_T by over 90% of patients who had tried both treatments [250].

13.18.2 Side Effects

The short-term side effects from OA_T resemble those from OA_M including excessive salivation and a dry mouth [249, 250, 252]. In addition, patients who are treated with OA_T often complain about tongue soreness and problems to keep the appliance in place [250, 252]. Bite alterations with anterior or posterior open bites and a reduced overjet have been reported from OAT_T [253].

13.18.3 Summary of OA_T

Tongue-retaining devices may reduce obstructive sleep apneas, but poor tolerance and side effects make this treatment less useful in patients with snoring or obstructive sleep apnea.

Conflict of Interest The author certifies that she has no affiliations with or involvement in any organization or entity with any financial interest, except for a consultancy fee from ResMed, or non-financial interest in the subject matter or materials discussed in this manuscript.

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Other Therapies and Emerging Options for Management of OSA

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Contents

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14.1 Introduction

Continuous positive airway pressure (CPAP) therapy is considered to be initial and gold standard therapy for obstructive sleep apnea (OSA). It is a safe and cost-effective therapy [1], though compliance with CPAP can be challenging in many patients with OSA. It has been reported that as high as 10% of patients may refuse CPAP therapy [2]. Discussions about treatment other than CPAP therapy are common in both academic and non-academic sleep medicine clinics as patients often seek alternatives options. The scope of this chapter is to discuss other non-PAP treatment options and emerging therapies for the treatment of OSA. Patients with OSA can be counseled on these options as clinically appropriate.

14.2 Positional Therapy

OSA is often found to be prominent in supine or REM sleep. Positional obstructive sleep apnea is often defined when the AHI is twice as high in the supine sleep compared to non-supine sleep. Stricter definitions of positional therapy include normalization of AHI (<5/hr) in the lateral position.

The prevalence of positional OSA is estimated to be as high as 55–60% [3] but much higher in Asian population, reaching nearly 70% [4]. On average patients with positional OSA are younger, thinner, and have less severe OSA than their counterparts [3, 4]. When using stricter criteria for positional OSA, with normalization of AHI (<5) in the lateral position, the prevalence of positional OSA was found to be about 27%. When severity of OSA is considered, in patients with mild OSA, the prevalence was about 50%. The prevalence dropped dramatically to only about 19% of moderate severity OSA and 6.5% in cases with severe OSA [5].

There is a strong inverse correlation with BMI and position-dependent OSA; changes in body weight have been shown to affect positional dependence [3]. Patients with positional OSA who over time converted to non-positional OSA had gained weight and had overall worsening of OSA. The converse was also shown in patients with non-positional OSA converting to less severe, positional OSA with weight loss [6].

The supine position is associated with increased apnea severity in terms of apnea duration, desaturation, arousal length, and frequency. [7] Few studies have examined the anatomical changes in the airway during lateral position and the exact mechanisms responsible for the improvement in breathing in the lateral position is not entirely known but the effect of gravity likely plays a role. Anatomical optical coherence tomography of the upper airway in awake OSA patients and controls in the supine and lateral positions has shown airway changes from a more transversely oriented elliptical shape when supine to a rounder shape in the lateral recumbent position, but it does not show changes in the overall cross-sectional area [8]. The increased circularity of the airway in the lateral position may render it less likely to collapse.

Drug-induced sleep endoscopy performed on OSA patients investigated the effect of body posture on the site of airway obstruction. When changing from supine to lateral position, obstruction at the tongue base and larynx was significantly improved; however, the prevalence of lateral wall obstruction was not affected suggesting that those with non-positional OSA have persistent lateral wall obstruction [9].

There are numerous strategies developed to maintain the lateral position during sleep. One of the simplest has been called the "tennis ball technique." This therapy consists of a tennis ball fastened to the back by straps or in a pocket or similar construction. Commercially made waist-bands with foam pillows worn on the back to prevent supine sleep are also available. More recently, a vibrating device worn on the neck to alert the user of being in the supine position has been developed.

While the tennis ball technique has been shown to significantly reduce AHI and time spent in the supine position, studies on long-term compliance have been poor with 38% reporting compliance at 6 months and less than 10% reporting continued use over 30 months [10, 11]. The main reasons for discontinuing therapy were discomfort, ineffectiveness due to the ball moving too much or no improvement in sleep quality or daytime sleepiness [10].

Self-made and commercially made waistbands for positional therapy have been shown to be successful in reducing AHI (by ≥50% and below 20) in 68% and reducing AHI < 5 in 40% of patients with positional OSA, with no statistically significant differences between ESS and time spent supine [12]. Despite reduction in AHI, 60% of those treated with these positional devices had stopped therapy after 13 months.

More recently, vibrating neckband has been developed, which senses when the user has rolled in to the supine position and alert the user to turn to the lateral position. One such commercially available device, Night Shift, is worn around the neck. This device has been shown to significantly reduce sleep in the supine position and reduce AHI by 69% in patients with positional OSA (defined as overall AHI ≥ to 1.5 times greater than the non-supine AHI) [13]. The device was shown to improve sleep architecture, decrease cortical arousals, increase N2 sleep, and decrease N1 sleep and improve Epworth Sleepiness Scale scores. Over the 4-week study 7.4–13.4% of participants reported perceived worsening sleep quality due to the device. No long-term studies

have been done to assess compliance, but at 4 weeks, the median compliance was 96%.

A chest-worn device, commercially available as NightBalance device, vibrates to alert the user when in the supine positon. After 1 month of usage in patients with mild-to-moderate positional OSA defined as AHI at least twice as high in the supine position compared to non-supine, AHI was significantly decreased from a mean of 16.4–5.2. About 48% of the study patients demonstrated resolution of OSA with an overall AHI < 5 [14]. This device has been also shown to effectively decrease time spent in the supine position, from a median of 49% to 0%, and improved subjective sleepiness and sleep quality. At 6 months, compliance defined as 4 hours of nightly use was 64.4% and median ESS decreased from 11 at baseline to 8 [15].

In patients with mild positional OSA, with AHI <5 in the lateral position, positional therapy using a commercially available foam pillow secured to the back (ZZoma Positional Sleeper) has been found to be as effective as CPAP in treated OSA, with the same proportion of patients achieving AHI < 5 with the positioning device as CPAP with no differences in sleep quality or oxygen saturation [16]. This study was limited to one night of therapy and therefore efficacy over long term remains unclear.

CPAP has been compared with positional therapy in patients with positional OSA. A study comparing treatment of positional OSA, defined as 50% reduction of OSA in the lateral position, with 2 weeks of CPAP and 2 weeks of positional therapy, found CPAP to be more effective reducing AHI and improving oxygen saturation than positional therapy [17]. Despite this finding, there were no significant differences in sleep architecture, ESS, MWT, mood, or quality of life measures between therapies.

A similar study using a thoracic anti-supine band (TASB) mimicking the tennis ball technique compared the efficacy of positional therapy in mild–moderate positional OSA patients to nasal CPAP. This study found that with CPAP, there was a statistically significant greater reduction in AHI and higher percentage of patients achieving treatment success (AHI <10) than with positional therapy [18]. TASB significantly reduced the time spent in the supine position to a mean of 6.3% versus 35.4% with CPAP.

In summary, positional therapy has been shown to reduce AHI and time spent in the supine position. Positional therapy may be beneficial and potentially as effective as CPAP in a select population whose OSA normalizes to AHI < 5 in the lateral position. Limited studies have shown long-term compliance with positional therapy is poor demonstrating the need for close clinical follow-up and also need for more comfortable positional therapy options.

14.2.1 Weight Loss

It is estimated that in the United States, 5.7% adults aged 30–69 years have moderate or severe sleep disordered breathing (SDB) and 58% of those adults have sleep disordered breathing attributable to excess weight [19]. If this is expanded to include adults with mild SDB, the percentage with sleep disordered breathing increases to 17%, with 41% attributable to excess weight [19]. In people with SDB, there is a dose–response relationship between weight gain and severity of SDB, with each percentage change in weight was associated with a 3% change in AHI or for each 1 kg/m^2 increase in baseline BMI, an increase in AHI of about 1% [20]. A 10% weight gain was found to be associated with a 32% increase in AHI, relative to maintaining a stable weight and a sixfold increase in the odds of developing moderate-to-severe obstructive sleep apnea [20]. This strong relationship between SDB and the obesity epidemic suggests the weight loss strategies should be an integral part of management of SDB. The current American Academy of Sleep Medicine quality measures recommend at least yearly discussion of weight management for adult patients with moderate-to-severe obstructive sleep apnea [21].

Dietary weight loss has been shown to improve OSA. In OSA patients with diabetes, an intensive lifestyle interventions consisting of a behavioral weight loss program, portion-controlled diet with a prescribed calorie intake, and 175 minutes of physical activity per week has shown to be more effective than diabetes education and support [22]. The participants in the lifestyle intervention lost significantly more weight than those in the diabetes support and education group, 10.8 kg versus 0.6 kg with significantly greater reductions in waist and neck circumferences. AHI in the intensive lifestyle intervention group decreased from 22.9 to 18.3, with an adjusted mean decrease of 9.7 events per hour, versus increased from 23.5 to 28.3 in the diabetes education group. The difference in the two groups was due to changes in the obstructive apnea episodes and not hypopneas. In patients in the lifestyle intervention group, there were also significant changes in the severity of OSA as well with remission of OSA (AHI <5) being three times more common compared to the diabetes education group. The greatest benefit was found to be in men and participants with higher baseline AHI values. At 4 years, the beneficial effects of intensive lifestyle intervention persisted despite weight gain of almost 50%.

Very low-calorie diets have been shown to improve OSA in obese patients (with BMI between 30 and 40) with moderate-to-severe OSA, with the greatest effect on those patients with severe disease [23]. The diet for

one such study consisted of a very low-calorie liquid diet for 7 weeks followed by 2 weeks of gradual introduction of normal, followed by a weight loss maintenance program. Significant weight loss was achieved by 9 weeks, and 73% of patients following the diet were no longer classified as obese, losing an average of 18 kg. Weight loss following this diet was associated with a reduction in AHI by 21 events per hour. Weight gain was noted at the 1-year mark, with 56% of participants being categorized as obese. However, despite weight gain, at 1-year follow-up, the average reduction in AHI was largely maintained at 47% of baseline (reduced by a mean of 17 events/hr) with 10% having total remission of OSA at 1 year. Patients with severe OSA showed greater reduction in the AHI amounting to 25 events per hour. In patients with moderate OSA, AHI was reduced by seven events per hour.

In overweight and obese patients (with BMI between 28 and 40) with mild OSA, following a very low-calorie diet (VLCD), that is, 600–800 calories per day) for 12 weeks, was more effective in terms of weight loss and improvement in OSA compared to lifestyle intervention (diet and exercise counseling) [24]. Weight was reduced by 10.6% in the VLCD group compared to 2.6% in the lifestyle intervention group. After 12 weeks, AHI of those in the VLCD group was reduced by 40%, from 10 to 5.6, with 61% having complete resolution of OSA. With lifestyle intervention, there was a 14% reduction in AHI from 9 to 8.3, with 32% demonstrating resolution of OSA. At 1 year, the odds ratio for having mild OSA was 0.24 in the VLCD group compared to the lifestyle intervention group. In this study, a weight reduction of 5 kg corresponded to a reduction in AHI by two events per hour.

Meta-analysis of nine studies investigating the effects of dietary weight loss on OSA, including low-calorie diets (800–8000 kcal/day), very low-calorie diets (600–800 kcal/day), and weight loss programs, found a reduction in AHI from 52.5 to 28.3 events/hr with higher weight loss associated with greater reduction in OSA. OSA cure rates ranged from 61% at 3 months to <10% at 1 year [25].

Weight loss surgery has been shown to improve or in some cases resolve OSA. A meta-analysis of investigating the effects of various bariatric procedures on OSA, including gastric banding, Roux-en-Y, biliopancreatic bypass, and gastroplasty, found a mean reduction in BMI from 55.3 to 37.7 kg/m^2. There were significant reductions in mean AHI from 54.7 to 15.8 events per hour (71% from baseline) after bariatric surgery [26]. This is a significant reduction in AHI; however, the mean AHI after surgery was still consistent with moderate OSA. Notably, in 6 of the 12 studies for which individual patient data were available, 25% effectively cured OSA with surgery, attaining AHI < 5. Those "cured" of OSA were overall younger (38.9 years vs 46.5 years) and lighter at baseline (102.7 vs 173.3 kg) than those with residual OSA after surgery [26].

When comparing the specific types of weight loss surgeries, the results of a 2014 meta-analysis of 69 studies including 13,900 patients found 75% patients of all procedure types had improvement in obstructive sleep apnea [27]. Biliopancreatic diversion was the most successful with 99% of patients experiencing improvement in OSA and 82.3% experiencing resolution of OSA. Laparoscopic gastric banding was the least successful with 32% with resolution of OSA, though 70.5% had improvement in OSA.

The degree of weight lost and reduction in AHI has been shown to be greater in those patients undergoing weight loss surgery than with medical weight loss. Laparoscopic gastric banding in obese adults (BMI >35 and < 55) with moderate-to-severe OSA has been shown to be a more effective weight loss method than medical weight loss. Those who underwent gastric banging lost 27.8 kg on average compared to 5.1 kg with medical weight loss [28]. AHIs in both groups showed reduction from baseline; however, the mean reduction in AHI in the laparoscopic gastric banding group was 25.5 events per hour compared to 14.0 events in the medical weight loss group; however, the difference in AHI reduction between groups was not statistically significant. Only one participant in the study, in the medical weight loss group, had remission of OSA (AHI < 5); however, reduction to mild OSA (AHI <15) was achieved by 27% of the surgical group compared to 7% of the medical weight loss group.

A study comparing effects of weight loss from Roux-en-Y gastric bypass (RYGB) to intensive lifestyle intervention (ILI) on patients with all severities of OSA found significantly greater weight loss and reduction in AHI in the RYGB group [29]. Participants undergoing RYGB had a mean weight loss of 42 kg (reduction if BMI by 14 kg/m^2) compared to 12.1 kg, reduction in BMI of 5.4 kg/m^2 in the ILI group. The mean reduction in AHI of 21.6 events per hour in the RYGB group, compared to mean reduction in ILI group of AHI of 8.8 events per hour. In this study, a significantly larger proportion of participants undergoing RYGB had remission of OSA (AHI <5, 66%), compared to 40% of participants in the intensive lifestyle intervention.

A meta-analysis of surgical versus nonsurgical weight loss methods demonstrated that both methods were associated with statistically significant overall reductions in AHI and BMI; however, the reductions in AHI and BMI were greater with surgical intervention [30]. Weight loss surgery was associated with a 15 kg/m^2 reduction in BMI compared to 3.1 kg/m^2 with nonsurgical interventions. Surgical weight loss was associated with a decrease in AHI by 29 events/hr compared to

11 events/hr with nonsurgical weight loss. In this meta-analysis, nonsurgical weight loss included lifestyle modification through exercise, dietary invention, or both, and in some cases, behavioral counseling and pharmacotherapy were used.

Patients considering weight loss surgery should be counseled regarding the expected weight loss but also about improvement or resolution of their sleep apnea as measured by AHI. Follow-up with the treating physician is necessary and repeat sleep apnea testing should be performed following significant weight to assess for residual OSA.

14.2.2 Nasal EPAP Therapy

The nasal EPAP device is a bidirectional valve, which is applied externally to each nostril and kept in place by an adhesive that forms seal between the valve and the nare. Each device is one size fits all, disposable, and designed for a single night of use. The valve has a fixed expiratory resistance of 80 cm H_2O/L/sec at a flow rate of 100 mL/sec [31]. There is minimal inspiratory resistance, thus making exhalation through the nose more difficult creating expiratory positive airway pressure. Unlike CPAP, no inspiratory positive pressure is provided in the nasal EPAP device.

The exact mechanism by which nasal EPAP treats OSA is not known. Several mechanisms have been proposed including positive pressure at end expiration leading to dilation of the airway carrying over into inspiration and preventing collapse and increased lung volume creating traction on the upper airway making it less collapsible. [32, 33].

The device is FDA cleared for treatment of obstructive sleep apnea and requires a prescription [34]. Nasal EPAP is contraindicated in patients with severe respiratory disorders, hypercapnic respiratory failure, respiratory muscle weakness, bullous lung disease, bypassed upper airway (tracheostomy), pneumothorax, pneumomediastinum, severe heart disease including congestive heart failure, hypotension (pathologically low blood pressure), acute upper respiratory inflammation or infection (including sinus, nasal, and inner ear), or perforation of the ear drum. [35].

Efficacy of the device was evaluated by Rosenthal et al. in a multicenter study of 34 adult subjects with OSA with mean age 49.8, mean BMI 30.1, 21.4% female [36]. Subjects were excluded if they had previously tried CPAP, had uncontrolled or serious illness, or had comorbid sleep conditions. Subjects were also excluded if nasal patency was poor due to blockage of one or both nostrils, difficulty breathing through the nose, sinusitis, frequent, and/or poorly treated nasal allergies. The participants completed four polysomnograms (PSGs) in random order. PSGs included one control night and three nights using the device with varying expiratory resistances. Subjects then used the device with the resistance most effective at reducing AHI at home for 30 days. On the control night, average AHI was 24.5. The AHI was significantly reduced to an average of 13.5 on the first treatment night and 15.5 after 30 days of use. At the 30-day follow-up, 41% had an AHI reduction greater than or equal to 50% compared to control. Statistically significant improvement in mean oxygen saturation was noted between the control night and final therapy; however, the mean improvement was 0.4%, which is usually not the goal in clinical practice. ESS scores improved significantly from 8.7 at baseline to 6.9 after 30 days of treatment, and PSQI scores improved significantly from 7.4 at baseline to 6.5 at 30-day follow-up. Despite these findings, there was no improvement in sleep architecture after the initial night of treatment or after 30 days of use [36]. There were no significant differences in ODI, minimum oxygen saturation between the control night and any of the treatment nights. Participants reported using the device all night for 94.4% of nights.

Patel et al. investigated factors predictive of response to treatment. Patients with position-dependent OSA, defined as lateral AHI lower than supine AHI, were more likely to respond to treatment, defined as a > 50% reduction in RDI from baseline and an absolute RDI < 20/hr, but this was not statistically significant [32]. Demographic factors and severity of baseline OSA were also not predictive of therapeutic success.

The long-term efficacy of nasal EPAP was investigated by [37] during a 12-month study of the device involving 41 participants with mean age of 50.1 years, mean BMI of 32.5 kg/m^2, and 63.4% were male. Over the duration of the study, there was a statistically significant reduction in AHI, from 15.7 to 4.7 at month 12 of treatment. ODI was decreased from 12.62 to 7.6 at 12 months. Additionally, there were statistically significant reductions in median arousal index from 23.9 to 19.0 [31]. After 12 months, the median proportion of sleep time spent snoring was reduced by 74.4%. After 12 months of treatment, significant improvement was noted in sleepiness measured by the Epworth Sleepiness Scale with scores decreasing from 11.1 to 6.0. Median device usage during the 12 months was 89.3% of nights for the entire night. Participants with a positive clinical response at month 3 were found to have excellent adherence for the remainder of the 12 months. Forty-two percent of the participants reported adverse events with the device; difficulty exhaling, nasal discomfort, dry mouth, headache, and insomnia were reported most frequently.

The effects of nasal EPAP after withdrawal of CPAP were investigated by Rossi et al. This study aimed to test the effectiveness of nasal EPAP to prevent recurrence of obstructive sleep apnea following CPAP withdrawal.

Sixty-seven participants, previously diagnosed with moderate-to-severe OSA and using CPAP, were randomized to continuing CPAP, nasal EPAP, or placebo nasal EPAP for 2 weeks. The baseline characteristics of all three groups were reported to be similar. After 2 weeks, there was recurrence of obstructive sleep apnea in the nasal EPAP and placebo nasal EPAP groups. There was no significant difference in AHI or ODI between the groups treated with nasal EPAP versus the placebo. Mean AHI in the nasal EPAP group was significantly higher than the group continuing treatment with CPAP with AHIs of 27.6 and 2.4, respectively. Additionally, ODI was 4.3 in the group using CPAP versus 35.8 with nasal EPAP [38]. The findings of this study suggest that the device may not be a suitable alternative for use in patients with moderate-to-severe OSA currently using CPAP.

14.2.3 Oral Pressure Therapy

A proprietary oral pressure therapy device, Winx Sleep Therapy System (ApniCure Inc., Redwood City CA), was introduced as an alternative to CPAP for patients who are unable to tolerate PAP therapy or are unwilling to use CPAP. The device consists of a pump console connected to a polymer mouthpiece via tubing. Oral pressure therapy is delivered as a light negative pressure (as a vacuum) in the oral cavity. This negative pressure allows for stabilization of the tongue and pulls the soft palate forward with an expectation of the dilatation of the retropalatal area.

Winx Sleep Therapy System consists of a mouthpiece, tubing, and a console. The mouthpiece has a built-in-lip seal with a connector to the tubing. While the mouthpiece is not customizable, it is available in ten sizes, sized using a bite wax impression to determine the width and arc measurements. The console has the pump that generates the oral pressure that is transferred via the tubing and delivered to oral cavity. Console also holds the reservoir that collects any saliva that is drained via the mouthpiece and tubing. The proprietary technology oral pressure therapy (OPT) has been shown to treat obstructive sleep apnea [39]. Efficacy of the therapy is contingent upon patient's ability to breathe through their nose while using the oral pressure therapy for it to be effective. The negative pressure delivered is non-titratable; however, feedback control maintains a continuous negative pressure. Once the console has reached the target vacuum level of 51 cm of water, the indicator light on the console is turned on.

A study of utilizing magnetic resonance imaging in wakefulness has shown that the negative pressure generated by the device moves the soft palate anteriorly and superiorly and the anterior–superior segment of the tongue forward increasing the retropalatal airway caliber in the lateral and anterior-posterior dimensions [40]. Patients with a clinical response to the device have been shown to have a significantly greater superior displacement of the soft palate and anterior displacement of the tongue than nonresponders and greater increases in cross-sectional areas of the retropalatal region. Interestingly, in the same study, responders to treatment were shown to have significant decreases in retroglossal cross-sectional area [40].

The efficacy of the Winx device has been investigated in adults with OSA in a multicenter prospective, randomized, crossover trial. [39] The study population included 63 subjects with mild (apnea–hypopnea index [AHI] ≥ 5 and < 15) to severe (AHI ≥ 30) obstructive sleep apnea, with and without prior treatment with CPAP. The study population primarily consisted of men (69.8%) with mean age 53.6 and mean BMI 32.3. Study excluded patients with poor nasal patency, poor mouthpiece fit, severe medical or dental conditions, or were unable to tolerate the device. The subjects underwent initial PSG without the device, with the device, and again with the device after 28 days of use at home. The sequence of the initial PSG (control or with the device) was randomized.

Overall success rate was low in this study, though success was seen in patients with moderate (50%) and severe (23%) obstructive sleep apnea. The median AHI was 27.5 events per hour on the control night. With the device, there was clinically significant response, defined by the study team as treatment AHI $\leq$ 10/hr and $\leq$ 50% of control values, in 20 of 63 (31.7%) subjects. Average nightly use of the device during the 28-day period of home use was 6 hours, and 84% of subjects used the device for >4 hours per night. Over the 28-day treatment period, the mean Epworth sleepiness scale score was significantly reduced from 12.1 to 8.6 among subjects who were naïve to treatment for OSA and remained unchanged in subjects using CPAP up until the trial period.

The device was generally well tolerated with three subjects discontinuing the study due to discomfort and 76% indicating that they would use the device to treat their OSA. Adverse events were reported on average of 50% of nights of use, were generally mild, and included oral or dental discomfort or irritation and dry mouth. No significant occlusal or tooth movement was demonstrated over the 28-day period of use.

Subsequent studies done have shown the success rate, achieving AHI $\leq$ 10 events/hr with OPT have remained below 50% [41, 42]. In general, the success rates were higher in patients with moderate obstructive sleep apnea, which may be due to definition of success in the study group.

The device is available without a prescription though many insurances are not covering the cost of therapy. OPT may be a significant out-of-pocket cost

for most patients, ascertaining the effectiveness of OPT may be more desirable. Winx can be connected to polysomnogram (PSG) system using the Winx PSG adapter. Sleep study using OPT would allow to ascertain frequency of apneas/hypopneas, hypoxemia, and improvement in the sleep architecture. In addition to ascertaining efficacy, PSG with OPT would also assess for tolerance and assess for maintenance of target oral pressure of 50–51 cm of water. Sleep center providing this test must have a wide array of mouthpiece sizes available, as well as staff trained for appropriate fitting. This may be resource intensive for some centers limiting its clinical use.

As of yet, there are no predictors of success with the Winx device, and thus successful treatment with the device should be confirmed. The device is not recommended for use in patients under 18 years of age, patients with central sleep apnea, or with severe pulmonary disease, pneumothorax, loose teeth, or advanced periodontal disease. OPT may not be appropriate in patients with BMI ≥ 40 and nasal obstruction. OPT leads to reduction in the AHI in select group of patients with obstructive sleep apnea, though majority may still have residual sleep apnea. Oral pressure therapy may be an option in patients with claustrophobia or less than optimal dentition [43].

14.2.4 Hypoglossal Nerve Stimulation

At this time, the only commercially available hypoglossal nerve stimulator is the Inspire device (Inspire Medical Systems, Inc.), which is an implantable programmable neurostimulator, FDA approved for the treatment of obstructive sleep apnea. The device consists of three implanted components: the neurostimulator, the sensing lead, and the stimulation lead. The neurostimulator is implanted in the right infraclavicular region and is connected to the sensing lead, placed between the internal and external intercostal muscles, and the stimulation lead placed on the medial division of the right hypoglossal nerve. The neurostimulator delivers electrical pulses to the hypoglossal nerve via the stimulation lead, which is synchronized with respiration by the sensing lead. The device can be turned on and off by the patient using a hand-held remote control. [44].

A study of 15 patients being treated with upper airway stimulation (UAS) examined hypoglossal stimulation effects on retropalatal and retrolingual dimensions. Comparisons of the airway during awake laryngoscopy and drug-induced sleep endoscopy (DISE) found that unilateral stimulation of the hypoglossal nerve, timed with ventilation, leads to multilevel increases in airway area [45]. The study demonstrated increases in the anterior-posterior area of the retropalatal airway area by 180% and retrolingual area by 130% with stimulation at a therapeutic amplitude during DISE compared to wakefulness with progressive increases in area with higher amplitudes of stimulation. During awake endoscopy, both responders (defined as a 50% reduction from baseline AHI and treatment AHI < 20) and nonresponders had significant changes in retrolingual area; however, on DISE "responders" to treatment were also found to have larger, statistically significant retropalatal enlargement than nonresponders [45].

A small retrospective study of 14 patients treated with UAS sought to examine the tongue motions associated with stimulation. Three motions were identified in the cohort: right protrusion, bilateral protrusion, and mixed activation (all other tongue motions). After 6 months, patients with bilateral protrusion were found to have a greater reduction in AHI than patients with mixed activation [46].

As per the device manufacturer, the Inspire device is indicated in patients of ages 22 or greater, in those with moderate-to-severe OSA with AHI of 15–65 with less than 25% central apneas, and in those without complete concentric collapse of airway at the level of the palate. The device is indicated in patients who have failed PAP, defined as AHI > 15 despite PAP use, or in those with PAP intolerance defined as unable to use CPAP for >4 hours per night for at least five nights per week or unwillingness to use PAP. Treatment with hypoglossal nerve stimulation in patients with BMI > 32 is not recommended due to unknown effectiveness in this population as these patients were excluded in trials of the device.

In the original trials of the device, subjects with moderate-to-severe OSA with difficulty accepting or adhering to CPAP treatment were eligible for enrollment. Subjects were excluded for BMI > 32, with significant neurological, active psychiatric disease or cardiopulmonary disease. After initial screening with polysomnogram, subjects were excluded if AHI was less than 20 or more than 50 events per hour, if sleep disordered breathing comprised >25% mixed or central events, or if the AHI in a non-supine position was <10 per hour. Subjects were also excluded on the basis of airway anatomy for anatomical abnormalities preventing the effective assessment of stimulation or if complete concentric collapse of the retropalatal airway was observed on DISE [44].

The mean age of participants was 54.5 years with 83% male with mean BMI 28.4. Seventeen percent of participants had undergone prior uvulopalatalpharyngoplasty. At 12 months, 66% of subjects responded to therapy, as median AHI scores decreased 68% from 25.4 events per hour to 7.4 events per hour. The median ODI decreased 70% from 25.4 events per hour to 7.4 events per hour. Score on the Function Outcomes

of Sleep Questionnaire increased from 14.3 to 17.3, and Epworth sleepiness scale scores decreased from a mean of 11.6 to 7.0 at 12 months and were stable at 36-month follow-up. At 3 years, decreases in AHI remained stable with the average AHI of 14.2 at 36 months and with a median AHI of 7.3. Sixty-five percent of the cohort were deemed responders at 36 months defined as a least 50% reduction in AHI from baseline and treatment AHI of less than 20 [47].

Analysis of baseline characteristics of long-term responders versus non-responders over the 36-month period demonstrated statistically significant differences in baseline AHI with responders' baseline AHI 28.8 versus 35.0 in nonresponders. Eighty-one percent of the subjects reported the use of nightly therapy at 36 months. Eighty-four percent reported use at least 4 days per week.

During clinical trials, 40% of the participants reported discomfort related to stimulation. By 3 years, reports of discomfort decreased to 24 from 80 in year 1. Throughout the first year, 21% reported tongue soreness. In most cases, this resolved after acclimatizing to therapy or after the device was reprogrammed. In some instances, a tooth guard was necessary. Temporary tongue weakness was experienced by 18% of participants, but no permanent tongue weakness was reported. At two instances, there were device-related serious adverse effects causing discomfort, necessitating repositioning of the neurostimulator. Most nonserious adverse effects were related to the surgical procedure and were expected postsurgical events.

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Surgical Management of OSA: Adenotonsillectomy

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15.1 Introduction: Background Information

Tonsillectomy with or without adenoidectomy (T&A) is one of the most common pediatric surgical procedures with over half a million procedures performed annually in the USA. Overall, the rate of T&A has nearly doubled from 1996 to 2006, despite a sharp decline in the procedure being performed for recurrent acute tonsillitis. This growth reflects the increased awareness of the morbidity of obstructive sleep apnea (OSA) in children and a resultant increase in T&A for this sleep disorder [1]. OSA prevalence is 6% in all children but 59% in obese children [2]. Up to 20% of children have sleep-disordered breathing (SDB) [3] that is a clinical diagnosis and includes a spectrum ranging from primary snoring to OSA. OSA is characterized by recurrent obstruction causing disruption in normal sleep architecture that often leads to periods of hypoxemia and is diagnosed with polysomnography (PSG). Adenotonsillar hypertrophy is the principal cause of SDB in children, and the most common indication for T&A.

15.2 Tonsil and Adenoid Anatomy, Physiology, Immunology, Purpose

Waldeyer's ring refers to a ring of lymphoid tissue within the pharynx including the lingual tonsils, palatine tonsils (tonsils), and pharyngeal tonsils (adenoids). The second branchial pouch forms the tonsil and its arches also known as the "tonsillar pillars." The anterior tonsillar pillar consists of the palatoglossus muscle, while the posterior tonsillar pillar is the palatopharyngeus muscle. The tonsils are located within the oropharynx just distal to the junction of the hard and soft palate. The palatoglossus (anterior pillar), palatopharyngeus muscles (posterior pillar), and superior pharyngeal constrictor muscles lie anterior, posterior, and lateral to the tonsil respectively. The main arterial supply to the tonsil includes branches from the facial, dorsal lingual, ascending pharyngeal, ascending, and lesser palatine arteries. The arterial supply is primarily inferiorly along the lower pole. Nerve supply is primarily from tonsillar branches of the glossopharyngeal nerve and the descending branch of the lesser palatine nerve [4]. Non-keratizing squamous epithelium lines a series of 10–30 mucosal invaginations forming crypts, thus increasing the surface area of the tonsillar epithelium. The tonsils function as a secondary lymphatic organ. Specialized "M" cells line the epithelium, internalizing antigens and initiating a predominantly antibody-driven B-cell adaptive immune response. The level of the immunologic activity of the tonsil is typically greatest between the ages of 3 and 10 years, after which time the tonsil begins to involute [5].

Two lateral primordia fuse together to form the midline adenoid tissue which lies within the nasopharynx medial to the torus tubaris, superior to Passavant's ridge, and posterior to the choana and posterior nasal septum. The main arterial supply includes pharyngeal branches of the ascending pharyngeal, ascending palatine, and maxillary artery with small contribution from the pterygoid canal and tonsillar branch of the facial artery. The pharyngeal plexus supplies innervation. The adenoid pad is lined with pseudostratified ciliated columnar epithelium. Like the palatine tonsil, the epithelium is plicated to increase the surface area of the epithelium. The adenoids are also a secondary lymphoid organ whose immunologic function mirrors that of the palatine tonsils [4].

15.3 OSA as an Indication for Adenotonsillectomy: Guidelines

Clinical Practice Guidelines in 2019 by the American Academy of Otolaryngology – Head and Neck Surgery (AAO-HNS) recommend T&A for OSA [5]. SDB has detrimental, well-known, long-term effects on patient health that include behavioral problems, diminished quality of life, enuresis, growth impairment, and decreased school performance. There is evidence for improvement in all of these areas post-T&A and therefore the procedure is recommended as the first-line treatment in children with adenotonsillar hypertrophy and SDB/OSA.

Clinical practice guidelines by the American Academy of Pediatrics, revised in 2012, also recommend T&A as the first-line treatment in children with OSA and adenotonsillar hypertrophy while CPAP should be considered in those with OSA but without adenotonsillar hypertrophy or if surgical risks are significant [6].

15.4 Preoperative Assessment

15.4.1 Physical Examination

Prior to proceeding with T&A, it is important to document tonsil size and perform a full head and neck examination. The most commonly utilized scale for assessing the tonsil size is the Brodsky grading scale [7]. Tonsils are given a grade of 1–4 with grade 0 denoting absence of tonsils (◘ Fig. 15.1). Tonsils encompassing ≤25% of the oropharyngeal airway (lateral dimension) is given a grade of 1, while tonsils occupying between 26–50%, 51–75%, and 76–100% of the oropharyngeal space are of grades 2, 3, and 4 respectively. Tonsil grade is often recorded as 1+, 2+, 3+, or 4+ instead of grade 1, 2, 3, or 4 and both should be considered synonymous.

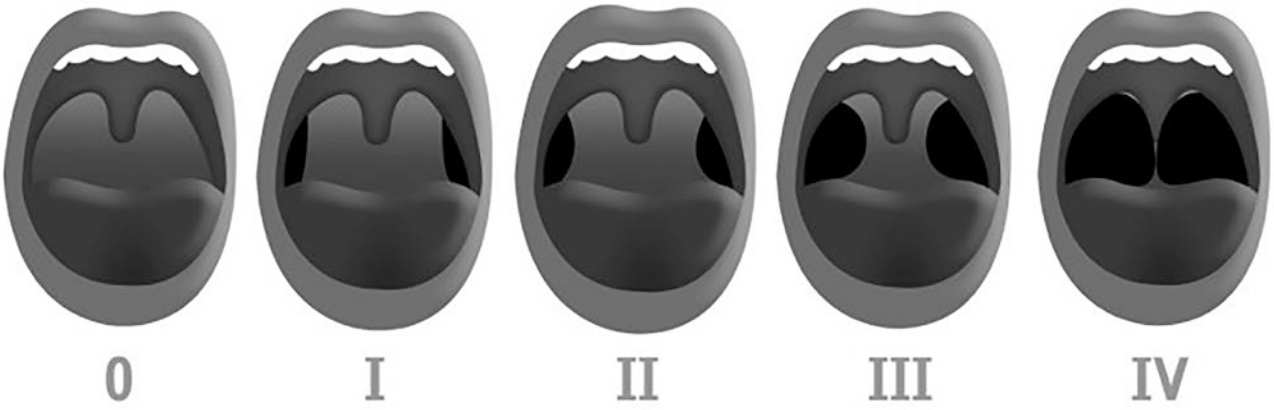

Fig. 15.1 Brodsky scale for grading tonsil hypertrophy [7]

The Friedman scale is the second most common grading scale. Tonsils are given a grade of 0–4. Tonsils not visible (often post-tonsillectomy) are given a grade of 0. Tonsils within the tonsillar fossa are given a grade of 1. Tonsils visible beyond the anterior pillars are grade 2. Tonsils extending 75% of the way to midline are grade 3. Completely obstructing ("kissing" tonsils) are grade 4 [8]. Both grading systems are limited due to the inability to account for endophytic tonsils that may obstruct the oropharynx significantly that is not visualized without endoscopy.

A recent study demonstrated higher mean intraobserver and interobserver reliability for the Brodsky grading scale than for the Friedman scale, 0.954 and 0.721 versus 0.932 and 0.647 respectively. The authors supported the adoption of the Brodsky scale for exclusive use in clinical documentation to make future research reporting uniform [9].

While tonsil size is important to record, its correlation with the apnea–hypopnea index (AHI), the principal measure of OSA severity, is complex. In a 2011 systematic review of tonsil size and OSA severity as measured by AHI, no correlation between tonsil size and OSA severity was reported. Of the 20 studies included, only four studies were high quality and all found no significant correlation between tonsil size and AHI. Although obese and syndromic children were excluded, there is no reason to assume that tonsillar size and OSA severity will correlate differently in these children [10].

In a 2015 retrospective case series of 70 patients with baseline AHI ≥ 5, Tang et al. showed that neither adenoid nor tonsil size correlated with OSA severity [11]. However, patients with larger Brodsky grade tonsils were more likely to have a resolution of OSA following T&A (AHI < 1). Overall resolution of OSA was seen in 25%, 50%, and 36% of children with 2+, 3+, and 4+ tonsils, respectively. Significant improvement in AHI and hypopnea index was seen in all tonsil size groups, while improvements in apnea index and oxygen saturation nadir were significant only in the 3+ and 4+ groups. Limitations included a lack of a large 1+ tonsil cohort and a lack of objective measurements, such as volumetric analysis.

Unlike subjective grading methods, objective tonsil size measures have been shown to correlate with OSA severity. In a study by Howard and Brietzke, tonsil weight (as measured postoperatively) was significantly correlated with preoperative AHI, but subjective adenoid size, tonsil size (Brodsky grade), Mallampati score, or any of the pharyngeal measurements were not [12]. However, this is complicated by the fact that objective measurements of tonsil volume and weight correlated well with subjective tonsil measurements [12]. Thus objective tonsil size is a better representation of airway constriction particularly with endophytic tonsils, but can generally only be done postoperatively.

For a more accurate measurement, some advocate for endoscopic analysis of tonsil size. In 2017, Patel et al. proposed a novel endoscopic tonsil grading system comprising measurements in the anterior-posterior and medial-lateral dimensions performed in 50 patients prospectively [13]. Grade 1 was given if tonsil occupied 0–25% of oropharyngeal width to midline or depth, while 26–50%, 51–75%, and 76–100% correlated with grade 2, 3, and 4. A grade number was given to both dimensions and an average of the two was taken to determine the final grade. This system was compared to the Brodsky scale, modified Brodsky scale (with tongue depressor), and Parikh adenoid grading scale (reviewed below). All scales had good interrater reliability: 0.83 for the modified Brodsky scale, 0.89 for the Brodsky scale, 0.94 for the Parikh scale, and 0.98 for their newly proposed endoscopic scale. They also studied the correlation of the different scales with quality of life, as measured by the obstructive sleep apnea-18 (OSA-18) and the BMI. The OSA-18, a subjective quality of life (QOL)

instrument with a maximum score of 126, assesses physical symptoms, daytime functions, sleep disturbance, emotional distress, and caregiver concerns [14]. They found that neither BMI nor the OSA-18 correlated with tonsil size using any of the scales, but adenoid size did correlate with the OSA-18 score.

Adenoid size is usually graded based on obstruction of the nasopharyngeal airway by flexible endoscopy in clinic, on lateral X-ray findings, or at the time of T&A. Intraoperatively, the percent of obstruction of the choana by the adenoid pad is graded from 0% to 100% in a similar manner to the Brodsky tonsillar scale (grade 1: 0–25%, grade 2: 26–50%, grade 3: 51–75%, and grade 4: 76–100%). With endoscopy, adenoid size preoperatively is most accurately graded by assessing the percent of obstruction caused by adenoid hypertrophy on surrounding structures, including the vomer, soft palate, and torus tubaris. Parikh et al. proposed a new endoscopic grading system based on contact of the adenoid pad with surrounding structures. When the adenoid pad was completely isolated abutting no structures, it was given a grade 1. Grade 2 assignment was given if torus tubaris was obstructed, grade 3 if torus tubaris and vomer were obstructed, and grade 4 if torus tubaris, vomer, and soft palate at rest were obstructed. Its use was validated with an intergrader agreement Kappa score of 0.71 (0.62 for residents and 0.83 for consultant physicians) indicating good reproducibility of grading between participants [15].

15.4.2 Polysomnography

Polysomnography (PSG) is the gold standard for the diagnosis and quantification of OSA. However, PSG is expensive, cumbersome, and often unavailable in children. A 2004 systematic review found the diagnostic accuracy of history and physical exam in predicting OSA to be only 55% when compared to PSG [16]. Nonetheless, it is not routinely performed in the majority of children prior to T&A. In fact, PSG is only obtained in about 10% of children undergoing T&A for SDB [11]. In a survey of pediatric otolaryngologists, 17% did not have access to a pediatric sleep laboratory, and the average wait time was over 6 weeks [17].

However, PSG should be obtained prior to T&A in certain populations of children. The American Academy of Otolaryngology – Head and Neck Surgery, in 2011, published guidelines on the indication for PSG prior to T&A [18]. PSG was recommended routinely in children with obesity, Down syndrome, craniofacial abnormalities, neuromuscular disorders, sickle cell disease, or mucopolysaccharidoses. This reflects increased perioperative risks and the likelihood of persistent OSA after T&A in children with significant co-morbidities. The guidelines also recommend obtaining a PSG if tonsil size does not correlate with reported severity of symptoms, that is, small tonsils and severe symptoms, or need for surgery is unclear. Postoperative PSG is recommended in children with severe OSA or persistent symptoms.

Guidelines have also been published by the American Academy of Pediatrics and the American Academy of Sleep Medicine that differ from those published by the American Academy of Otolaryngology – Head and Neck Surgery and reflect more routine use of PSG prior to T&A. The AAP guidelines published in 2012 recommend obtaining a PSG in all children with symptoms and signs of OSA or referring to a specialist, especially when PSG is not immediately available, and to repeat the PSG post-T&A in patients who are obese, have OSA sequela, significant OSA preoperatively or remain symptomatic [19]. The AASM guidelines, published in 2011, state that PSG is indicated preoperatively when T&A is considered for OSA, post-T&A when symptoms persist in patients with preoperative mild OSA, and post-T&A in all patients with preoperative moderate–severe OSA, obesity, neurologic disorders, or craniofacial anomalies that narrow the upper airway [20].

15.4.3 Clinical History

When PSG is not obtained, the decision to proceed with T&A often relies on clinical history supported by an evaluation of tonsillar size. Many studies have been performed on the utility of several symptom-related instruments, mostly in the form of questionnaires, to successfully predict OSA. However, no tool has been shown to be specific and sensitive for diagnosing OSA.

Ishman et al. evaluated the ability of the OSA-18 quality of life instrument to predict OSA in comparison to PSG. Using a cut-off of a total symptom score of $\geq$60 (out of 126) and obstructive AHI of $\geq$1 on PSG, the OSA-18 had 100% specificity and 50% sensitivity in white children, while only 67% specificity and 56% sensitivity in non-white children. The sensitivity, also known as the true positive rate, determines the probability of correctly identifying those with the condition. Therefore, regardless of race, the OSA-18 had an unacceptably high false-negative rate. The specificity, also known as the true negative rate, determines the ability of a test to correctly identify those without the condition. In this study, the OSA-18 had an unacceptably high false-positive rate in non-white children but no false-positive rate in white children. Therefore, a score of $\geq$60 on the OSA-18 in white children is likely to correctly diagnose OSA. However, given the poor sensitivity, a score $\leq$ 60 on the OSA-18 does not rule out OSA, regardless of race. It was determined that the OSA-18 cannot be used in lieu of PSG to accurately predict OSA in either population [21].

Chervin et al. first analyzed the use of the 22-item Sleep-Related Breathing Disorder (SRBD) scale within the larger Pediatric Sleep Questionnaire (PSQ) to predict OSA. A score of 0.33 (33%) was indicative of OSA on PSG with a sensitivity and specificity of 83% and 87%, respectively, for patients with AHI ≥5, and a sensitivity and specificity of 88% and 87%, respectively, for patients with AHI ≤5 [22]. A follow-up retrospective analysis of their longitudinal study by the same group revealed an increased odds ratio of 2.80 for OSA (AHI ≥1) for a high SDRB score (1 SD above the mean). Utilizing the same SBRD cut-off value of 33%, OSA (AHI ≥1) was accurately predicted in 74% of cases with a sensitivity and specificity of 78% and 72% respectively. They also found that improvement in the SBRD score 1-year post-T&A more accurately reflected improvements in the Attention Deficit Hyperactivity Disorder (ADHD) scale than did PSG results [23], reflecting its ability to predict behavioral outcomes better than PSG. Both were equally effective at predicting improvement in daytime sleepiness and attention quotient.

These findings were replicated as part of the Childhood Adenotonsillectomy (CHAT) multi-institutional study. The SDRB portion of the PSQ predicted postsurgical improvement of subjective measures of morbidity including executive dysfunction, behavior, quality of life, and sleepiness. Those with greater symptom burden preoperatively were more likely to improve. In contrast, the severity of OSA on PSG did not independently correlate with improvement in these areas postoperatively. The authors reflected that the SDRB, a one-page questionnaire, is an easy, quick method of assessing the severity of many subjective OSA-related symptoms and should be utilized in addition to, not in place of, objective PSG measures in predicting the surgical response of a child with OSA following T&A [24].

In another CHAT study paper, demographic and physical exam data as well as three subjective questionnaires as filled out by the parents – PSQ, OSA-18, and Epworth Sleepiness scale (an 8-item questionnaire out of 24 points evaluating daytime sleepiness) – were analyzed for their ability to determine OSA severity, when compared with PSG [25]. Despite several correlations on their linear regression analyses, no statistical model accurately predicted OSA severity. They hypothesized that the subjective nature of the questionnaires may contribute to their poor efficacy as a predictive tool.

15.5 Preoperative Consent

Informed consent is an important and necessary part of any surgical procedure including T&A. Adequate discussion regarding the risks, benefits, and alternatives to T&A is important with ample opportunity for the caregiver to ask questions and receive well-informed answers. Any child old enough to partake in the decision should be included in the process. The specifics of the consent process vary between institutions, state, and country. However, risk discussion should include those of general anesthesia, airway fire, intraoperative and postoperative bleeding, need for blood transfusion, pain, perioperative respiratory complications (requiring the need for reintubation or non-invasive positive pressure ventilation), perioperative cardiac complications, death, nausea, vomiting, bad breath, referred ear pain, velopharyngeal fever, dehydration, reduced oral intake, prolonged hospitalization, readmission, delayed return to normal activities and/or school, regrowth of adenoids or tonsils, disturbance of taste, need for further surgery, atlantoaxial subluxation, velopharyngeal insufficiency, nasopharyngeal stenosis, continued SDB, change in voice/speech, damage to the teeth, lips, gums, tongue, pharynx, or eye [5]. In a prospective cohort study in 2016 in which the informed consent process was videotaped and the parents' ability to recall risks and benefits was assessed, only one-third of the surgical risks were recalled. Benefits were recalled easier than risks, with 11.9% of parents reporting that no risks were mentioned. Interestingly, parents who were less likely to recall surgical risk were more likely to proceed with surgery [26]. This highlights the importance of spending adequate time on counseling and documentation during the consent process, as well as having the consent witnessed by a non-partial party.

One of the most common risks includes post-tonsillectomy hemorrhage (PTH). The 2011 clinical practice guidelines report a rate of primary PTH (within 24 hours of surgery) and secondary PTH as 0.2–2.2% and 0.1–3%, respectively [5]. A 2017 comparative effectiveness review of 104 studies of low-to-moderate risk bias including 6299 children reported an average PTH of 4.2% for total tonsillectomy and 1.5% for partial tonsillectomy. PTH was greater for those undergoing tonsillectomy for SDB than those for recurrent infections. However, significant overlap in the confidence intervals precludes any definitive conclusions [27]. Readmission rate in most studies was less than 5%. In a larger data sample of 1,778,342 children, four deaths were reported following tonsillectomy. No one surgical instrument technique provided significantly better rates of PTH.

15.6 Preoperative Assessment

15.6.1 Surgical Setting

Many patients can undergo T&A safely as an outpatient including at a free-standing surgical center. Determining which patients can undergo surgery

outside of a hospital setting is crucial. Any patients with severe OSA (AHI > 10), Down syndrome, cerebral palsy, sickle cell disease, neuromuscular disorders, craniofacial abnormalities, obesity (body mass index >30 or >95th percentile BMI z-score), failure to thrive, recent respiratory infection, major heart disease, bleeding diatheses, age less than three, or any other significant co-morbidity should be observed overnight for increased risk of complications [5]. Inpatient setting after T&A may also be considered for those who live a far distance from a medical center or those with a higher American Society of Anesthesia Class score (3 or greater) [28].

In addition to postoperative overnight observation, patients with sickle cell disease are usually admitted 24 hours preoperatively for aggressive hydration and transfusion with a goal of <40% hemoglobin S ratio and/or >100 g/L hemoglobin level. Their pain should be well-controlled postoperatively and fluid regimen adequate to avoid a sickle pain crisis [28].

15.6.2 Special Laboratory Evaluation or Imaging

In general, no routine preoperative laboratory analysis is obtained prior to T&A in children unless there is a significant bleeding history, or a personal or family history of a bleeding disorder. There have been several studies investigating the utility of obtaining routine preoperative coagulation studies such as activated partial thromboplastin time (aPTT), prothrombin time (PT), and/or international normalized ratio (INR) prior to T&A to exclude a risk of hemorrhage [29–31]. The studies report low sensitivity and specificity and show that routine preoperative coagulation studies are not cost-efficient. Screening with coagulation studies and/or a hematology consult may be warranted if clinical history suggests a major bleeding episode and/or bleeding disorder. The presence of a coagulation disorder should not be an absolute contraindication to T&A and is based on the risks and benefits for the individual child. In a recent retrospective review, only 1 of the 14 patients with an identified hematologic disorder experienced a postoperative bleed [32].

Routine imaging is also not performed. However, patients with Down syndrome should undergo preoperative cervical spine flexion, extension, and lateral X-rays as well as a neurologic exam. Approximately, 10–20% of Down syndrome patients are at risk of atlantoaxial subluxation which can lead to permanent neurologic deficits. Therefore, any patients with neurologic deficits on exam or atlantodental interval >4.5 mm should be referred to a spine specialist [33].

15.6.3 Screening Tools for Identifying At-Risk Children in the Perioperative Period

In order to properly counsel patients and their families, it is important to try and predict which children may be at increased risk for perioperative respiratory adverse events (PRAE), especially when PSG has not quantified OSA severity. Tait et al. investigated the predictive value of individual questions within the SRBD questionnaire as part of a standardized approach to quickly identify children at risk for PRAE. They found that answering yes to five questions pertaining to the child's sleep at night (snoring loudly, snoring more than half the night, struggling to breath, witnessed apneas, and awakening unrefreshed) to be strongly indicative of PRAE. The STBUR scale was developed from this, isolating these five questions within the SBRD, to identify at-risk children [3]. PRAE likelihood increased threefold if three questions were true (answered yes) and tenfold if all five questions were true.

Similarly, in another multidisciplinary study involving anesthesiology, pulmonology, and otolaryngology, six questions from the PSQ reliably identified children with OSA that had perioperative complications leading to a prolonged post-anesthesia care unit (PACU) stay and supplemental oxygen need [34]. This short PSQ questionnaire had a sensitivity of 89% and specificity of 41% for identifying OSA when compared with PSG. Both OSA on PSG and a score of >2 out of 6 on the questionnaire were significantly associated with the need for supplemental oxygen in PACU, while neither was associated with a prolonged PACU stay.

15.7 Tonsillectomy Technique: Extracapsular Versus Intracapsular

The modern and most common method of tonsillectomy performed is extracapsular (ECT), also known as complete tonsillectomy. In this method, the tonsil is fully removed by dissecting in a bloodless fascial plane outside of the tonsillar capsule and medial to the pharyngeal musculature (superior pharyngeal constrictor, palatoglossus, and palatopharyngeus). Popularized by Fuller in 1930 [35], in order to perform the technique correctly and in a bloodless fashion, suture ligation or cauterization of feeding vessels is required with the latter being more common today.

Postoperative pain and hemorrhage are the two major postoperative concerns after performing tonsillectomy. Depending on the electrocautery device selected, the heat dissipation within the tonsillar bed can reach up

to 400 °C and may spread to surrounding tissues. This is thought to be a major cause of the postoperative pain associated with tonsillectomy [36]. The pain associated with extracapsular tonsillectomy does not subside until the pharyngeal musculature is remucosalized [35].

Due to the concerns over postoperative pain associated with cautery techniques, intracapsular tonsillectomy (ICT) also known as tonsillotomy or partial tonsillectomy is regaining popularity. Once popular in the early twentieth century, it was deserted due to concerns over the residual tonsil causing reinfection and an increase in sequela such as rheumatic or scarlet fever [36]. Koltai et al. proposed that decreased pain with ICT would occur by leaving a small amount of tonsillar tissue on the tonsillar bed/pharyngeal musculature that acts as a "biological dressing" reducing inflammation and subsequent pain. He also hypothesized an inverse relationship between post-tonsillectomy hemorrhage and the amount of tonsillar tissue removed, arguing that the diameter of the vessels (entering at the capsule) is larger, the deeper (or lateral) one gets into the tonsil [35].

Proponents for ICT argue that it reduces postoperative pain and thus unplanned admissions for pain and/or dehydration. A recent meta-analysis by Kim et al. supported this by showing ICT (adenoidectomy included) performed with a microdebrider significantly reduced postoperative pain, readmissions, analgesia amount, and days to normal diet and activity as compared to extracapsular techniques [37]. A separate meta-analysis of 15 studies by Lee et al. investigated the efficacy of ICT (645 individuals) versus ECT (620 individuals) for the management of OSA. In this meta-analysis, a comparison between microdebrider and Coblator ICT technique revealed no difference in postoperative pain or bleeding outcomes. Similarly, they found significantly reduced postoperative pain, postoperative bleeding, analgesic use, days until normal activity, and diet resumed within the ICT group as compared to the ECT group [38].

Advocates for ECT argue that ICT leads to a significant increase in tonsillar regrowth, which could obscure the benefit of tonsillectomy for OSA. Both meta-analyses demonstrated a significant increase in tonsillar regrowth [37, 38], with a relative risk ratio of 6.02 in the ICT group versus ECT group in one meta-analysis [38]. In a multi-center retrospective case series of 870 children undergoing microdebrider ICT, Solares et al. showed a regrowth rate of only 0.46% but over a relatively short follow-up period of 14 months [39].

The clinical significance of tonsillar regrowth is unknown as no prospective, randomized controlled trial has been performed evaluating postoperative polysomnography (PSG) results between ICT and ECT cohorts. In a recent case series of 70 children undergoing microdebrider-assisted ICT significant reductions in AHI, mean and nadir oxygen saturation were seen between preoperative and postoperative PSG. However, the study lacked a comparison ECT or control group [40]. In another 2016 retrospective review of the efficacy of microdebrider-assisted ICT versus ECT on postoperative PSG parameters in OSA, an ECT cohort of 52 children, who were significantly more obese and older, were compared to an ICT cohort of 37 children. Both ECT and ICT cohorts had high postoperative OSA cure rates of 79% and 76% respectively, but the follow-up in this study was short with a small study population and lack of control for age and obesity. Furthermore, children with neurological or craniofacial disorders were excluded [41]. The largest retrospective review included 75 ICT and 93 ECT patients. As in the previous study, the ICT cohort was significantly younger and less obese, while improvements in AHI, oxygen saturation nadir, and postoperative complication rates were similar for the two groups. The only postoperative complication that was significantly different between the groups was tonsillar regrowth, 2.2% versus 0% in the ICT and ECT cohorts respectively [42].

In Lee et al.'s meta-analysis, tonsillar regrowth did not have adversely affect or worsen AHI [37]. However, most of the studies in both meta-analyses utilized the OSA-18 to evaluate the clinical impact of tonsillar regrowth. Both meta-analyses revealed no difference in the quality of life when utilizing these instruments between the ICT and ECT cohorts [37, 38].

While data from preliminary case series and retrospective cohort reviews show promise in the success rates in OSA cure after ICT, all studies lack a large enough sample size or long enough follow-up. Therefore, it is imperative to have higher level quality of evidence on the effect of tonsillar regrowth on postoperative AHI in normal, overweight, and obese children as well as those with medical co-morbidities with OSA prior to adopting ICT as an equal or superior method.

15.8 Instrumentation

15.8.1 Tonsillectomy

There are a variety of surgical instruments used to perform T&A including, but not limited to, bipolar radiofrequency ablation, monopolar electrocautery, bipolar electrocautery, microdebrider, harmonic scalpel, thermal wielding, KTP or CO_2 laser, ultrasonic dissection, and cold steel techniques (Snare). The most common techniques are reviewed here.

Bipolar radiofrequency ablation, also known as coblation or plasma-mediated ablation, has become an increasingly popular technique since being introduced in 1998. The Coblator creates tissue dissociation by

producing an alternating current within a sodium-rich medium such as isotonic saline producing a maximum thermal temperature of 70 °C [43].

In a 2017 Cochrane review including 29 studies and 2561 participants undergoing extracapsular dissection, coblation was compared with other surgical techniques for tonsillectomy to determine intraoperative and postoperative morbidity as well as cost [44]. Heterogeneity between studies precluded conclusions on cost, need for reoperation or postoperative infections. There was equal risk of primary (risk ratio (RR) = 0.99; 95% confidence interval (CI) 0.48–2.05) and elevated secondary (RR = 1.36; 95% CI 0.95–1.95) post-tonsillectomy hemorrhage rates, lower pain rates at day 1, and equal pain rates at day 7 but the quality of the evidence was poor. The evidence supporting coblation over other techniques is lacking but costs are often higher. However, the evidence that does exist suggests it of equal efficacy and safety to other methods with possibly lower pain rates. Many favor this technique as the lower thermal injury is presumed to create less collateral tissue injury, while still obtaining hemostasis.

Monopolar electrocautery (i.e., Bovie) is another widely used technique. Monopolar electrocautery is a handheld unipolar device that utilizes a ground electrode placed on the patient to complete the circuit. As compared with the Coblator, the device does not require a saline-rich medium and creates its thermal effect by a unipolar electrode with direct current with temperatures reaching 600–700 °C [45]. It should not be used in patients with metallic implant devices such as a cochlear implant, defibrillator, or pacemaker.

The Agency for Healthcare Research and Quality 2017 systematic review of the literature regarding tonsillectomy reached few conclusions on the effectiveness of different surgical techniques. They identified four randomized controlled trials (RCTs) comparing coblation and electrocautery. Half of the RCTs found a return to normal diet and activity sooner in the coblation group, while the other half found no difference. In three small RCTs comparing monopolar electrocautery with cold steel dissection, return to normal activity and diet was quicker in the electrocautery group in one but in cold steel dissection in two RCTs. Therefore, the literature consists of mixed evidence regarding the effectiveness of different surgical techniques for tonsillectomy with very little high quality of evidence to support the use of one device over another.

15.8.2 Adenoidectomy

Similar to tonsillectomy, various instruments can be used to remove the adenoid pad. Traditionally, adenoids were removed with a curette – a blind, cold steel technique. When utilizing this technique, the tissue can be removed en bloc and a specimen obtained. Bleeding can be difficult to control and requires packing with a vasoconstrictive agent. Most utilize a more modern technique of indirect visualization with an angled mirror. Once visualized, the adenoid pad can be removed with suction electrocautery (suction bovie), bipolar radiofrequency ablation (Coblator), or microdebrider. However, the microdebrider also requires hemostasis via packing or cautery. Bleeding rates following adenoidectomy are markedly less frequent than after tonsillectomy.

15.9 Postoperative Management

15.9.1 Pain

Pain management after T&A varies between providers and institution. There are a variety of approaches to pain management that include over-the-counter and narcotic pain medications. Pain management should be started with over-the-counter analgesics (that are often prescribed) before the consideration of narcotics [5]. Abstaining from narcotics is especially important in obese children with severe OSA, as the sensitivity to opioid side effects such as respiratory depression is amplified. A safety investigation was launched by the FDA in 2012 following several deaths post-T&A in children receiving an appropriate weight-based dose of codeine [46]. In February 2013, the Food and Drug Administration (FDA) issued a black box warning following a safety investigation of the use of codeine after T&A in children under 12 years of age following reported deaths in a number of children deemed "ultra-rapid metabolizers." This refers to the highly polymorphic CYP2D6 enzyme, which is part of the P450 system responsible for conversion of the pro-drug codeine to morphine [47] (see ◘ Fig. 15.2). Normally, only 10% of codeine is converted into morphine. However, in "ultra-rapid metabolizers," a generally acceptable dose of codeine is converted to a larger, fatal amount of morphine in the liver. The ultra-rapid metabolizer phenotype incidence varies by ethnic group and is most common in those of Ethiopian, Arab, and North African descent [48]. In April 2017, the FDA expanded their warning against the use of both codeine for post-T&A pain control in children 12–18 years of age if they have OSA, chronic lung conditions, or are obese. The AAP has also issued a broad recommendation against the use of codeine in all children under the age of 18 as both an analgesic and antitussive [49].

A contraindication, the FDA's strongest warning, has also been issued for tramadol use in patients less than 18 years of age following T&A [50]. Tramadol, also a prodrug, is metabolized via the CYP2D6 pathway

Drug	Active or prodrug	Enzyme pathway	Active metabolites
Codeine	Prodrug	CYP2D6	Morphine
Morphine	Active	UGT2B7	M6G (Morphine 6-glucuronide)
Tramadol	Prodrug	CYP2D6	O-DMT (O-demethylated)
Hydrocodone	Active	CYP2D6 (major) CYP3A4 (minor)	Hydromorphone
Oxycodone	Active	CYP3A4 (major) CYP2D6 (minor)	Noroxycodone Oxymorphone

Fig. 15.2 Narcotic pain medicine properties

as well as to an active metabolite O-DMT that acts on the μ-opioid receptor. Severe respiratory depression following T&A in an "ultra-rapid metabolizer" has been published [51].

While there are multiple recommendations against the use of codeine and tramadol, there is relatively little published about the use of other narcotic pain medications in the post-T&A period. Hydrocodone and oxycodone are two oral narcotic medications often used in adults for pain control. Hydrocodone is an active drug with twice the potency of morphine whose major route of metabolism is also via the CYP2D6 pathway to create hydromorphone, also known as dilaudid [51]. This would lead to an eightfold greater concentration of hydromorphone in "ultra-rapid metabolizers." Furthermore, serious drug–drug interactions may occur due to its equal metabolism through the CYP34A pathway which is utilized by several different drug classes. A fatal overdose in a child taking clarithromycin (utilizing the CYP34A pathway) concurrently with hydrocodone has been reported [52]. Oxycodone is also an active drug metabolized primarily through the CYP34A enzyme pathway and minimally via the CYP2D6 pathway. While this may lessen the risk of opioid toxicity in ultra-rapid metabolizers, data regarding its safety in children are lacking. Both are schedule II drugs (as of 2014 for hydrocodone). Schedule II drugs cannot be called in, faxed, emailed, or refilled [53]. The AAP recommends against the use of narcotic analgesics when outpatient pain control is needed given the relative similarities between the drugs and lack of safety information in children [49].

In the 2011 clinical practice guidelines from the American Academy of Otolaryngology – Head and Neck Surgery (AAO-HNS) [5], weight-based dosing of over-the-counter medication was recommended for post-T&A pain control delivered via a scheduled basis orally or rectally if oral medications are refused. There is no evidence to support better pain control from the utilization of medication on a scheduled rather than an as-needed basis. However, caregiver compliance is vital to the achievement of proper postoperative pain control, and caregivers may be more vigilant if given instructions to dispense medications on a scheduled basis. They also emphasized the need for caregiver education to encourage pain assessment frequently in the postoperative period. Both ibuprofen and acetaminophen are recommended by the AAO-HNS as over-the-counter analgesics for post-T&A control [5]. The use of perioperative local anesthetics, antibiotics, ketorolac, or topical agents was not recommended.

Once debated, the use of non-steroidal anti-inflammatory medications is not associated with an increased risk of PTH. A Cochrane review in 2005 including 13 randomized controlled trials with about 1000 children found no significant increased risk of PTH with the use of non-steroidal anti-inflammatory medications compared with other analgesics with an odds ratio of 0.91 for PTH requiring reoperation [54]. This excludes ketorolac which is thought to have a significantly higher risk of PTH ranging from 4.4% to 18% [5]. A recent multi-institution cross-sectional survey of caregiver's perceptions of post-tonsillectomy pain revealed superior pain control with ibuprofen as compared to narcotic use alone or with an ibuprofen/narcotic combination, though the children receiving ibuprofen were significantly younger [55].

15.9.2 Diet

Post-tonsillectomy diet recommendations are highly variable among surgeons despite a paucity of data to support one diet over another. Common variations include a fully liquid diet, soft diet, dairy-free diet, citrus-free diet, or unrestricted (regular) diet. A systematic review of post-tonsillectomy diet advice published in 2017 includes evidence from 17 articles, three of which were small RCTs in the 1990s. The review could not group the data into a meta-analysis due to the heterogeneity of the studies. However, all three RCTs found no difference between restricted and non-restricted diets in terms of postoperative pain, bleeding, or healing [56–59]. However, one study found lower pain scores in the group given ice-pops within 4 hours of surgery versus those who did not receive an ice-pop [59].

15.9.3 Follow-Up

All patients with OSA should be contacted 3 months following T&A to ensure symptomatic improvement. Roughly 75% will be asymptomatic and can be discharged [60]. If symptoms persist, a full head and neck exam should be completed including a flexible laryngopharyngoscopy to exclude upper airway obstruction and specifically adenoidal obstruction. A trial of nasal saline and steroid spray should be started and an allergy evaluation considered. Repeat PSG should be obtained in cases when there is concern for persistent OSA, particularly in the medically complex children, that is, those who are obese or with Down syndrome, craniofacial, or neuromuscular disorders.

15.10 Expected Outcomes by Population

T&A does not always normalize OSA. Higher rates of persistent OSA occur in children with obesity, neuromuscular, craniofacial, or chromosomal disorders. Additional caregiver counseling is needed in these children.

15.10.1 General Population

Within the general pediatric population, the efficacy of T&A to resolve OSA (definition ranges from AHI reduced less than 1 to less than 5 depending on the study) was reported to be 82.9% in a 2006 meta-analysis. A mean reduction of AHI by 14 events per hour was reported [61]. Similarly, in a prospective cohort study of 79 healthy children, OSA resolution following T&A was 90% when defined as AHI <5 and 71% when defined as AHI <1 [60]. A 100% resolution of OSA occurred in all children with a preoperative AHI ≤ 10. Also, persistent snoring was reported in 28% of children after T&A and all with persistent OSA were symptomatic.

15.10.2 Complex Children

In a 2009 meta-analysis, including 23 studies, Friedman et al. reported on the cure rate of OSA following T&A [62]. Nine studies included "complicated" patients defined as morbid obesity, having severe OSA and/or under the age of 3. The cure rate was 66% when defined as AHI <5 and 60% when defined as AHI <1. The mean preoperative and postoperative AHI was 18.6 and 4.9 respectively. The cure rates for uncomplicated and complicated patients were73.8% and 38.7% respectively. Despite this, the overall mean change in AHI from preoperative to postoperative was greater in the complicated patients than in the uncomplicated patients (22 versus 12). The study showed that improvement in OSA occurs regardless of patient population, but resolution is less likely in certain populations particularly in children with morbid obesity.

15.10.2.1 Obese Children

In another 2009 meta-analysis assessing the cure rate in obese children, T&A improved OSA severity with a weighted mean decrease in AHI of 18.3 events and mean increase in oxygen saturation nadir of 6.3%. However, T&A was curative in only 12% of cases (reduced AHI <1) [63]. This is an important consideration given that the rate of pediatric obesity (defined as BMI at or above the 95th percentile of the sex-specific CDC BMI-for-age charts) in the United States as of 2012 data is 16.9% [64], but the prevalence of OSA in those with obesity is 59% [2].

15.10.2.2 Down Syndrome

Children with Down syndrome have a prevalence of OSA of 57–66%. T&A is a common procedure in children with Down syndrome in a pediatric otolaryngology practice [65]. However, the rate of incomplete resolution mirrors that of the obese population and this must be considered when counseling caregivers about the best intervention. In a study of 27 patients with Down syndrome, OSA resolved in 29.6% of patients, while 44.4% had at least a 50% reduction in AHI [65]. Greater reduction in AHI was seen in those with more severe OSA (higher AHI) preoperatively while it worsened with hypothyroidism. They also noted worsening of the central apnea index (CAI) in patients with congenital heart disease (in 71%) and hypothyroidism (in 32%).

In a recent 2017 systematic review of T&A for OSA in children with Down syndrome, 51% had improvement in AHI [66]. The improvement in AHI was equal regardless of initial OSA severity. Within the qualitative analysis, several studies revealed no change in sleep efficiency, sleep stage distribution, or arousal index despite improvement in AHI, while up to 75% required postoperative positive airway pressure (PAP) or nocturnal oxygen [67].

Many factors contribute to incomplete resolution of OSA in children with Down syndrome. These include a narrowed airway, macroglossia, lingual tonsillar hypertrophy, propensity for collapsibility of airway, and a myriad of comorbidities such as congenital heart disease, hypothyroidism, obesity, and lung disease [66]. All of this should be considered when counseling caregivers. Despite this, T&A is considered a first-line treatment in these children but with a high likelihood of persistent OSA.

15.10.2.3 Craniofacial Syndromes

In children with congenital craniosynostosis, the prevalence of OSA is estimated to be 40–85%. The management of OSA in these children is complex due to the multilevel airway narrowing, in the nasopharynx, oropharynx, and hypopharynx. Surgical options include T&A, midfacial advancement, and/or tracheostomy. In a 2013 review of 47 children with Apert, Pfeiffer, and Crouzon, 83% had OSA and 62% underwent T&A [68] with no significant change in AHI in the 45% who had preoperative and postoperative PSG. In three patients, AHI worsened. While there are several studies investigating the efficacy of T&A in this patient population, this was the only one comparing preoperative and postoperative PSG results. The authors did not recommend T&A as first-line surgical treatment in these children, though the lack of data in the majority of the cohort suggests a selection bias with the more symptomatic children undergoing post-T&A PSG.

15.10.2.4 Synchronous Airway Lesion

A retrospective chart review in children less than 3 years of age reported that the presence of a synchronous airway lesion, as identified in 8 of 15 children undergoing flexible laryngoscopy, direct laryngoscopy, or bronchoscopy, was not associated with failure to cure OSA with T&A [69]. Instead, children with higher preoperative AHI and lower oxygen saturation nadir were identified as the at-risk population for residual OSA.

15.10.3 Hypopnea Versus Apnea Predominant

In a 2016 case series investigating OSA resolution rates following T&A, no difference was seen between those with hypopnea predominant OSA and those with apnea predominant OSA [70]. AHI, apnea index (AI), hypopnea index (HI), and oxygen saturation nadir also improved regardless of the race following T&A. Their resolution rate of 41% was lower than previously described in the literature. This was attributed to the inclusion of children with obesity, asthma, and those greater than 12. Low baseline AHI was the only predictor in the normalization of AHI.

15.11 Outcomes: QOL, Cognition, Behavior, Cardiovascular Parameters

15.11.1 Quality of Life

The impact of T&A for OSA on a child's quality of life has been extensively studied. A positive correlation between OSA severity, as measured by AHI, and quality of life (QOL) scores has not been demonstrated [71]. However, overall improvement in QOL following T&A has been shown. In a 2008 meta-analysis, a significant improvement in OSA-18 scores was seen in children undergoing T&A both in the short- (<4 weeks) and long terms (>6 months) [72]. All five individual domain scores within the OSA-18 – sleep disturbance, physical suffering, emotional distress, daytime problems, and caregiver concerns – were significantly improved after T&A. A more recent meta-analysis, in 2013, confirmed these findings [73]. The authors cautioned that only one study utilized a control group of children with OSA that did not undergo T&A, while the others used healthy children undergoing T&A for recurrent infections as controls. In the 2015 CHAT trial, children with OSA were randomized to T&A or watchful waiting over a 7-month period. QOL was significantly more improved in the surgical group compared with the watchful waiting group at 7 months for all four of the parental questionnaires including the OSA-18, SRBD scale of the PSQ, modified ESS, and the Pediatric quality of life inventory (PedsQL). Unlike previous studies, a true control group was utilized [74]. Furthermore, improvements in QOL were not correlated to baseline OSA severity, that is, patients with mild OSA received equal QOL benefit post-T&A than those with severe OSA. Further studies with longer follow-up are needed for definitive evidence. However, the best evidence to date suggests improvement in both short- and long-term QOL scores after T&A for OSA.

15.11.2 Cognition and Behavior

Improvements in behavior and neurocognition after T&A for OSA are less dramatic than those in QOL. In the CHAT trial, no significant difference in the Developmental Neuropsychological Assessment [NEPSY] was seen between the observation and T&A groups over a 7-month period following T&A for OSA [75]. Further analysis of the neuropsychological data in the CHAT trial of 23 individual tests, assessing 5 main domains of function, showed only slight significant improvements in two tests in the T&A group, one in nonverbal reasoning, and the other in fine motor skills [76]. The current body of evidence suggests no significant improvement in overall cognitive abilities in children undergoing T&A for OSA, at least not in the first 6 months.

Behavioral issues are prevalent in children with OSA. Children are often more hyperactive and have poorer school performance [77]. Many studies have reported improvements in behavior after T&A for OSA. In a prospective study of 23 children with PSG-proven OSA (AHI >5), behavior was measured using

the Behavior Assessment System for Children (BASC) as completed by caregivers prior to surgery, 6 months and 9–18 months following surgery [78]. The BASC specifically assessed aggression, atypicality, depression, hyperactivity, somatization, and overall behavior symptom index. Children with psychiatric disorders and developmental delay among others were excluded. Behavior was shown to significantly improve in all subscales and overall BSI at both 6 and 9–18 months following T&A. However, the improvement at 9–18 months was less pronounced than at 6 months implying some regression to the mean.

The CHAT trial also investigated behavior as assessed by the caregivers and teachers with both the Conners' Rating Scale (assessing restless-impulsive and emotional lability) and the Behavior Rating Inventory of Executive Function [BRIEF] (assessing behavioral regulation and metacognition) [75]. Significant improvements in the BRIEF and Conners' rating scale were noted in the T&A as compared to the observation group as reported by the caregiver. While only significant improvements in the Conners' rating scale were noted in the T&A as compared to the observation group as reported by the teacher. Interestingly, in both the BRIEF and Conners' rating scale, caregiver reported scores were higher than teacher-reported scores in both the T&A and observation groups both at baseline and at 7 months. Overall, the literature to date suggests that T&A improves behavior more than that seen by observation.

15.11.3 Cardiovascular Parameters

The deleterious long-term effects of OSA on cardiovascular health are well studied in the adult population, which is a main point in counseling patients and their families on the need for effective treatment of OSA. A 2013 systematic review of 14 studies investigating the impact of T&A on cardiovascular health in children showed significant improvement in diastolic blood pressure, mean pulmonary artery pressure, variability in heart rate, and right and left ventricular function [79]. Recently, a 2017 meta-analysis demonstrated similar findings with significant improvement in mean pulmonary artery pressure, heart rate, mitral valve function, right ventricular diastolic diameter, and C-reactive protein [80]. Conversely, in the prospective, randomized controlled Childhood Adenotonsillectomy Trial (CHAT) over a 7-month period, there was no significant change in cardiovascular parameters between those undergoing T&A and observation [81].

Overall, there is mixed evidence regarding the effects of T&A for OSA on cardiovascular health but the overall evidence supports a positive impact.

15.12 Comparison to Conservative Management

To date, three RCTs compared the safety and effectiveness of T&A with conservative (non-surgical) management of OSA in children aged 2–16 and were analyzed in a 2015 Cochrane review [82].

The Childhood Adenotonsillectomy (CHAT) trial, published in 2013, was the largest with the lowest risk of bias. The CHAT study involved randomization of 464 school-aged children (5–9 years of age) with mild–moderate OSA to T&A or conservative management (watchful waiting) with follow-up over a 7-month period. Significant findings included that T&A leads to an improvement in QOL, caregiver reported behavior, and symptoms compared to non-surgical intervention in these school-aged, healthy and obese, non-syndromic children with mild-to-moderate OSA. However, no improvement in attention or neurocognitive performance was seen in the surgical compared to the conservative (non-surgical) cohort as measured by the Developmental Neuropsychological Assessment [NEPSY]. Approximately 79% of children in the T&A cohort had normalization of PSG versus 46% in the non-surgical cohort. In both cohorts, normalization of PSG findings was more likely in children that were non-obese, non-black, and with a baseline AHI at or below the median level of 4.7. Serious adverse events were similar, 3% and 4% respectively in surgical and non-surgical cohorts [75].

The Goldstein trial was also included in the Cochrane review and investigated the efficacy of T&A for children with a Clinical Assessment score (CAS) suggestive of OSA (>40) but a negative PSG by randomizing patients to a T&A or non-surgical cohorts [83]. The CAS encompasses nighttime and daytime symptoms, physical exam, sleep tape, echocardiogram, and lateral neck X-ray findings, and is weighted according to the likelihood of association with OSA. It has not been validated against PSG. While the median reduction in CAS was significantly greater in the T&A (−49) versus the non-surgical cohort (−8), the PSG recordings were similar in both groups at 6 months. The study was considered as having a low quality of evidence for recommending T&A over non-surgery in children with negative PSG [82].

The last randomized, cohort trial included in the Cochrane review was a trial by Sudarsan et al. This study compared outcomes in cohorts receiving continuous positive airway pressure (CPAP) or T&A in children with mucopolysaccharidoses (MPS) and Down syndrome (DS). No significant differences were reported in QOL by the OSA-18 score, but the mean modified Epworth Sleepiness Scale (ESS) was lower in the T&A cohort at 12 months. Rates of resolution of OSA were

similar between the groups. Complication rates were similar with a 5% rate of PTH in the surgical cohort and 3% rate of nasal dorsal rash in the CPAP group [84]. The study was considered as having a low quality of evidence for recommending T&A over CPAP in children with Down syndrome or mucopolysaccharidoses [82].

Lastly, it is important to note that most of the high-quality evidence in support of T&A for OSA is in children over the age of 5. This is despite a large portion of T&As for OSA being performed at ages 2–5, reflecting the lymphoid growth occurring over this time period.

15.13 Conclusion

T&A is a first-line treatment for OSA in most children. However, T&A does not resolve OSA in all children. The rate of resolution varies depending on the severity of OSA and other medical comorbidities. There is no evidence that one method of tonsillectomy is superior to another in terms of PTH, pain, days to a normal diet, or activity level. Further research focusing on the long-term outcomes of intracapsular tonsillectomy in OSA patients is needed. Caregivers can be counseled that T&A may improve the patient's quality of life, behavior, and health. However, there is little evidence to support an improvement in cognitive abilities.

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Further Reading

Baugh RF, Archer SM, Mitchell RB, Rosenfeld RM, Amin R, Burns JJ, et al. Clinical practice guideline: tonsillectomy in children. Otolaryngol Head Neck Surg. 2011;144(Suppl 1):1–30. https://doi.org/10.1177/0194599810389949.

The American Academy of Otolaryngology – Head and Neck Surgery evidence-based medicine guidelines for when to perform tonsillectomy in children, as well the perioperative management of these children. The paper also provides general information on the health care burden of tonsillectomy, overview of tonsil function and structure, risks and benefits of the procedure.

Francis DO, Chinnadurai S, Sathe NA, Morad A, Jordan AK, Krishnaswami S, et al. Tonsillectomy for obstructive sleep-disordered breathing or recurrent throat infection in children [Internet]. Agency for Healthcare Research and Quality. AHRQ Comp Eff Rev. 2017;16(17):EHC042-EF.

A comprehensive systematic review of 218 studies on tonsillectomy for sleep disordered breathing in children. Provides breakdown of evidence by population group, surgical technique and alternatives to surgery.

Kim JS, Kwon SH, Lee EJ, Yoon YJ. Can intracapsular tonsillectomy be an alternative to classical tonsillectomy? A meta-analysis. Otolaryngol Head Neck Surg. 2017;157:178–89. https://doi.org/10.1177/0194599817700374.

A recent meta-analysis comparing intracapsular and extracapsular tonsillectomy providing a good overview of the available literature for each technique.

Marcus CL, Moore RH, Rosen CL, Giordani B, Garetz SL, Taylor HG, et al. A randomized trial of adenotonsillectomy for childhood sleep apnea. N Engl J Med. 2013;368:2366–76. https://doi.org/10.1056/NEJMoa1215881.

A prospective, multi-center, single-blind, randomized, controlled trial of healthy children aged 5–9 years old undergoing adenotonsillectomy or watchful waiting for obstructive sleep apnea. Polysomnographic, attention and executive function, behavior and health outcomes were studied. There are several other follow-up papers investigating other outcomes published following the main trial utilizing the same data.

Roland PS, Rosenfeld RM, Brooks LJ, Friedman NR, Jones J, Kim TW, et al. Clinical practice guideline: polysomnography for sleep-disordered breathing prior to tonsillectomy in children. Otolaryngol Head Neck Surg. 2011;145(Suppl 1):1–15. https://doi.org/10.1177/0194599811409837.

The American Academy of Otolaryngology – Head and Neck Surgery evidence-based medicine guidelines for when to obtain polysomnography prior to adenotonsillectomy in children aged 2–18 as well as recommendations for inpatient observation following adenotonsillectomy based on the findings on polysomnography.

Nasal Obstruction and Sleep-Disordered Breathing

William C. Scott and David T. Kent

Contents

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16.1 Introduction

Many patients with sleep-disordered breathing (SDB) also suffer from significant nasal obstruction. In a study of over 5000 subjects, Young et al. found that individuals who reported symptoms of nasal congestion, especially at night, were more likely to complain of snoring, chronic daytime sleepiness, and nonrestorative sleep. Those who reported nasal congestion due to allergy were nearly twice as likely to have moderate-to-severe sleep apnea [1]. The nature of the relationship between nasal breathing and sleep is complex. This chapter examines this relationship in detail from an anatomic and physiologic perspective. It goes on to discuss the various treatments for nasal obstruction, particularly surgical techniques, and the extent to which those treatments improve sleep quality.

16.2 Anatomy of the Nasal Airway

The external nose is comprised of the nasal bones, the upper lateral cartilages, the cartilaginous septum, and the lower lateral cartilages. The shape of the nasal airway more posteriorly is defined by the relationship between the septum and the bony lateral wall of the nose. The septum is a rigid structure composed of the quadrangular cartilage anteriorly and a posterior bony portion made up of the perpendicular plate of the ethmoid bone, the vomer, and the maxillary crest along its most inferior border. The lateral wall contains the inferior, middle, and superior turbinates, which are soft tissue-covered bony structures that protrude medially.

Air inspired through the nose first passes through the external nasal valve. This passage is bounded by the lower lateral nasal cartilages, the nostril sill, and the caudal septum. The internal nasal valve, first described by Mink in 1903, is defined by the caudal end of the upper lateral cartilage, the nasal septum, and the head of the inferior turbinate [2]. It is commonly described as the narrowest segment of the upper airway, accounting for almost 50% of resistance to airflow in the nose [3]. Air passes through the internal valve and into the nasal cavity, passing around the nasal turbinates before entering the nasopharynx. (Fig. 16.1).

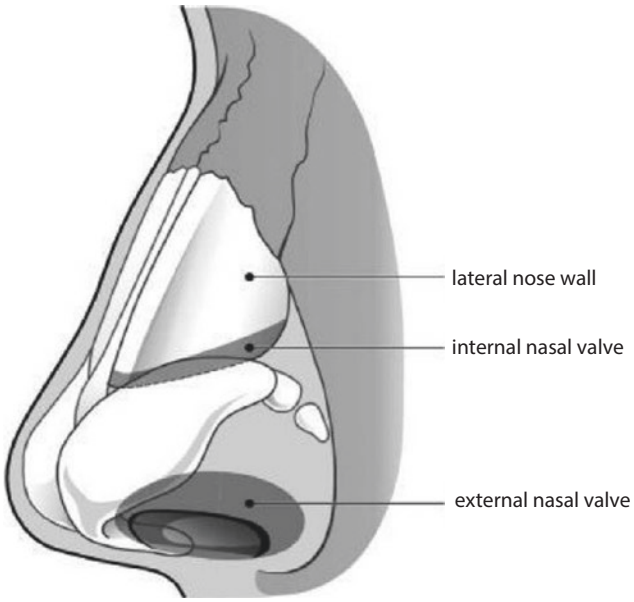

Fig. 16.1 The cartilages of the nose and the nasal valves.

16.3 Etiologies of Nasal Obstruction

The nasal airway contains rigid and soft tissue components, and causes of obstruction can therefore be static or dynamic. In many cases, obstruction is multifactorial and involves an interplay of unfavorable anatomic and inflammatory factors. Treatment requires a complete understanding of the various contributing etiologies and their interaction.

16.3.1 Anatomic Factors

Structural abnormalities and lack of rigidity of the external nose can affect airflow. Unlike the rest of the nasal airway, the external valve is composed of soft tissue elements that can dynamically collapse, completely obstructing airflow. Impingement of the internal nasal valve may further restrict flow through the area of highest natural resistance. The positioning and shape of the lateral cartilages vary between individuals and, in some patients, can lead to excessively narrow nasal valves. The strength of the lateral cartilages is also important. Due to the Venturi effect, the flow of air into the nose will tend to collapse the cartilages inward if they are not sufficiently rigid to hold their shape when inspiratory pressure exceeds the transmural pressure of the nasal wall [4].

Collapse or restriction of the external nasal valve can be caused by several primary and secondary factors. Narrow nasal valves can be congenital, either as a variant of normal anatomy or associated with anatomic abnormalities such as cleft palate, which can significantly distort the external anatomy of the nose [5]. Craniofacial deficits are common in SDB, with a narrow maxilla often limiting the potential width of the external nasal pyramid [6]. Anatomic deficits can also be acquired. As patients age, cartilage loses rigidity and the supporting musculature becomes less robust, increasing collapsibility [7]. Nasal trauma may distort the external anatomy of the nose and lead to unilateral or bilateral

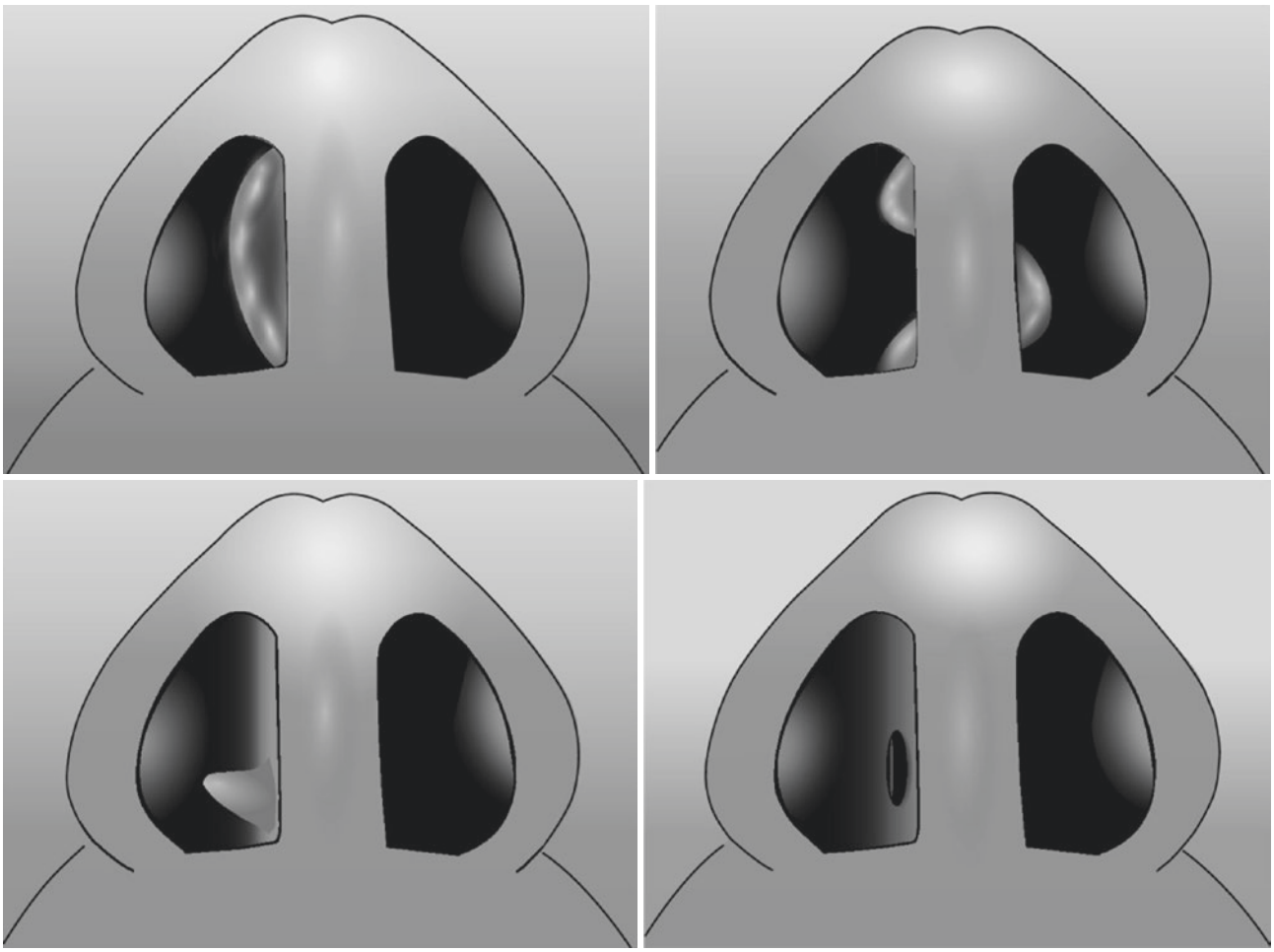

Fig. 16.2 Septal deformities, including an anterior-posterior" C shaped" deformity (top left), an anterior-posterior "S shaped" deformity (top right), a septal spur (bottom left), and a septal perforation (bottom right)

valve collapse. Significant burn and inhalational injuries can cause stenosis of the nasal valves [5]. Iatrogenic nasal valve collapse can occur following functional or cosmetic rhinoplasty. Sheen estimated that 75–80% of patients experience narrowing of the nasal valve after rhinoplasty, and Kosh et al. found that previous rhinoplasty was the cause of valve collapse in 79% of patients presenting for repair of nasal valve collapse [8, 9].

Nasal septal deviation is a common anatomic cause of obstruction frequently requiring surgical correction. A study of 2589 patients found that only 15.4% of women and 7.5% of men demonstrate a straight septum [10]. While most deviated septums are not clinically significant, septal deviation is still the most common etiology of unilateral or bilateral breathing difficulty. The nasal septum runs the length of the nasal airway and can be a cause for obstruction anywhere along its course, although most clinically significant cases involve anterior structural abnormalities. The septum forms the medial wall of the internal nasal valve and may limit this already narrow portion of the airway. Deviations often occur not as a single deflection, but as a complex series of deflections that can cause obstruction at multiple sites along the nasal airway. There have been many attempts to classify the different types of septal deflections. Teixeria et al. published a review comparing these different classification systems in 2016. The authors concluded that the most useful common denominator between these systems was thinking of septal deviations as "C shaped," "reverse C shaped," "S shaped," or "reverse S shaped" when viewed from either the anterior-posterior direction or the craniocaudal direction [11]. Septal deformities can also include "spurs" of bone or cartilage that protrude into the airway. Fig. 16.2 illustrates some common septal deformities.

Turbinate hypertrophy is a common structural cause of nasal obstruction that may be exacerbated by inflammatory disease. Structural issues are often secondary to an overly prominent inferolateral turn of the inferior turbinate resulting in medialization into the nasal air-

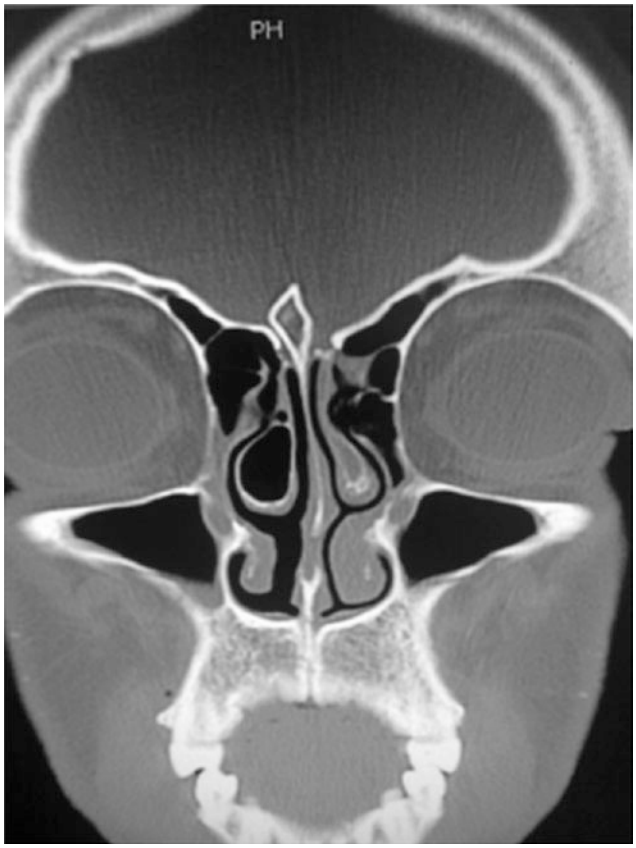

Fig. 16.3 Coronal CT view of a concha bullosa (patient right)

way [12]. The inferior turbinates are covered in a soft tissue envelope of pseudostratified, ciliated columnar epithelium. This tissue can swell as a result of changes in blood supply mediated by parasympathetic stimulation, which regulates the vasomotor tone and level of secretions [13]. Many environmental factors can cause turbinate hypertrophy including allergies, overuse of medication, and inflammatory disorders. Often, obstruction due to turbinate hypertrophy is a "mixed" picture of unfavorable bony anatomy and tissue hypertrophy secondary to inflammatory factors.

Other turbinate abnormalities are less common. Concha bullosa is an anatomic variant wherein the middle turbinate is pneumatized, resulting in enlargement and obstruction of the middle meatus. In a review of 998 patients who underwent sinus CT, Stallman and colleagues found that 44% had a concha bullosa on at least one side, with 21% demonstrating bilateral conchae (Figs. 16.3 and 16.4) [14]. A paradoxical middle turbinate curves medially toward the septum instead of the lateral nasal wall, resulting in middle meatus obstruction. Neither of these anatomic variants is pathologic in and of themselves, but both can contribute to nasal resistance, compounding problems in patients with other anatomic or physiologic factors.

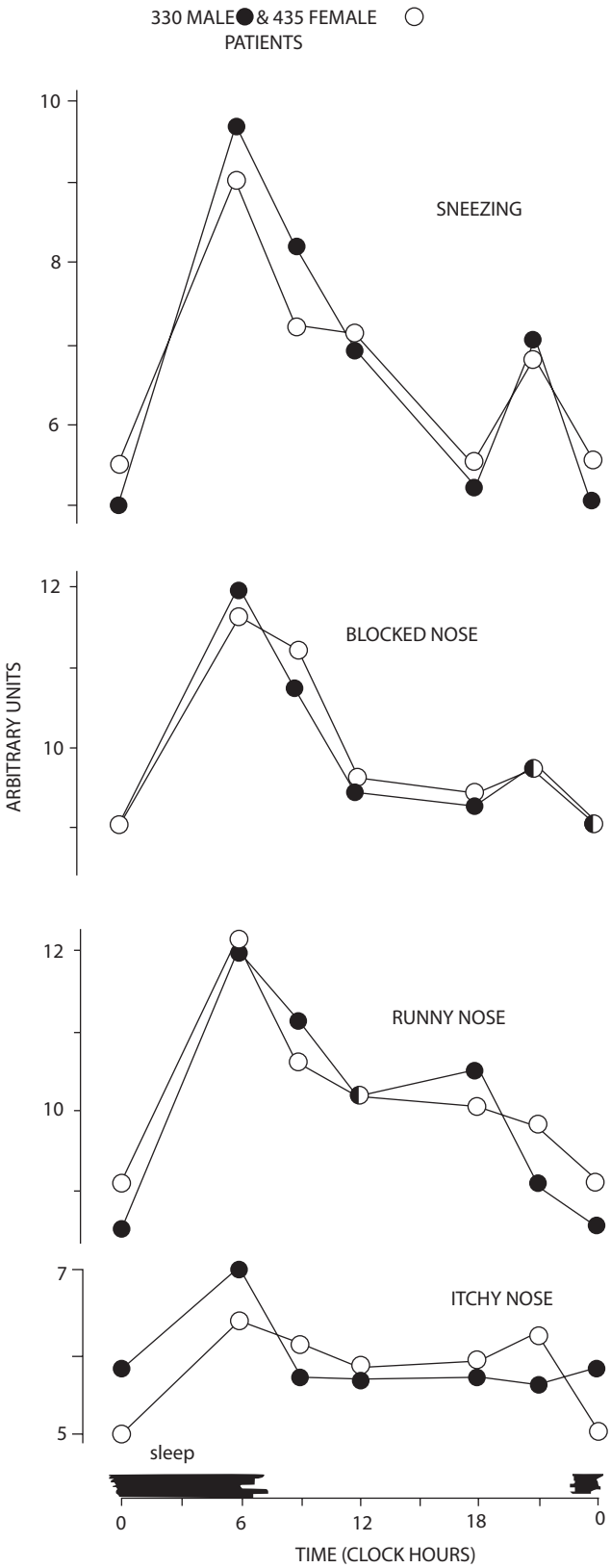

Fig. 16.4 Chronogram demonstrating the variation in nasal symptoms by time of day in a study of 330 male and 435 female patients by Reinberg et al. [80]

The adenoids are a collection of lymphoid tissue located on the posterior wall of the nasopharynx. They are part of the circle of lymphoid tissue known as "Waldeyer's ring," which includes the lingual and palatine tonsils. Adenoids typically grow until approximately age 6 and then begin to regress, often disappearing completely by late adolescence or early adulthood. Adenoid hypertrophy is a common cause of nasal obstruction in children. A meta-analysis excluding studies of children with overt obstructive sleep apnea (OSA) found that the prevalence of adenoid hypertrophy ranges from 42% to 71% [15].

16.3.2 Inflammatory Factors

In addition to anatomic abnormalities, nasal obstruction can be caused or exacerbated by various inflammatory factors. The nasal airway is highly sensitive to inflammation, and variations in the level of inflammation with associated nasal congestion significantly impact nasal airflow. Epidemiologic studies differ in their estimation of the prevalence of allergic rhinitis, but it is estimated to affect between 9% and 42% of the population [16]. Several inflammatory factors associated with allergic rhinitis follow circadian cycles, include interferon gamma, tumor necrosis factor alpha, and IL-4, IL-1b, and IL-10 [17, 18]. Many factors upregulate during sleeping hours, with peaks in the early morning worsening nasal obstruction. Some patients with allergies develop overreliance on topical decongestants such as oxymetazoline, which can lead to a rebound congestion affect known as rhinitis medicamentosa, further worsening nasal breathing [19].

Several nonallergic rhinitis conditions exist. Vasomotor rhinitis is linked to autonomic dysfunction and causes nasal congestion triggered by odors, changes in temperature or pressure, or consumption of certain foods [20]. Pregnancy-induced rhinitis is caused by hormonal effects on the nasal mucosa, affecting 7–9% of pregnant women [21]. Rhinitis is also associated with a variety of other systemic inflammatory conditions, such as lupus, Sjogren's syndrome, Churg Strauss syndrome, and others.

Nasal resistance also increases when moving from upright to supine positioning, common during sleep [22]. This is likely modulated in part by autonomic reflexes triggered by pressure receptors at the body's surface, but also by passive congestion of the nasal vasculature [23]. Patients who already experience congestion during the day are likely to have these effects magnified during sleep by the effects of gravity with increased vascular congestion within the nose.

16.3.3 The Effect of the Nasal Airway on Sleep and Sleep Apnea

There is a well-established relationship between nasal congestion and poor sleep quality. Several large epidemiologic studies have shown that nasal obstruction, either due to anatomic constraints or inflammatory disease, is an independent risk factor for snoring and sleep apnea [24, 25, 26]. The link between these two entities is complex and multifactorial. The nose is the primary breathing pathway under normal conditions. Nasal breathing accounts for 92% of inhaled air while awake and 96% of inhaled air while asleep [27]. In healthy individuals, nasal resistance accounts for 55–60% of total airway resistance [28]. This proportion can be significantly impacted by nasal obstructive issues. Pharyngeal airway collapse has been approximated by many authors using a Starling resistor model. The pharynx is modeled as a homogenous hollow tube suspended between rigid upstream and downstream segments. Nasal airway resistance represents a partial obstruction at the aperture of the tube and the pharynx represents a collapsible downstream segment connected to the rigid trachea. Increasing nasal resistance causes negative intraluminal pressure downstream, increasing the tendency of the pharynx to collapse [29, 30]. If nasal obstruction is severe enough, many individuals convert to oral respiration (i.e., mouth breathing). Experiments have demonstrated that oral breathing is physiologically disadvantageous, with upper airway resistance increasing 2.5-fold as compared to nasal breathing during sleep [31].

Induction of nasal obstruction has been shown to affect sleep quality in several studies. Regli et al. found that postoperative nasal packing increased the apnea-hypopnea index (AHI) of patients with OSA [32]. Other studies have demonstrated that experimentally packing the nares of healthy subjects increased their AHI, arousal index, and decreased total sleep time [33, 34]. However, Friedman et al. showed in 49 subjects with OSA that only those with mild sleep apnea had an increase in AHI with postoperative nasal packing as opposed to those with more severe disease [35]. Conversely, relieving nasal obstruction has been found to improve sleep quality. In a 2019 study, An et al. found that applying oxymetazoline before sleep in patients with OSA and chronic nasal congestion improved AHI, duration of REM sleep, and mean O2 saturation [36].

In addition to its role in physical resistance, studies suggest that sensorineural feedback from the nose affects breathing patterns during sleep. White et al. showed that administration of intranasal lidocaine with a decongestant significantly increased AHI compared to saline and a decongestant (mean AHI: 25.8 vs 6.4, respectively) [37]. Nasal lidocaine has also been found to significantly

Table 16.1 Inflammatory mediators and their effect on sleep

Mediator	Effect on sleep	Effect on nasal airway obstruction
Histamine	Involved in balance between wakefulness and slow-wave activity during sleep through H_1 receptors in the brain	Slight ↑
CysLT	↑ Slow-wave sleep	↑
IL-1	Effects probably mediated in part through ↑ PGD_2; ↑ non-REM sleep; associated with ↓ latency to sleep onset: ↑ latency to REM and ↓ REM duration	
IL-4	Associated with ↓ latency to sleep onset; ↑ latency to REM and ↓ REM duration	
IL-10	Associated with ↓ latency to sleep onset; ↑ latency to REM and ↓ REM duration	
TNF-α	Probably mediated through ↑ PGD_2	
PGD_2	↑ REM, ↑ non-REM	↑
Bradykinin		↑
Substance P	↑ Latency to REM, arousing effect	↑

Adapted from Ferguson et al. (2004)

increase the length of apneic events by decreasing the arousal response to apnea [38]. In normal, awake breathing, the nasal route of breathing vs oral breathing lead to higher minute ventilation and respiratory rate. These differences are largely erased when topical anesthesia is applied to the upper airway, suggesting that nasal airflow receptors help regulate respiratory rate [39]. Thus, the absence of airflow due to nasal congestion may impair breathing through dysregulation of neural feedback mechanisms in addition to simple airway mechanics.

Allergy contributes to obstruction and can affect breathing during sleep through multiple mechanisms. In addition to mechanical obstructive effects, inflammatory mediators associated with allergy are also associated with poor sleep quality (Tables 16.1 and 16.2) [18]. Many medications for the treatment of allergic rhinitis bind central nervous system receptors related to sleep regulation, increasing the potential for sleep-related side effects. Oral antihistamines, the most commonly used medication for treating allergic rhinitis, are highly effective in controlling sneezing and itching symptoms but reduce congestion less effectively, limiting their utility in decreasing nasal resistance during sleep [18]. Side effects of oral antihistamines can include mouth dryness, which can exacerbate nighttime breathing difficulties [40]. H_1-receptor antagonists cause drowsiness in susceptible individuals and may be useful as a sleep aid but can result in undesirable sleep inertia. For this reason, many antihistamines are widely used as sleep aids despite lack of evidence supporting improvements in sleep quality. Conversely, systemic decongestant medications (e.g., pseudoephedrine) and beta agonists may cause or exacerbate insomnia [40].

There is also evidence that continuous positive airway pressure (CPAP) therapy interacts negatively with allergy and sinonasal inflammation. Alahmari et al. found that CPAP usage resulted in an upregulation of sinonasal inflammatory markers and reduced mucociliary clearance [41]. Saka et al. also found that CPAP led to increased inflammation and fibrosis in sinonasal mucosa based on tissue biopsies of 25 subjects [42].

16.4 Evaluation of the Nasal Airway

It is critical to assess history along with physical examination in a patient with symptoms of nasal obstruction and SDB, as perception of nasal obstruction does not always directly correlate with physical exam findings. Many patients experience obstructive symptoms of a waxing and waning nature. If a patient complains of nasal obstruction, historic information and exam findings will inform whether it is secondary to anatomic abnormalities or inflammatory factors and will directly impact management decisions. Additional testing, such as nasal endoscopy, imaging, or rhinomanometry, may provide additional information but are not always necessary.

Allergic rhinitis and other nasal inflammatory disorders often include rhinorrhea in addition to obstructive symptoms. Many patients with allergic rhinitis are able to pinpoint seasonal variations in symptoms or known exposures which exacerbate symptoms. Patients with vasomotor rhinitis may describe atypical exacerbating factors which are not seasonal or associated with traditional environmental triggers. Patients with perennial allergic rhinitis may not be able to describe any triggers at all. A good social history can clue a clinician into occupational exposures such as mold, sawdust, and industrial chemicals. A medication history can gather information regarding previously successful and unsuccessful therapies (and sometimes even the cause of pathology, as in rhinitis medicamentosa). Anatomic abnormalities tend to lead to obstruction that is constant rather than variable, and may be unilateral in nature. A history of trauma or prior surgery can also lead a clinician to more strongly consider anatomic obstruction in their diagnosis. Many patients experience their nasal obstruction only at night when supine, so questions should always include assessment of nighttime symptoms. Validated questionnaires such as the Nasal Obstruction Symptom Evaluation and the

Table 16.2 Useful elements of the history and physical exam to evaluate nasal symptoms and sleep

History	
HPI	- Assess chronicity, seasonality, duration, severity of obstruction - Unilateral vs bilateral - Nighttime vs daytime symptoms - Associated rhinorrhea? - Alleviating or relieving factors? (potential allergens, medication usage)
PMH	- History of facial trauma - History of nasal surgery - Allergy testing
SH	- Smoking - Occupation (exposure to mold, sawdust, industrial chemicals)
Physical Exam	
External nasal examination	- Note position of nasal cartilages and dorsum - Cottle/Modified Cottle for assessment of nasal valves
Anterior rhinoscopy	- Assess septal position - Note character of nasal mucosa

HPI History of Present Illness; *PMH* Past Medical History; *SH* Social History.

Sinonasal Questionnaire can help quantify symptom severity (Figs. 16.5 and 16.6) [43, 44].

Physical examination will include a careful examination of the external nose before assessment of the nasal airway, including the position of the nasal dorsum and nasal cartilages. Anterior rhinoscopy can be performed with a nasal speculum. Assessment of the anterior nasal septum and location of deviation, if present, may be useful in surgical planning. The turbinate mucosa may appear boggy and inflamed in an allergic patient during anterior rhinoscopy. Dynamic maneuvers, such as the modified Cottle maneuver, provide important information regarding nasal valve structural support if suspicion is raised for dynamic valve collapse during quiet respiration. First, the patient is asked to inspire gently and the external nasal wall is observed carefully for dynamic collapse. Then, to perform the modified Cottle maneuver, a small instrument or cotton-tipped applicator is used to support the nasal sidewall medially while the patient repeats the exam. Relief of symptoms suggests that nasal valve collapse may be contributing to their symptoms [45]. The Cottle maneuver is performed similarly, except that instead of using an instrument to support the nasal valve from the inside, a finger is pressed laterally to the alar skin to draw the nasal sidewall open. Significant improvement in inspiration with this maneuver may increase suspicion of static valve collapse.

Nasal endoscopy helps to further characterize anatomy in many cases. A rigid endoscope is passed through the nare after application of topical decongestant and anesthetic. Initial nasal examination should be completed prior to administration of medication, as this may mask inflammatory conditions such as inferior turbinate hypertrophy. Nasal endoscopy allows for evaluation of more posterior structures, such as the middle turbinates, the posterior aspect of the inferior trubinates, and the posterior septum. It may help identify sinonasal polyps, adenoid hypertrophy, or other obstructing masses not easily visualized on anterior rhinoscopy.

Sinus computed tomography imaging helps to objectively characterize the bony anatomy of the nose and paranasal sinuses. Soft tissue lateral neck roentgenograms may be useful for assessing adenoid hypertrophy in children intolerant of fiber optic endoscopy.

Acoustic rhinometry is a diagnostic tool that measures reflected soundwaves to generate information about the internal geometry of the nose [46]. It has the advantage of being relatively noninvasive and requires minimal participation from subjects such as children who may not cooperate with endoscopy or imaging.

Fig. 16.5 The Sinonasal Questionnaire

Over the last 3 months how often, on average, did you have the following symptoms?

	Never	1 - 4 times per month	2 - 6 times per month	Daily
Runny Nose	☐	☐	☐	☐
Post nasal drip	☐	☐	☐	☐
Need to blow your nose	☐	☐	☐	☐
Facial pain/pressure	☐	☐	☐	☐
Nasal obstruction	☐	☐	☐	☐

Scoring: Never (0), 1 -4 times per month (1), 2- 6 times per week (2), and daily (3).

Score reported as average of items: range of possible scores 0 - 3.

Fig. 16.6 The NOSE survey

Over the past 1 month, how much of a problem were the following conditions for you?

Please circle the most correct response

	Not a problem	*very mild problem*	*moderate problem*	*fairly bad problem*	*severe problem*
1. Nasal congestion or stuffiness	0	1	2	3	4
2. Nasal blockage or obstruction	0	1	2	3	4
3. Trouble breathing through my nose	0	1	2	3	4
4. Trouble sleeping	0	1	2	3	4
5. Unable to get enough air through my nose during exercise or exertion	0	1	2	3	4

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Rhinomanometry is a technique which uses pressure transducers and a facemask to objectively measure nasal airflow [47]. Both techniques require equipment beyond that routinely used in clinical practice, but they may be useful adjuncts to the evaluation of nasal obstruction in select patients.

16.4.1 Medical Treatment for Nasal Obstruction

Many allergic patients can reduce nasal congestion symptoms with stimulus avoidance strategies. For those who cannot, there are many oral and topical therapies available for the treatment of allergic rhinitis. Intranasal corticosteroids are the current gold standard of medical therapy. In a 2016 meta-analysis, Liu et al. analyzed five double-blind randomized controlled trials that compared intranasal steroids (either fluticasone or mometasone) against a saline spray placebo in patients with OSA with AHI as the primary outcome. Results showed a statistically significant but clinically minimal change in AHI. The mean difference in AHI was −0.95 (95% CI −1.42 to −0.47) based on pooled results from 221 patients (Fig. 16.7) [48]. In a 2013 prospective cohort study, Lavigne et al. reported that supine AHI and baseline blood oxygen saturation improved in 34 patients with OSA and allergic rhinitis treated with intranasal steroids, compared with no change in a cohort with OSA but no allergic rhinitis [49]. While the objective data suggest that nasal corticosteroid treatment has minimal impact, several studies suggest that the impact on subjective sleep quality may be greater. In 2010, Meltzer et al. published a double-blind parallel study comparing intranasal corticosteroid with saline spray and found improvements in sleep quality using the

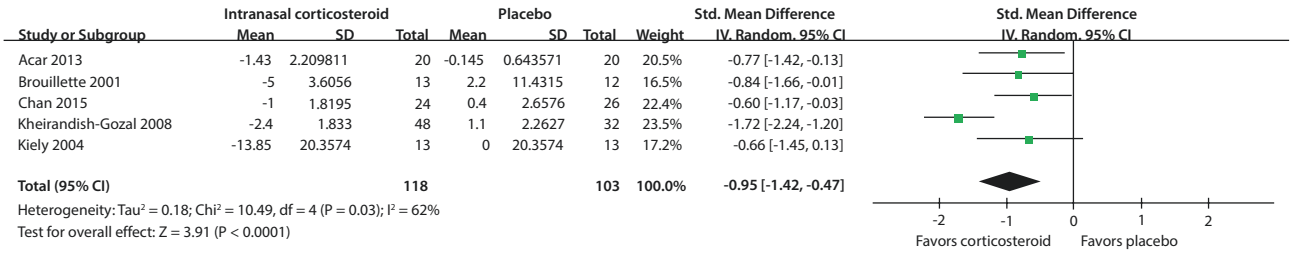

Fig. 16.7 Meta-analysis of the effect of intranasal corticosteroids vs placebo on apnea-hypopnea index from Liu et al. [48]

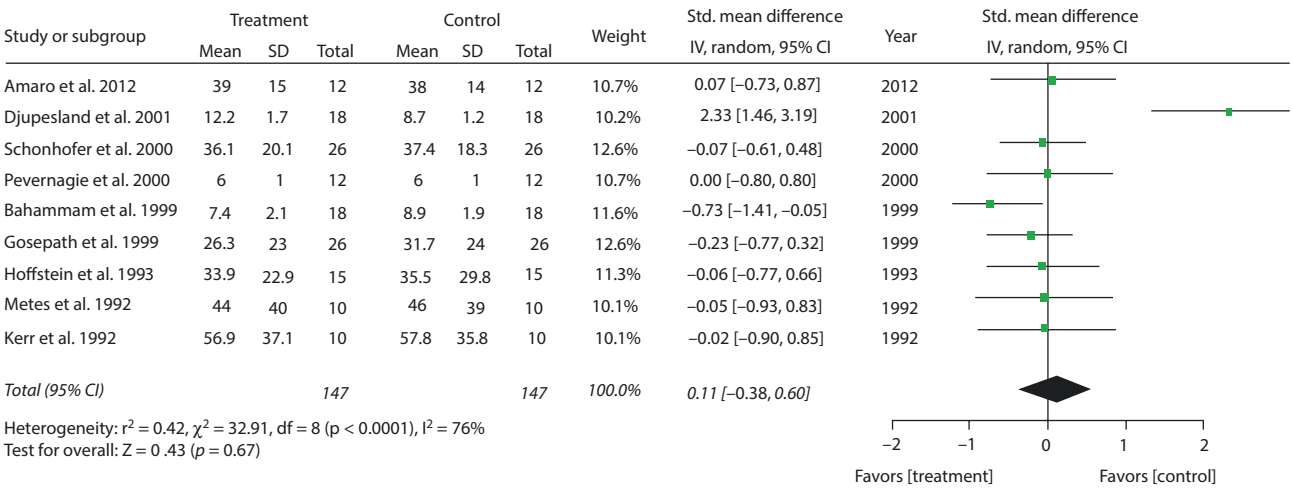

Fig. 16.8 Forrest plot showing the effect of nasal dilators on apnea-hypopnea index from a meta-analysis by Camacho et al. [54]

Rhinoconjunctivitis Quality of Life Questionnaire (RQLQ) (−1.82 vs −0.6), the Epworth Sleepiness Scale (ESS) (−1.9 vs +0.44), and reported fewer hours missed and less daily activity impairment on the Work-Productivity and Activities Impairment-Allergy Specific score (WPAI-AS). This study also measured AHI and did not note a statistically significant difference in the intervention group, although no subjects with an AHI over 20 were included [50]. In 2005, Craig et al. published pooled data from three double-blinded placebo-controlled crossover studies on patients with allergic rhinitis and associated sleep symptoms. Of note, all three studies excluded subjects with OSA [51].

Topical nasal antihistamines are often prescribed as an alternative or adjunct to nasal corticosteroids in the treatment of allergic rhinitis. In a double-blinded crossover study, topical azelastine was compared to saline in subjects with allergic rhinitis and SDB. Subjects in the azelastine group reported subjectively improved sleep, but no improvements in daytime somnolence or congestion [52]. In another trial, Santos et al. found that the use of the leukotriene inhibitor montelukast led to subjective improvement in sleep quality as measured by several validated scales including the Functional Outcomes of Sleep Questionnaire (FOSQ), the ESS, the Calgary Sleep Apnea Quality of Life Index in subjects with perennial allergic rhinitis [53].

Available clinical data support medical treatment in patients with SDB and allergic rhinitis. In particular, the use of nasal corticosteroids significantly improves subjective sleep symptoms, and may also have a modest effect on AHI and objective measures of sleep quality. Other medical allergy treatment may also be beneficial, but further research is needed to define the role of these medications in managing sleep symptoms.

Nonsurgical treatment of nasal obstruction in SDB is possible with the use of nasal dilators. Nasal dilators are noninvasive, externally applied devices meant to stent open the nasal valves. While they are occasionally cumbersome to wear during wakeful activities, they can be more easily tolerated by many sleeping patients. In a 2016 meta-analysis, Camacho et al. examined 147 subjects from 14 studies using either internal or external nasal dilators and reported that there was no significant difference in AHI, lowest O_2 saturation, or snoring index. There was a small decrease in AI (apnea index) with the use of internal dilators (4.87 events/hr. vs 0.64 events/hr.) (Fig. 16.8) [54]. In a 2018 randomized controlled trial using two types of external dilators, both types were found to be superior to a sham dilator in

improving sleep quality based on the Pittsburgh Insomnia Rating Scale (PIRS), Nocturnal Rhino conjunctivitis Quality of Life Questionnaire (NRQLQ) [55].

16.4.2 Nasal Surgery in the Management of Sleep-Disordered Breathing

Medical management of inflammatory disorders is inexpensive, has a favorable risk profile, and may obviate the need for surgical correction of observed structural abnormalities if patient symptoms sufficiently improve. However, in many cases, correction of anatomic causes of restricted nasal airflow requires surgical intervention. The surgical management of nasal obstruction is complex, and interventions must be tailored to the individual patient's anatomy. The evidence regarding nasal surgery and SDB comes primarily from retrospective observational cohorts due to the ethical concerns present in randomizing patients to alternative or sham surgical comparators.

16.5 Surgical Techniques to Address Nasal Obstruction

The specific techniques of nasal surgery to address obstruction are varied, nuanced, and beyond the scope of this chapter. However, it is crucial to have a basic understanding of available interventions and procedural selection. Septoplasty with or without treatment of inferior turbinate hypertrophy is the most commonly performed surgery for nasal obstruction and is perhaps the best studied with regard to SDB. In general, septoplasty involves elevation of the septal mucosa from the cartilaginous and bony septum, with removal or repositioning of the deviated portions. A dorsal and caudal strut of cartilage are preserved or constructed to ensure structural integrity of the nose, avoiding any significant change in external appearance. Turbinate reduction is a technique that involves reducing the size of the soft tissue covering the turbinates with a microdebrider or radiofrequency ablation and is often combined with surgical out fracture of the turbinate bones. This can be achieved in the operating room or in the office setting under local anesthesia in appropriately selected patients.

In patients with external nasal deformities, functional rhinoplasty with or without septoplasty may be performed. Functional rhinoplasty encompasses a variety of techniques with the goal of surgical modification of the external cartilages and bones of the nose, often with the intention of augmenting the cross-sectional area or rigidity of the internal and external nasal valve.

Adenoidectomy is a commonly performed procedure that involves surgically removing obstructive adenoid tissue using various techniques, including electrocautery, micro debridement, or cold steel. Functional endoscopic sinus surgery (FESS) involves using an endoscopic camera and instruments to correct bony anatomy in the sinus cavities in such a way as to optimize sinonasal drainage and remove inflammatory disease that is refractory to medical management. Additional structural anomalies directly obstructing nasal airflow, such as concha bullosa or nasal polyposis, can also be corrected during FESS. Any of these procedures can be performed alone, but are often performed in various combinations for patients with multifactorial anatomic obstruction.

16.6 Evidence for Nasal Surgery in Obstructive Sleep Apnea

Available data suggest that nasal surgeries often improve subjective sleep quality but do not reliably improve objective polysomnographic metrics of SDB. However, correction of nasal obstruction may significantly improve the ability of patients to tolerate CPAP, the first-line treatment for OSA. A large meta-analysis regarding the effect of nasal surgery on AHI and subjective sleep quality analyzed 225 subjects across 10 retrospective cohort studies. Surgical interventions were diverse and included turbinate reduction alone, septoplasty combined with turbinate reduction, and "any combination of septoplasty, turbinate reduction, nasal valve reconstruction, and endoscopic sinus surgery." No statistically significant difference in AHI was observed, but statistically significant improvements in the respiratory disturbance index and subjective sleepiness symptoms were observed (RDI: −11.06 events/hr., 95% CI [−5.92, −16.19]; ESS -3.53, 95% CI [−0.64, −6.23]), (◘ Fig. 16.9) [56]. A previous meta-analysis by Li and colleagues reported similar findings [57]. Other observational studies further support the impact of nasal surgery on subjective sleep quality. Li et al. found that disease-specific quality of life was significantly improved in patients undergoing septoplasty based on the Snore Outcomes Survey and the ESS, as did Ertugay et al. in a similar study [58, 59].

More recent data suggest that there may be subgroups of patients that achieve significant improvements in objective polysomnography metrics with nasal surgery. Shuaib et al. reported a statistically significant decrease in AHI of 35% after functional rhinoplasty (24.7 to 16), which increased to 57% when patients with a BMI over 30 were excluded (22.5 to 9.6) [60]. In 2015, Hisamatst et al. reported a significant reduction in AHI after patients with OSA and nasal obstruction underwent combination septoplasty and posterior neurectomy, a procedure to reduce nasal inflammation by resecting the vidian nerve (−12.46 in severe OSA and − 7.86 in moderate OSA) [61]. In a Norwegian

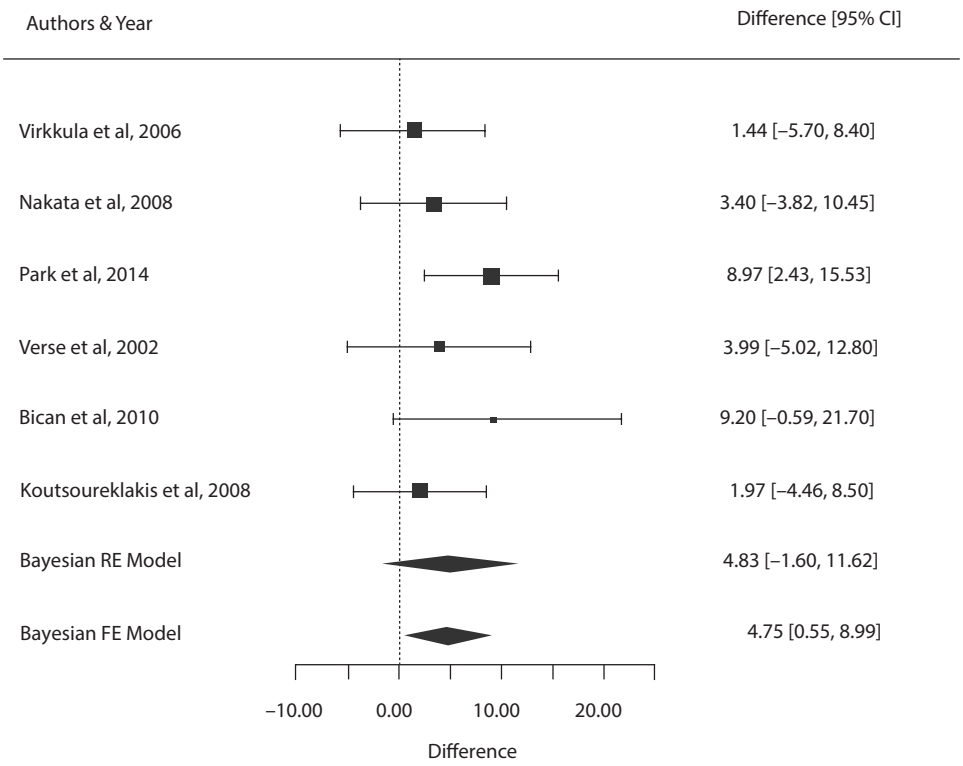

Fig. 16.9 Forrest plot showing pre- and post-operative apnea-hypopnea index after nasal surgery from a meta-analysis by Ishii et al. [56]

cohort study of 78 patients, Moxness et al. found a highly statistically significant improvement in ESS after nasal surgery (8.94 vs 10.74; $p < 0.01$). This study did not find a statistically significant change in AHI with nasal surgery overall, but in post-hoc subgroup analysis found a statistically significant decrease in AHI in patients undergoing combination septoplasty and turbinate reduction (17.4 to 11.7; $p < 0.01$) [62]. Recent studies in South Korea and China have also showed statistically significant decreases in AHI after nasal surgery [63, 64].

The effect of nasal surgery on AHI is varied and inconclusive. There may be select groups of patients that experience significant improvements in objective measures of OSA disease burden, but these subgroups have yet to be clearly identified. However, available evidence does suggest that nasal surgery may improve subjective sleep quality and daytime sleepiness.

There is also convincing evidence that nasal surgery may help reduce CPAP pressure requirements and improve CPAP compliance. CPAP is the first-line treatment for OSA, but adherence rates pose a significant problem with estimates of nonadherence ranging from 46% to 83% [65]. A meta-analysis by Camacho et al. in 2015 examined 279 patients across 18 studies reporting CPAP data before and after isolated nasal surgery. Nasal surgery resulted in a statistically significant decrease in mean therapeutic pressures from 11.6 ± 2.2 to 9.5 ± 2.0 cm H_2O. Following nasal surgery, CPAP compliance improved from 38.7% to 89.1% postoperatively [66]. A cost–benefit analysis of nasal surgery to improve CPAP compliance concluded it is a cost-effective strategy when weighed against the cost of untreated sleep apnea (Table 16.3.) [67].

16.7 Evidence for Nasal Surgery in Primary Snoring

There has also been extensive investigation into the effect of nasal surgery on primary snoring. Several studies assessing snoring objectively through audio recordings have not shown a significant decrease in the duration or intensity of snoring [68, 69]. An exception is Choi et al., who found a small but statistically significant reduction in snoring duration after nasal surgery of any kind including FESS, septoplasty, and/or turbinate reduction surgery (32.2 ± 16.4% to 25.8 ± 18.6% of the night spent snoring) [70]. Most studies on snoring after nasal surgery report subjective assessments from patients or sleep partners (Table 16.4) [71, 72, 73, 58, 74].

In contrast to adults, adenotonsillectomy is considered a first-line treatment for OSA in children. Isolated adenoidectomy is often performed when nasal obstruction is a primary complaint and evidence suggests it may be effective in improving AHI in OSA [75, 76]. Domany et al. found that adenoidectomy alone produced similar long-term subjective symptomatic improvement measured with the Pediatric Sleep Questionnaire (PSQ) as compared to adenotonsillectomy in nonobese children under the age of 7 [77].

Table 16.3. Theorized cost savings per quality of life year of performing septoplasty or turbinate reduction on patients with OSA based on a model developed by Kempfle et al. [67]. Model results are reported with assumed cost of untreated OSA (Cost OSA ($)) values ranging from 500 to 5000

Table I Cost QALY Saved With Nasal Surgen							
Septoplasty				**Turbinate Reduction**			
5 years		Cost ($) QALY		5 years		Cost (S) QALY	
	Cost OSA (S)	pCPAP 70%	pCPAP 30%		Cost OSA ($)	pCPAP 70%	pCPAP 30%
	500	1,823.53	618.81[a]		500	668.07	787.13
	1,000	1,907.56	1,051.98[a]		1,000	752.10	1,183.17
	2,000	2,075.63	1,918.32[a]		2,000	920.17	1,975.25
	5,000	2,579.83	4,517.33[a]		5,000	1,424.37	4,351.49
10 years		Cost ($) QALY		10 years		Cost (S) QALY	
	Cost OSA (S)	pCPAP 70%	pCPAP 30%		Cost OSA (S)	pCPAP 70%	pCPAP 30%
	500	1,174.37	1,383.66		500	596.64	702.97
	1,000	1,258.40	1,779.70		1,000	680.67	1,099.01
	2,000	1,426.47	2,571.78		2,000	848.74	1,891.09
	5,000	1,930.67	4,948.02		5,000	1,352.94	4,267.33
15 years		Cost (S) QALY		15 years		Cost (S) QALY	
	Cost OSA (S)	pCPAP 70%	pCPAP 30%		Cost OSA (S)	pCPAP 70%	pCPAP 30%
	500	957.98	1,128.71		500	572.83	674.92
	1,000	1,014.01	1,524.75		1,000	656.86	1,070.96
	2,000	1,210.08	2,316.83		2,000	824.93	1,863.04
	5,000	1,714.29	4,693.07		5,000	1,329.13	4,239.27

Calculations were made for two base populations with different pCPAP, 30% or 70%, and for time frames 5, 10, and 15 years

[a]Only septoplasty within the 5-year time frame is not cost-effective; at all other times, nasal surgery is favored (WTP = $50,000)

OSA obstructive sleep apnea, *pCPAP* probability of continuous positive airway pressure compliance, *QALY* quality-adjusted life year, *WTP* willingness to pay

Table 16.4 Subjective snoring outcomes after nasal surgery

Study Authors	Intervention	Patient population	Subjective outcomes
Sufioğlu et al. [71]	"nasal surgery" not specified	31 patients with OSAS and confirmed nasal pathology on PE	Snoring on VAS 4.9 ± 2.3 from 8.6 ± 2.3 $p < 0.001$
Friedman et al. (2007)	Septoplasty ± BITSMR ± FESS	50 patients with OSAS undergoing nasal surgery	17/50 patients noted improvement or resolution of snoring
Cigdem et al. (2015)	Septoplasty	64 patients with self-reported snoring and septal deviation	Snore symptom inventory mean 25.61 from 58.14 $p < 0.001$
Li et al. (2009)	Septoplasty	52 patients with OSAS and septal deviation	Snore outcome survey 41.5 ± 9.7 from 60.7 ± 14.4 $p < 0.001$ Spouse/Bed Partner Survey 39.7 ± 18.7 from 60.3 ± 21.5 p <0.001

Professional societies inconsistently recognize the utility and importance of nasal surgery in the management of SDB. The American Academy of Sleep Medicine produced a clinical practice guideline for the surgical management of sleep apnea in 2010, which did not specifically evaluate nasal surgery outcomes [78]. In contrast, an American Academy of Otolaryngology—Head and Neck Surgery position statement emphasizes the importance of nasal surgery for improvement in sleep quality and improvement of PSG metrics in a subset of patients [79].

16.8 Summary

Nasal obstruction and SDB are intimately linked. The relationship between the nose and sleep is complex and is affected by airway mechanics, sensorineural feedback loops, medical therapies, and inflammatory factors. Nasal obstruction is often multifactorial and can consist of inflammatory factors such as allergy, anatomic limitations, or a combination of both. Any patient who presents with sleep complaints should be assessed for nasal obstruction with a directed history and a physical examination that includes anterior rhinoscopy, with additional diagnostic evaluation modalities applied as indicated.

In symptomatic patients, there is significant evidence that medical treatment for allergy, especially with intranasal corticosteroids, can improve subjective sleep quality and may even modestly improve AHI. In some patients, especially those with obstructive symptoms refractory to medical treatment, nasal surgery should be considered. Evidence suggests that nasal surgery may not reliably improve AHI in patients with OSA, but it often improves daytime somnolence, subjective sleep quality, and CPAP tolerance and therapeutic pressures. Further research is needed to determine which surgical interventions are most effective in treating SDB and which patients stand to benefit the most from nasal surgery. Management of nasal obstruction in patients with SDB is an essential component of a comprehensive treatment strategy.

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Palatal Surgery for OSA Patients

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Contents

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17.1 Introduction

Obstructive sleep apnea (OSA) is a common form of sleep-disordered breathing. OSA usually results from the combination of a narrowed upper airway and loss of muscle tone during sleep, which leads to partial or complete cessation of airflow. The upper airway includes the nasal cavity, nasopharynx, oropharynx, hypopharynx, and supraglottis, ◘ Fig. 17.1. The oropharynx (retropalatal area) is considered the most common site of obstruction in patients with OSA [1]. Continuous positive airway pressure (CPAP) increases the intraluminal pressure to maintain the patency of the upper airway and overcome the tendency of airway collapse in individuals with OSA. Current clinical practice guidelines recommend positive airway pressure therapy for treatment of OSA in adult patients with impaired sleep-related quality of life, excessive daytime sleepiness, or other associated comorbidities [2]. Although PAP is the primary treatment, many patients experience tolerating PAP limiting the maximum benefits from therapy [2, 3]. Because of this, patients may seek alternative treatment modalities including surgical interventions to address their upper airway obstruction.

Surgery can be a primary treatment option in a select group of patients who have identifiable anatomical problems (e.g., maxillomandibular abnormalities, tonsillar hypertrophy), but is more commonly utilized as a "salvage" treatment option for CPAP-intolerant patients. Despite a variable cure rate, surgery has been shown to routinely decrease OSA severity and increase quality of life outcomes [4]. For an effective surgical outcome, determining the site or sites of upper airway obstruction is critical in selecting which surgical procedures may be most appropriate for each patient.

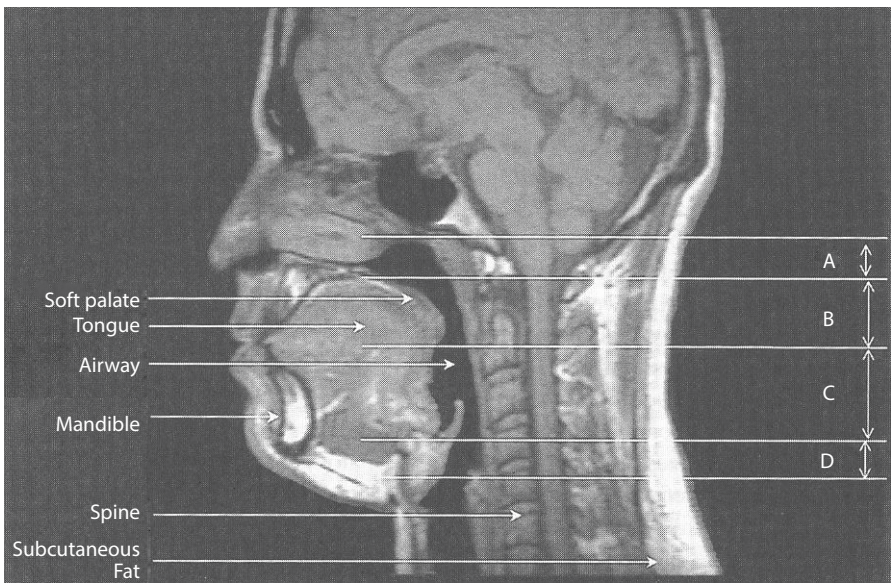
17.2 Presurgical Evaluation and Airway Assessment to Identify Site(s) of Collapse

Presurgical evaluation performed by a sleep medicine physician should include a comprehensive sleep evaluation assessing for comorbid sleep disorders, structured examination of the upper airway, and a diagnostic sleep study. Overnight polysomnography (PSG) is the gold standard for evaluation of OSA and is useful for determining the severity of sleep-disordered breathing as well as identifying other comorbid conditions such as periodic limb movements, hypoventilation, and nocturnal hypoxemia.

17.2.1 Friedman Tongue Position and Tonsillar Size

As part of a structured oral examination, Friedman tongue position and tonsillar size grading can be used, ◘ Fig. 17.2a, b [5]. The Friedman tongue position differs from the Mallampati classification, as the tongue is observed in a natural, neutral position (i.e., no protrusion). Also, the Friedman position has been correlated with surgical results for OSA, unlike the Mallampati class, which has been correlated with the likelihood of difficult intubation.

The Friedman clinical staging system is based on clinical exam findings including the Friedman tongue position, tonsil size, and BMI and is divided into four categories [6]. This staging system can help guide surgical interventions and predict the surgical success of uvulopalatopharyngoplasty (UPPP). Nevertheless, clinical examination is limited by the inability to directly



◘ **Fig. 17.1** Upper airway anatomy. A – nasopharynx; B – oropharynx; C – hypopharynx; D – supraglottis

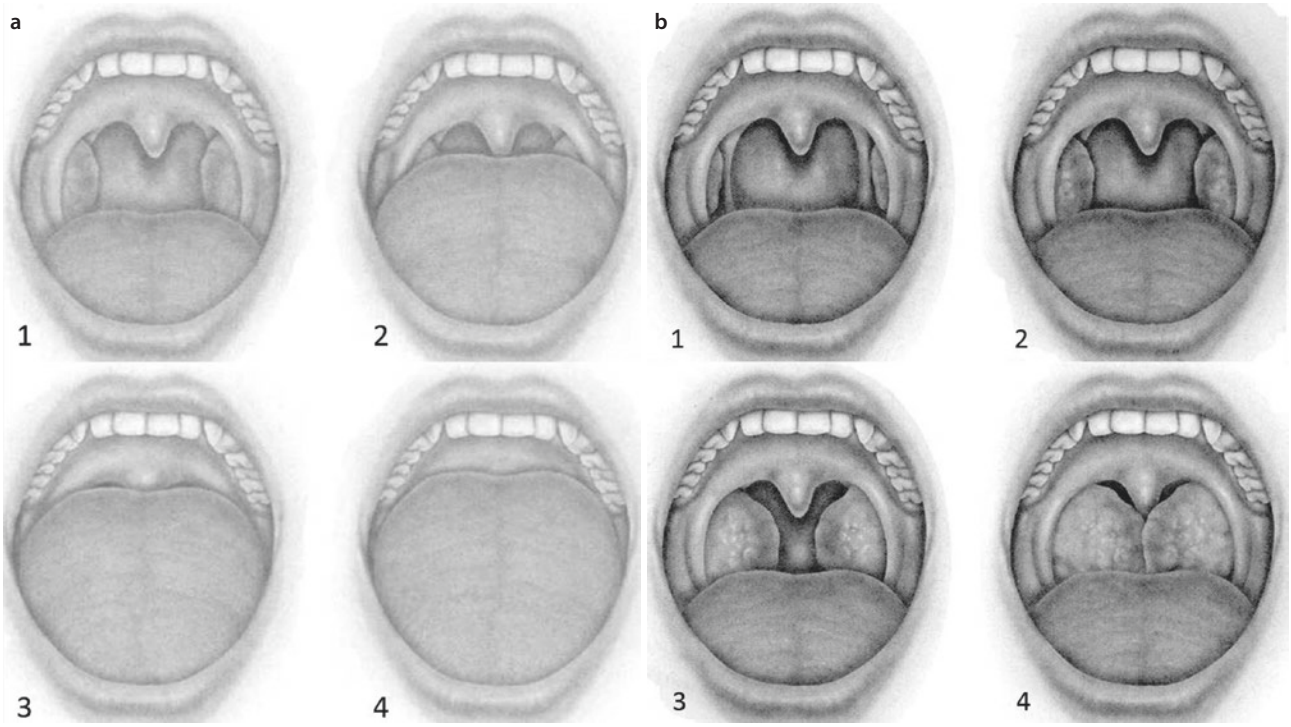


Fig. 17.2 **a** Friedman tongue/palate position grading. Grade 1: Entire uvula + tonsils/pillars visible. Grade 2: Base of uvula but tonsils/pillars not visible. Grade 3: Only soft and hard palate visible. Grade 4: Only hard palate visible. **b** Friedman Tonsil Size grading [10]. Grade 1: Tonsils hidden within pillars. Grade 2: Tonsils extend to the pillars. Grade 3; Extend past the pillars (3/4th way to midline). Grade 4: Extend to the midline ("kissing tonsils")

visualize the nasopharynx and hypopharynx to localize all potential sites of upper airway obstruction. Further diagnostic evaluation with nasopharyngeal endoscopy and cephalometric analysis can aid in further assessment of the upper airway.

17.2.2 Nasopharyngeal Endoscopy

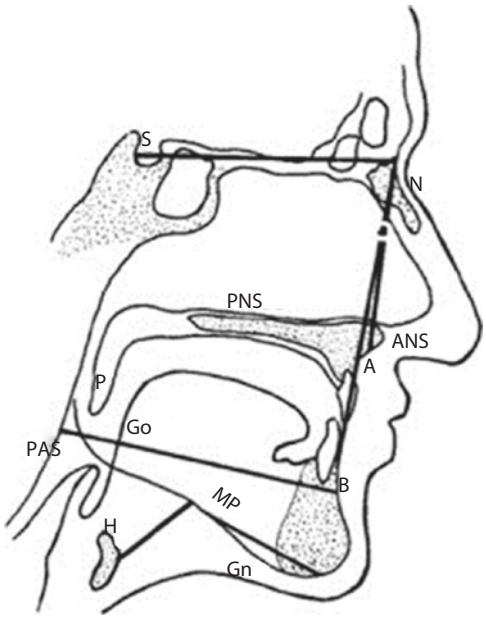
Flexible nasopharyngeal endoscopy aids in identifying potential sites of collapse of the upper airway and can be performed while awake or in a drug-induced sleep-like state. Endoscopic examination allows visualizing the entire nasal cavity and nasopharynx (assessing septal deviation, turbinate hypertrophy, nasal polyps, etc.), hypopharynx (assessing for the lateral pharyngeal walls, base of the tongue, and lingual tonsils), along with assessment of the supraglottis for potential epiglottic collapse.

During awake endoscopy, Mueller's maneuver is performed with a closed mouth and an obstructed nose during maximal inspiration. This maneuver increases the intraluminal negative pressure to mimic sleep-related dynamic upper airway collapse. Drug-induced sleep endoscopy (DISE) is performed under mild sedation (using propofol or midazolam), and has the ability to mimic the dynamics of upper airway collapse during a sleep-like state. DISE has been shown to be a valid assessment of the upper airway, with moderate to substantial test–retest reliability and moderate-to-substantial inter-rater reliability [6, 7]. Classification for obstruction during DISE is defined with regard to the degree of obstruction and pattern of collapse (i.e., anterior–posterior, lateral, and concentric) [6].

A recent meta-analysis of obstructive sites in OSA patients based on a DISE examination reported that patients were likely to have multilevel obstruction rather than single-site obstruction. The majority of patients were obstructed at the soft palate (84%), followed by the base of the tongue (52%) [7]. These results emphasize the importance of performing nasopharyngeal endoscopy for evaluation of patients with OSA due to the high likelihood of multilevel upper airway obstruction and the inability to appropriately assess the entire length of the upper airway on clinical exam alone.

17.2.3 Cephalometrics

Cephalometric analysis using plain x-rays or Cone Beam Computed Tomography (CBCT) is an additional tool to assess upper airway dimensions and aid in the surgical evaluation of OSA patients. CBCT data include a variety of objective airway measurements that help identify potential sites of obstruction in patients with OSA, Fig. 17.3.



Cephalometric Measures	Normal values
SNA	82° ± 3°
SNB	79° ± 3°
H-MP	15 mm ± 3 mm
PAS	11 mm ± 1 mm
PNS-P	37 mm ± 3 mm

Fig. 17.3 Cephalometric tracing and analysis with normal cephalometric values. SNA sella-nasion-infraspinale, SNB sella-nasion-supramentale, H-MP hyoid-mandibular plane, PAS posterior airway space, PNS-P length of soft palate

Studies have shown that a posterior airway space (PAS) <8 mm is predictive of tongue base collapse [8]. A recent study evaluating the correlation between Friedman Tongue Position (FTP) and airway cephalometric measures in OSA patients identified that the posterior airway space (PAS) and minimal retroglossal cross-sectional area had an inverse relationship with FTP [9].

17.3 Surgical Management of OSA

Current guidelines recommend that patients who are deemed to be surgical candidates should be counseled regarding success rates and complications of appropriate surgical techniques [10]. Based on localization of upper airway obstruction, a variety of surgical procedures have been described, primarily focusing on the palate in order to increase the physical size of the upper airway.

Surgeries for OSA can be broadly divided into two categories:
1. Single level – usually Palate.
2. Multilevel – usually Palate + Hypopharynx.

In this chapter, we will primarily focus on single-level surgeries on the palate.

17.3.1 Uvulopalatopharyngoplasty (UPPP) ± Tonsillectomy

Although a multitude of surgical treatment modalities exist, the most commonly performed technique remains the uvulopalatopharyngoplasty (UPPP). In theory, UPPP provides improved patency of the airway by addressing the obstruction at the retropalatal area.

Surgical Technique

Performed under general anesthesia, this procedure involves resection of the redundant soft palatal tissue and anterior tonsillar pillar, back cuts in the posterior tonsillar pillars, and re-approximation of the mucosal edges. If present, tonsillectomy is performed at the same surgical setting (Fig. 17.4).

17.3.1.1 Success Rate of UPPP

Historically, the definition of "success" following surgical intervention for OSA in the ENT literature is a reduction in apnea hypopnea index (AHI) of >50% and an AHI of <20 (the previous definition of mild OSA prior to the introduction of the new American Academy of Sleep Medicine severity scale adopted in 1999). The criteria for cure is defined as an AHI <5 following treatment [9]. In order to avoid controversy regarding definitions, a substantial "improvement" in the underlying OSA severity is probably a better description than true "success" for postoperative results.

Reported "success" rates of UPPP vary considerably. The overall "success" rate for UPPP in unselected patients is approximately 40% [10] with an overall reduction in AHI of 33% based on meta-analysis data. A retrospective analysis using a Friedman clinical staging system for patients with OSA appears to be a valuable predictor of UPPP success. Utilizing this staging system, the UPPP success rate was 80% for stage I patients, 37% for stage II patients, and 8% for stage III patients, Table 17.1 [11]. Another study selected OSA patients with Friedman stage I and II and combined findings of awake nasopharyngeal endoscopy with Mueller's maneuver in patient selection for UPPP. This subset of highly selected patients who were found to have retropalatal obstruction only had a success rate of 95% [12]. These reports of improved surgical success

with UPPP highlight the importance of accurately identifying the site or sites of obstruction in patient selection.

17.3.1.2 Limitations of UPPP

There are several limitations of UPPP surgery:

1. The principal improvement is an increase in the anteroposterior retropalatal airway.
2. UPPP does NOT improve lateral dimensions of the upper airway.
3. UPPP does NOT address potential retroglossal collapse.
4. UPPP does NOT address the decrease in the upper airway dilator muscle tone observed during sleep.

Table 17.1 Friedman clinical staging system for sleep-disordered breathing and UPPP "success" rates

	Stage I	Stage II		Stage III	
Friedman palate position	1–2	1–2	3–4	3–4	Any
Tonsil size	3–4	0–2	3–4	0–2	Any
BMI	<40	<40	<40	<40	>40
UPPP success rate[a]	80%	37%		8%	

[a]AHI reduction 50% and AHI <20

17.3.1.3 Impact of UPPP

- **Quality of Life Indices:**

Isolated UPPP has been shown to improve subjective outcomes such as excessive daytime sleepiness (assessed by Epworth sleepiness scale) and disease-specific quality of life measures (assessed by the validated Functional Outcomes of Sleep Questionnaire). The Epworth Sleepiness Scale has been shown to normalize in 75% of patients at 6 months following UPPP and the Functional Outcomes Sleep Questionnaire (FOSQ) normalized at 3 months postoperatively [13, 14].

- **Biomarkers:**

A significant reduction in serum levels of high-sensitivity C-reactive protein have been observed 6 months following UPPP in OSA patients without a pre-existing diagnosis of cardiovascular disease [15].

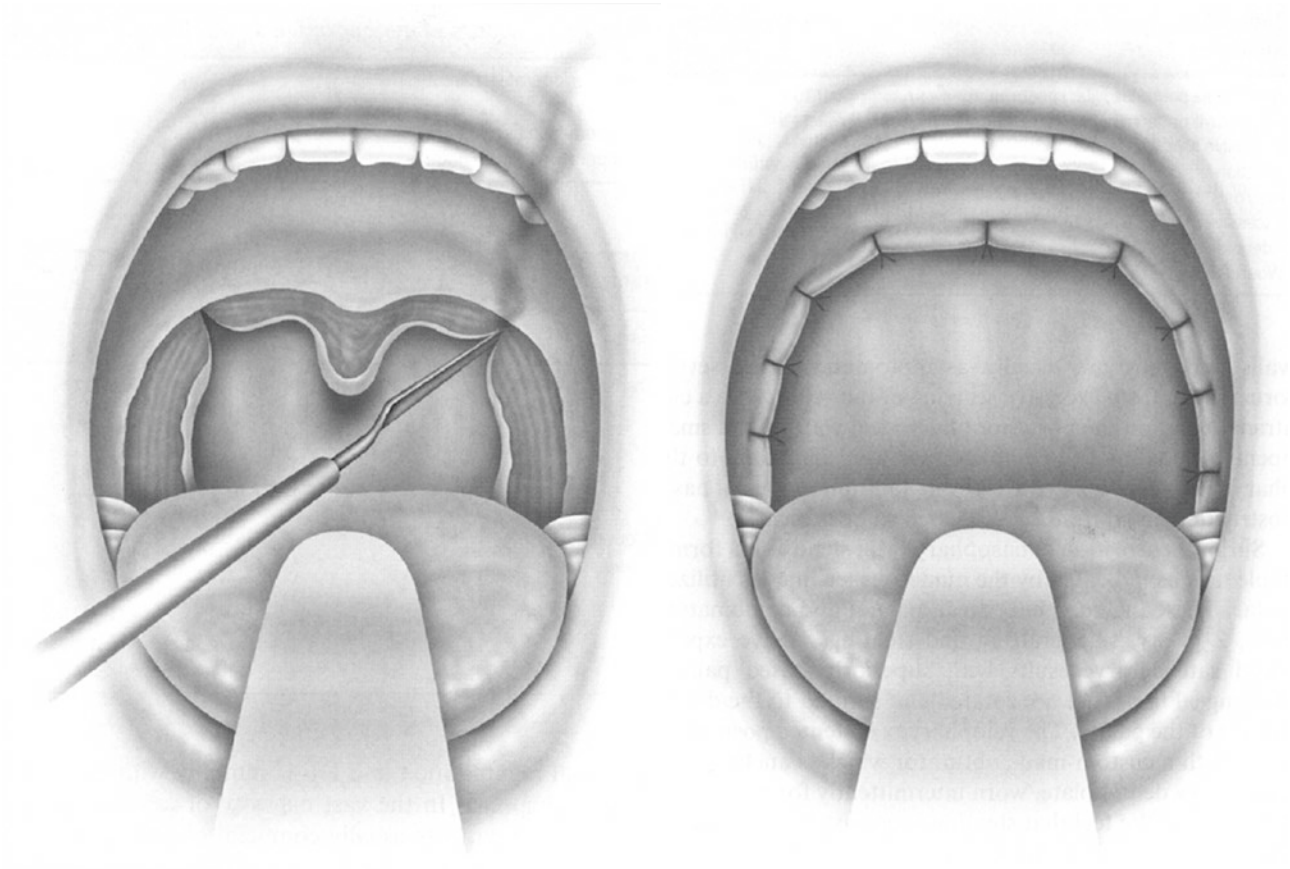


Fig. 17.4 Uvulopalatopharyngoplasty

- **Survival:**

A retrospective cohort study among veterans suggests that UPPP provides a 30% greater long-term survival than CPAP, after adjusting for age, gender, race, date of treatment, and comorbidity [16]. There was also a significant decrease in reported habitual sleepiness while driving and decreased risk of motor vehicle accidents following UPPP [17].

17.3.1.4 Complications of UPPP

The anatomic and physiologic abnormalities associated with OSA increase the risk of perioperative complication in OSA patients undergoing surgery.

- **Early Complications**

In a large cohort of UPPP patients, the reported incidences of serious nonfatal complications and 30-day mortality following UPPP are 1.5% and 0.2%, respectively [18]. Postoperative edema and respiratory depression increase the risk of reintubation or emergent tracheotomy within the first few hours following surgery. Most patients are able to tolerate liquids on the first postoperative day, although with significant pain. As with any procedure inclusive of tonsillectomy, the risk of postoperative bleeding requiring additional surgical intervention is 1–4%.

- **Late Complications**

Velopharyngeal insufficiency is a rare, but serious complication of UPPP, occurring in <1% of patients. This complication can usually be prevented by avoidance of aggressive resection of the underlying soft palatal musculature. A globus sensation in the oropharyngeal area is reported by nearly half of the patients following UPPP and is frequently described as a foreign body sensation or as a sense of excessive mucous accumulation at the free edge of the soft palate. Nasopharyngeal stenosis is an extremely rare late complication following UPPP, but can result in worsening of nasal airflow in patients who develop excessive scarring following surgery.

17

17.3.2 Modifications of UP3

Several modifications of UPPP have been introduced with the intent of improving success rates and reducing the rate of postoperative complications. The most common of these surgeries, including uvulopalatoflap, z-palatoplasty, and expansion sphincter pharyngoplasty, are described in detail below.

17.3.2.1 Uvulopalatoflap

This surgery is performed under general anesthesia. After tonsillectomy is done, the mucosa overlying the uvula and soft palate is denuded. The muscular tip of the exposed uvula is then retracted superiorly toward the hard–soft palate junction [19]. The goal of this modification of UPPP is to reduce the likelihood of complications related to scar contracture and the development of nasopharyngeal stenosis. An additional benefit of this modification is that it is potentially reversible since no muscular tissue is resected (see ◘ Fig. 17.5).

17.3.2.2 Z-Palatopharyngoplasty

This modification of UPPP was designed to improve surgical success rates in patients who had undergone previous tonsillectomy [20]. The procedure involves denuding the mucosa of the uvula and soft palate, splitting the soft palate, and completing a subsequent anterolateral advancement, without resection of muscular tissue (see ◘ Fig. 17.6). This results in contracture tension lines resulting in further widening of the airway, particularly in the lateral dimension, which is otherwise difficult to achieve with traditional UPPP in patients who have undergone previous tonsillectomy. The result is higher success rates for Friedman stage II patients. One potential downside to this procedure is that there is a higher incidence of temporary velopharyngeal insufficiency [21].

17.3.2.3 Expansion Sphincter Pharyngoplasty

This modification of UPPP was developed for a selected subset of OSA patients with small tonsils, Friedman stage II or III, and lateral pharyngeal wall collapse noted on endoscopic examination. The procedure involves transection of the inferior aspect of the palatopharygeus muscle with intact attachment to the superior constrictor muscles, rotation superolaterally, and subsequent submucosal attachment to the soft palate anteriorly (see ◘ Fig. 17.7). Once complete, uvulectomy is performed [22]. The goal of this procedure is a reduction in lateral pharyngeal wall collapse. This surgery has better success rates than traditional UPPP, but there may be a slightly increased incidence of postoperative dysphagia (see ◘ Table 17.2).

17.3.3 Transpalatal Advancement Pharyngoplasty

This procedure is typically used as a treatment for patients with persistent OSA and a persistently narrowed retropalatal airway. Surgery involves elevation of the mucosa off the hard palate and subsequent resection of a portion of the hard palate. The soft palate is then advanced anteriorly following tensor tendonolysis (see ◘ Fig. 17.8). The tensor tendons are then re-approximated to soft tissue near the hammulus and the anterior and posterior hard palatal segments are sutured together [23]. The benefit of this surgical technique is that it increases both the anteroposterior and the lateral dimensions of the retropalatal airway.

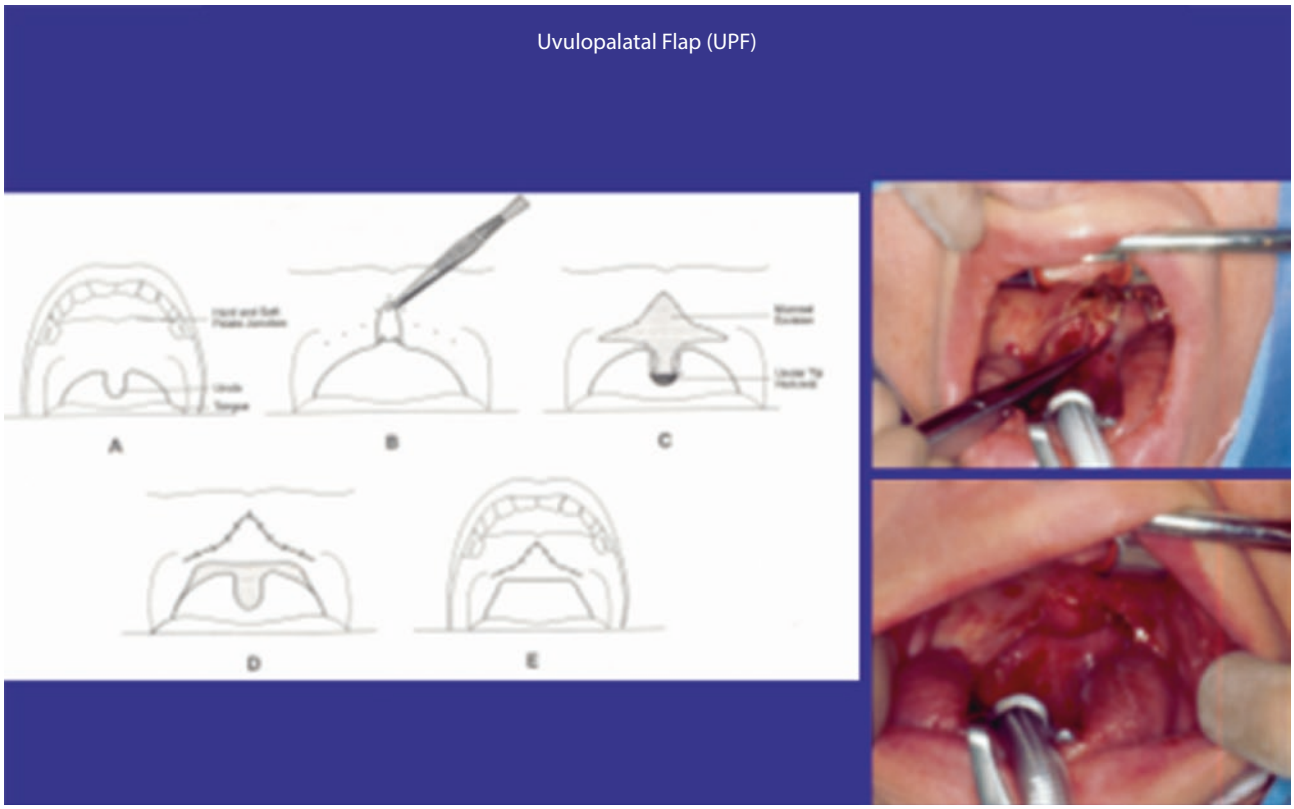


Fig. 17.5 Uvulopalatal flap procedure

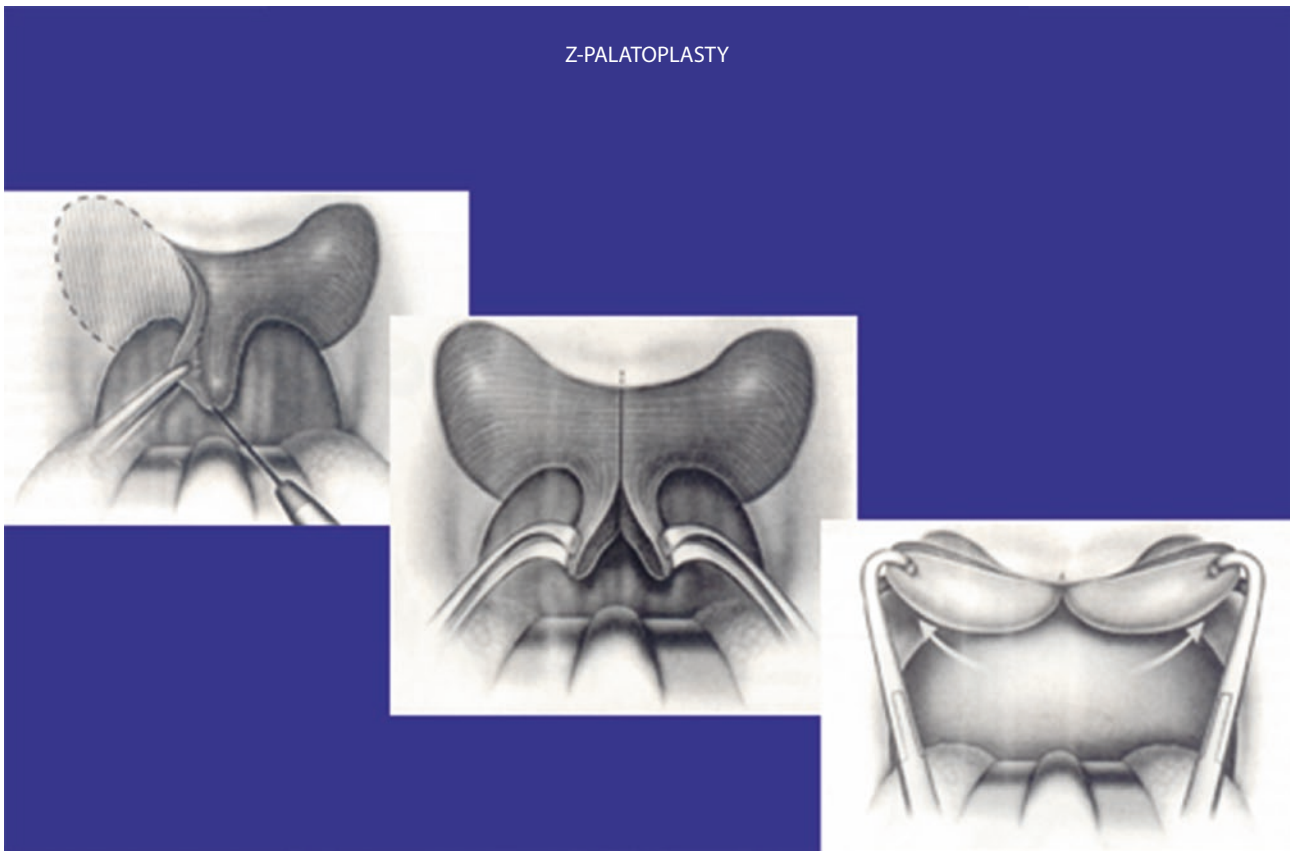


Fig. 17.6 Z-palatoplasty

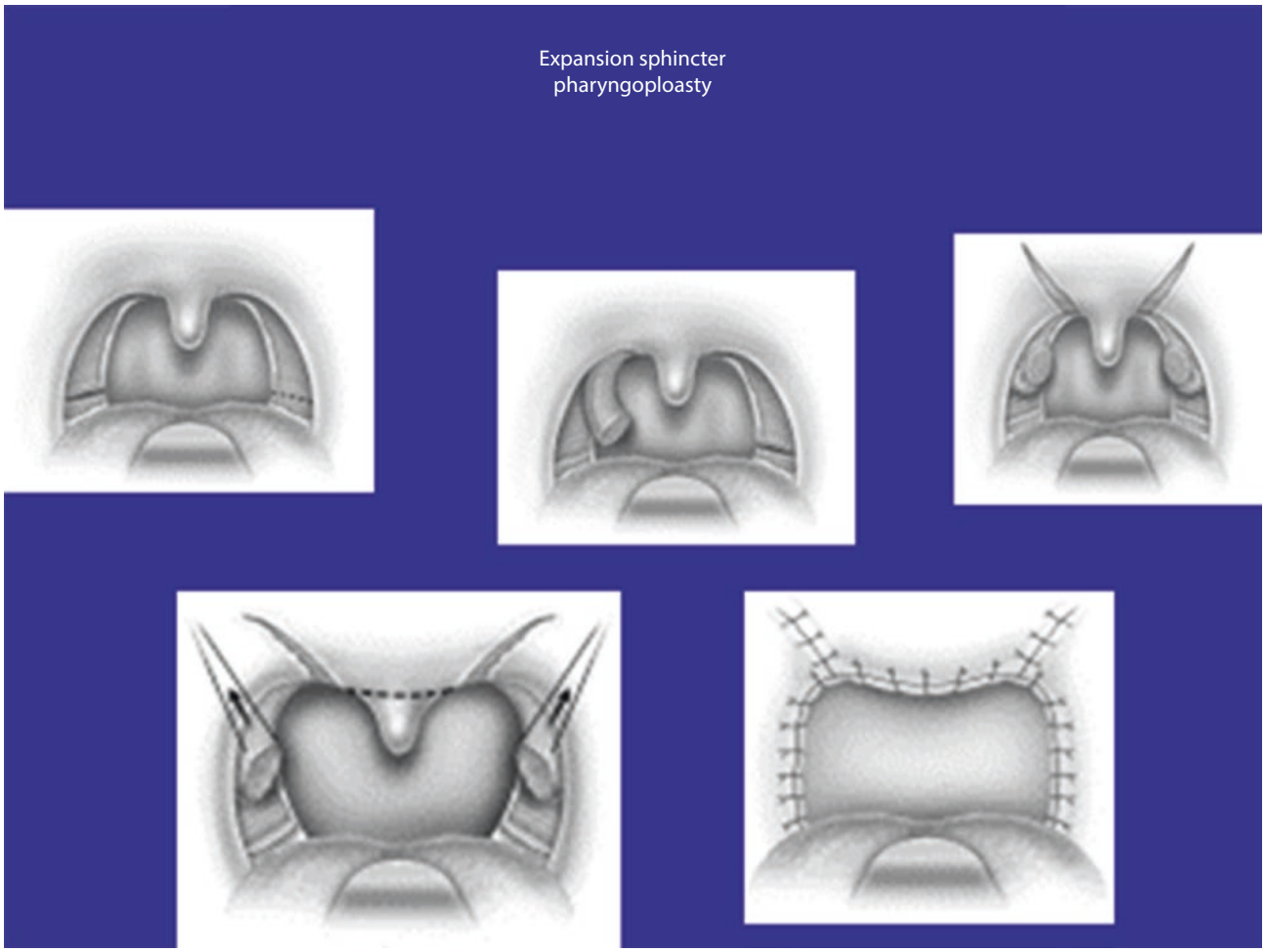


Fig. 17.7 Expansion sphincter pharyngoplasty

Table 17.2 Expansion sphincter pharyngoplasty (ESP) vs. uvulopalatopharyngoplasty (UPPP)

	ESP	UPPP
Preop AHI	44.2	38.1
Postop AHI	12	19.6
Success rate[a]	82.6%	68.1%

[a]AHI reduction 50% and AHI <20

17.3.4 Palatal Stiffening Procedures

Palatal stiffening procedures have been developed to be a less invasive means of improving retropalatal collapse in patients with OSA. These procedures include radiofrequency volumetric tissue reduction and cautery-assisted palatal stiffening operation.

17.3.4.1 Radiofrequency Volumetric Tissue Reduction (RFTA)

Radiofrequency volumetric tissue reduction is a minimally invasive multistep palatal procedure that involves delivery of a high-frequency alternating current into the palate with resultant protein coagulation and tissue necrosis. The ultimate goal is a reduction in soft palatal tissue volume. The efficacy of RFTA in management of mild OSA (defined as AHI 5–15) is comparable to UPP. However, because of less morbidity and fewer treatment-related complications, it is often thought of as a favorable surgical alternative to UPPP in patients with mild OSA [24]. In addition, this procedure can be performed under local anesthesia, avoiding the necessity of general anesthesia with its inherent risks.

17.3.4.2 Cautery-Assisted Palatal Stiffening Operation

This procedure is also less invasive than UPPP. It involves removal of a rectangular area (7 mm × 5 cm) of soft palatal mucosa, uvulectomy, and vertical cuts

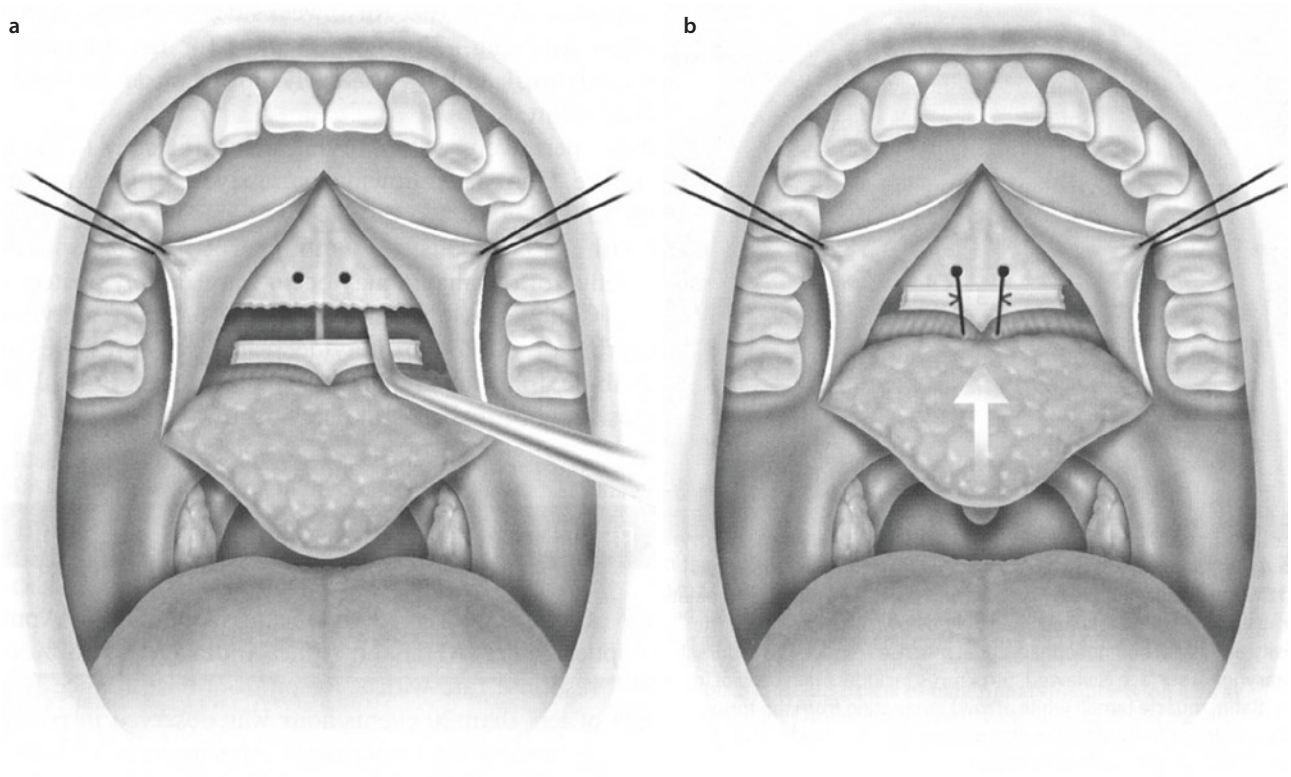


Fig. 17.8 Transpalatal advancement pharyngoplasty

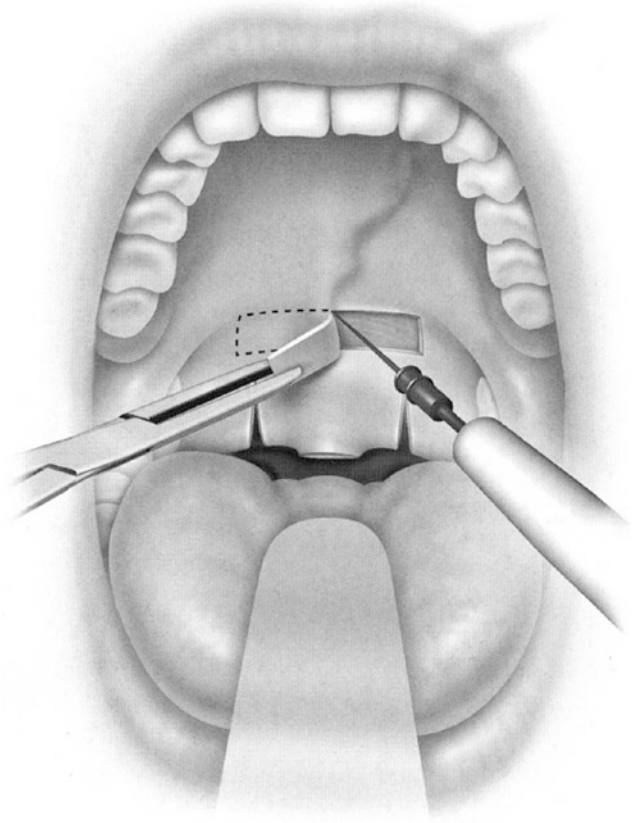


Fig. 17.9 Cautery-assisted palatal stiffening procedure

into the soft palate on either side of the uvula (see Fig. 17.9). Elevation of the soft palate results from fibrosis and retraction at the site of mucosal resection. Similar to RFTA, this procedure can also be performed under local anesthesia.

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Hypopharyngeal Surgery for OSA Patients

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Contents

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18.1 Introduction

Hypopharyngeal airway obstruction in obstructive sleep apnea (OSA) is caused by the prominence of the base of the tongue, lateral pharyngeal wall, and less commonly the aryepiglottic folds or epiglottis. Abnormal bony anatomy such as narrow maxillomandibular arch or maxillomandibular deficiency can significantly contribute to hypopharyngeal obstruction. While continuous positive airway pressure (CPAP) remains the gold standard for treatment for obstructive sleep apnea, surgery is an effective therapeutic option for patients who are intolerant of positive pressure therapy.

Successful obstructive sleep apnea surgery has been traditionally defined as a reduction in apnea–hypopnea index (AHI) to 50% and an AHI less than 20 in the surgical literature. Stuck and Maurer have described an adjusted AHI which also accounts for the percentage of time patients wear CPAP during the night [1]. The adjusted AHI success rate has demonstrated a similar reduction in AHI in a subset of highly selected surgical patients compared to those treated with CPAP.

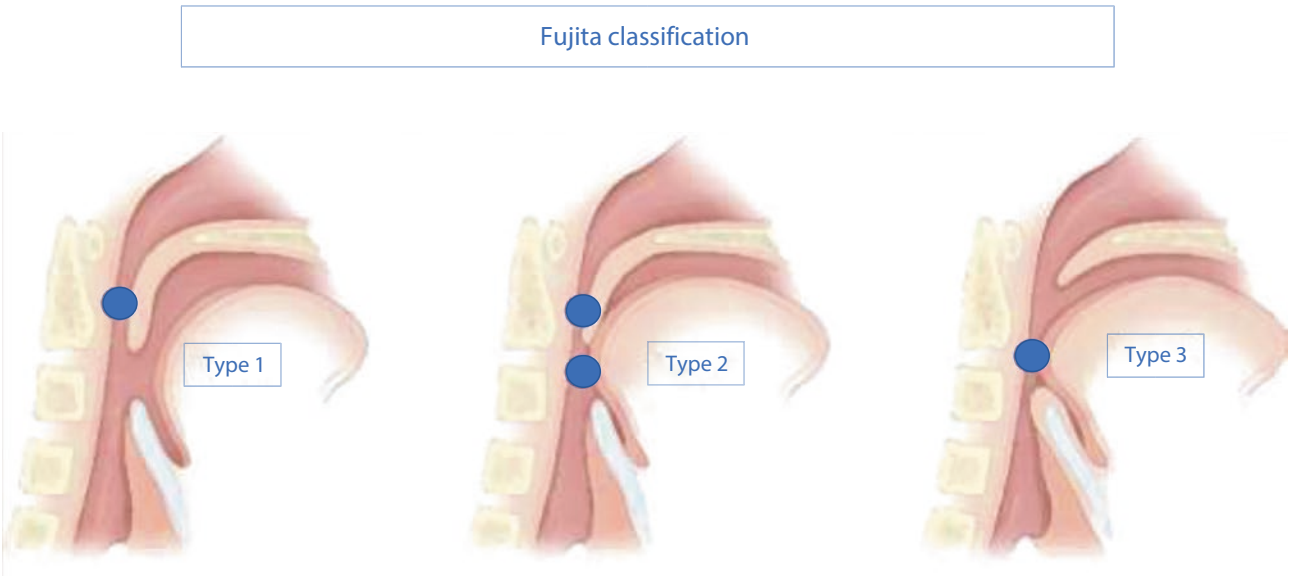
This chapter presents the current role of hypopharyngeal surgery for sleep apnea. The evolution of hypopharynx surgery over the last several decades, preoperative airway evaluation and patient selection, overview of procedures to address hypopharyngeal obstruction, and the future of the field of hypopharyngeal surgery to address OSA, are discussed.

18.2 Historical Perspective

The complexity of the soft tissues and bony anatomy that contribute to hypopharyngeal obstruction, as well as the importance of the hypopharynx to speech and swallowing have presented significant challenges over the last several decades in the surgical management of hypopharyngeal obstruction.

Fujita et al. [2] were the first to present a classification system of the upper airway based on different levels of obstruction, specifically retropalatal, retrolingual, or combined retropalatal and retroglossal obstruction, ◘ Fig. 18.1. Surgical treatment of OSA dates back to Fujita's introduction of the uvulopalatopharyngoplasty (UPPP) in the early 1980s. Based on these different levels of obstruction, the concept of multilevel surgery was defined by Riley et al. [3]. Thorough understanding of the complexity of airway obstruction by upper airway endoscopy demonstrated that the hypopharynx and base of tongue, in addition to the soft palate, are important anatomic components of obstruction in OSA [4]. In addition, lateral collapse of the airway has been noted to be of particular significance in recalcitrant cases [5].

Over the past 30 years, numerous techniques have been introduced to address base of tongue obstruction including CO_2 laser resection, radiofrequency ablation (RFA), suture suspension, skeletal framework surgery, and radiofrequency coblation. Success has been variable, and the gold standard surgical technique, beyond tracheotomy, remains bimaxillary advancement with a success rate greater than 80% [5].



◘ **Fig. 18.1** Fujita classification

The application of transoral robotic surgery (TORS) for safe, effective access to the base of tongue in treatment of OSA was first introduced in 2010 by Vicini et al. [6, 8]. For patients with retrolingual obstruction, TORS gained increasing acceptance among sleep surgeons, given its ability to provide excellent surgical exposure for safely resecting large volumes of tissue via transoral approach. While effective, the previously established transcervical tongue base reduction with hyoepiglottoplasty (TBRHE) procedure presented significant risk of morbidity, including need for both tracheostomy and feeding tube placement, as well as the possibility of tongue weakness and fistula. The comparatively limited morbidity of TORS for OSA resulted in acceptance among sleep surgeons as a part of a multilevel approach in highly selected patients. In 2014, the Federal Drug Administration gave its approval for removal of benign tissue from the base of tongue but stopped short of approving TORS for the clinical indication of OSA. More recently, increased attention has been given to upper airway neuromuscular activity during sleep, as neurostimulation has been utilized as an intervention for patients with OSA. Given that loss of compensatory neuromuscular responses has been shown to play a critical role in airway obstruction during sleep, electrical stimulation of pharyngeal dilator muscles such as the genioglossus have been designed to overcome deficits in airway neuromuscular control and augment airway patency during sleep. The first successful use of hypoglossal nerve stimulation (HNS) to reduce OSA severity in a small cohort of patients was reported in 2001. Apnex Medical then developed the first commercially available implantable HNS device for OSA but ultimately closed in 2013 due to disappointing results from the associated randomized control trial. Currently, Inspire Medical Systems manufactures the only FDA-approved HNS device for OSA, which is an implantable, pacemaker-like pulse generator with a sensing lead and stimulation lead. The sensing lead is implanted between the external and internal intercostal muscles for ventilator effort detection. The stimulator lead is implanted in the submental space to stimulate select branches of the hypoglossal nerve responsible for stimulation of the genioglossus muscle. In 2016, Inspire published 36-month outcomes data for its pivotal STAR trial, which demonstrated significant improvement in outcomes for patients with OSA after HGS implantation. This study was a multicenter, single-arm intervention followed by a randomized controlled, therapy-withdrawal design with study participants serving as their own controls. In total, 126 patients underwent implantation after extensive workup with polysomnography, clinical assessment, and drug-induced sleep endoscopy (DISE). Exclusion criteria included BMI > 32 kg/m^2, AHI > 50 events per hour, central or positional sleep apnea, and concentric palatal collapse. Resolution or significant improvement in sleep apnea was demonstrated in 66% of participants with responses in sleep apnea improvement and quality of life sustained in long-term follow-up at 36 and subsequently at 60 months. With the success of the Inspire system and increasing adoption, the field of neurostimulation for treatment of OSA continues to grow.

18.3 Patient Selection

A number of important factors must be taken into consideration to determine surgical candidacy and choice of intervention, including detailed sleep history, in-office physical exam including evaluation of base of tongue, endoscopy, preoperative contraindications, and predictors of success. Successful surgical outcome in hypopharyngeal surgery for the treatment of OSA is thought to depend on proper patient selection and choice of surgical procedure.

18.4 Physical Exam

Body habitus including body mass index (BMI) and neck circumference should be noted as this has been shown to influence surgical outcomes. A detailed head and neck examination is necessary to identify sites of upper airway obstruction, including the nose, soft palate, lateral pharyngeal walls, and tongue base. Specifically, examination of the nose should include identification of any external deformity, septal position, turbinate size, and the presence of polyps. Oral cavity assessment includes tongue size and position, palate and uvula elongation, tonsil size, Friedman tongue position, dentition, and crowding of the oropharynx. Additionally, evaluation of bony maxillofacial anatomy such as the size and position of the maxilla and mandible (Angle class) must also be considered, ◘ Fig. 18.2.

In addition to direct visual examination, fiberoptic nasopharyngoscopy is critical for complete assessment of the hypopharyngeal airway. With this examination technique, the dimensions of upper airway can be fully assessed including the prominence of the tongue base (Moore classification and Friedman lingual tonsil size) and the lateral pharyngeal wall. ◘ Figure 18.3 demonstrates different classification systems for assessment of upper airway obstruction.

Evaluation of the supraglottic structures may identify a retro-displaced epiglottis that may contribute to

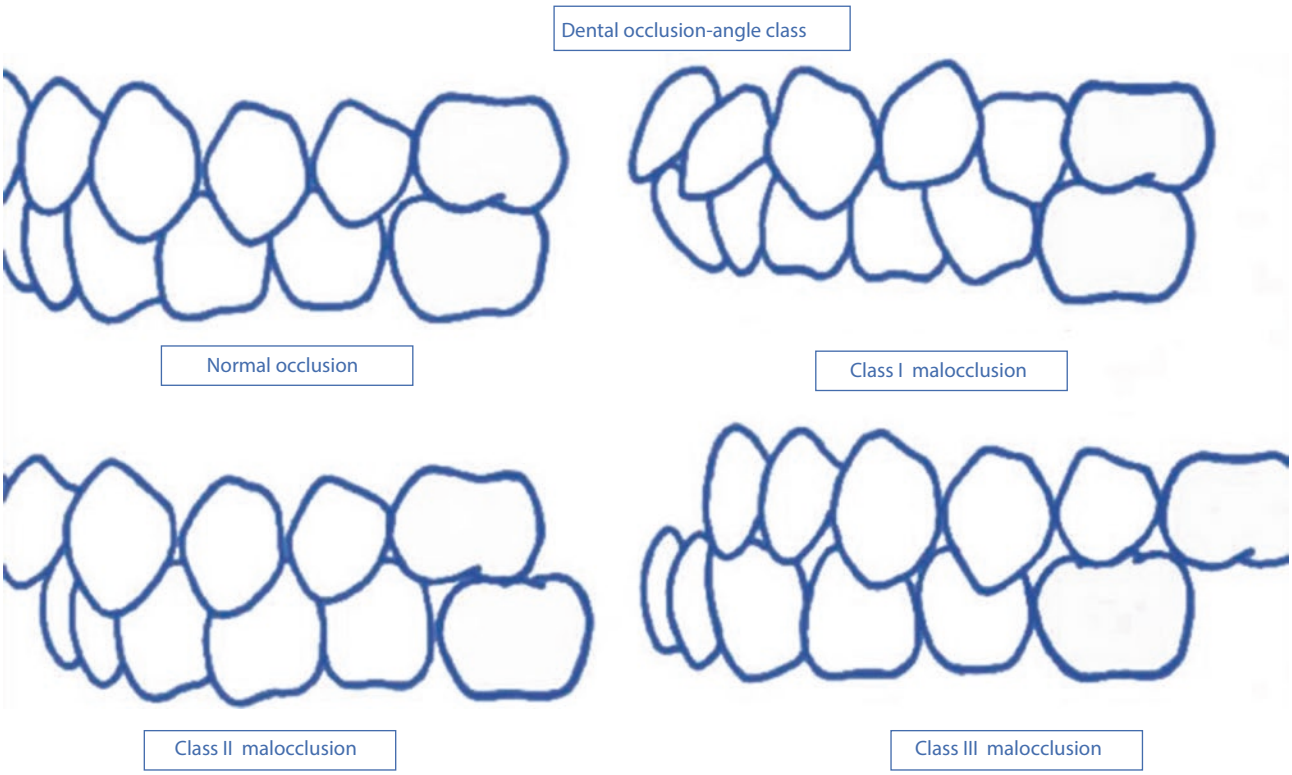


Fig. 18.2 Dental occlusion – Angle class: Class 2 occlusion often associated with significant posterior airway space narrowing due to retro-displacement of tongue

airway obstruction. Dynamic evaluation of the airway under sedation is the best way to identify collapse in the region of the velum, lateral pharyngeal walls, tongue base, and supraglottis. The Mueller maneuver for assessment of airway collapsibility, performed with the patient awake, has been reported to have poor inter-rater reliability and predictive value.

18.5 Imaging I

18.5.1 Imaging

Lateral radiographs can be used to assess facial skeletal anatomy that may contribute to upper airway obstruction. Cephalometric radiography is a widely available low cost two-dimensional representation of the airway that can aid in evaluation of both the bony skeleton and the associated soft tissues, Fig. 18.4. Inferior displacement of the hyoid (>25 mm below the hyoid mandibular plane), narrowed posterior airway (11 mm), and an elongated soft palate (35 mm) are common findings in OSA patients. CT imaging may also be used and has the advantage of allowing for three dimensional reconstruction measurements of the upper airway. At this time, use of dynamic MRI is typically limited to research settings.

18.6 Drug-Induced Sedated Endoscopy

In 1991, Croft and Pringle introduced drug-induced sedated endoscopy (DISE). This technique has gained wide acceptance in Europe and is rapidly gaining popularity in North America as surgeons have recognized its utility in identifying both sites of obstruction and in planning site-specific surgery. DISE is performed with the patient supine and sedated in the operating room, Fig. 18.5. Kezirian et al. popularized the VOTE classification which characterizes both the direction and degree of collapse at the level of the velum (V), oropharynx (O), tongue base (T), and epiglottis (E), Fig. 18.6.

The operating room setup for DISE exam is shown in Fig. 18.6 [7].

The indications for DISE include revision upper airway surgery, in cases where there is no obvious site of anatomic obstruction, and for all patients being considered for HNS. DISE has been shown to identify additional sites of obstruction, not identified on awake examination in the clinic, particularly at the tongue base and supraglottis that can alter the surgical plan in up to one-third of cases. Concentric (sphincter like) collapse at the soft palate is an absolute contraindication for HNS as it has been associated with decreased success rates, Fig. 18.7.

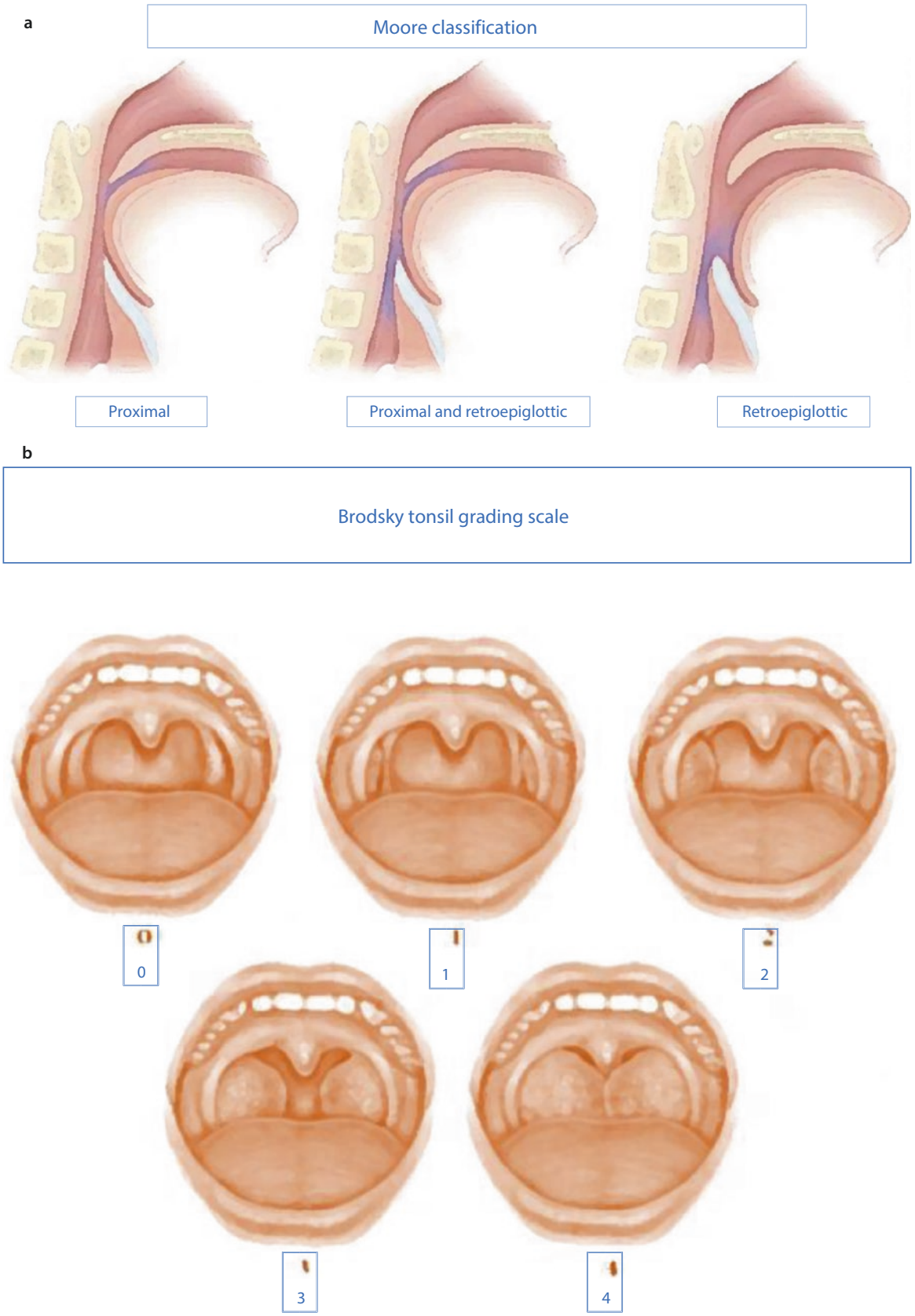


Fig. 18.3 Phenotypic characterization of the soft tissue components of the oropharynx and hypopharynx can be documented during the obstructive sleep apnea directed phyiscal examination using the Moore classification, Brodsky tonsil grading system, Friedman staging and Friedman lingual tonsil classification. The data obtained in the awake patient, combined with knowledge of the skeletal frame work, complements drug induced sleep endoscopy and optimizes surgical planning

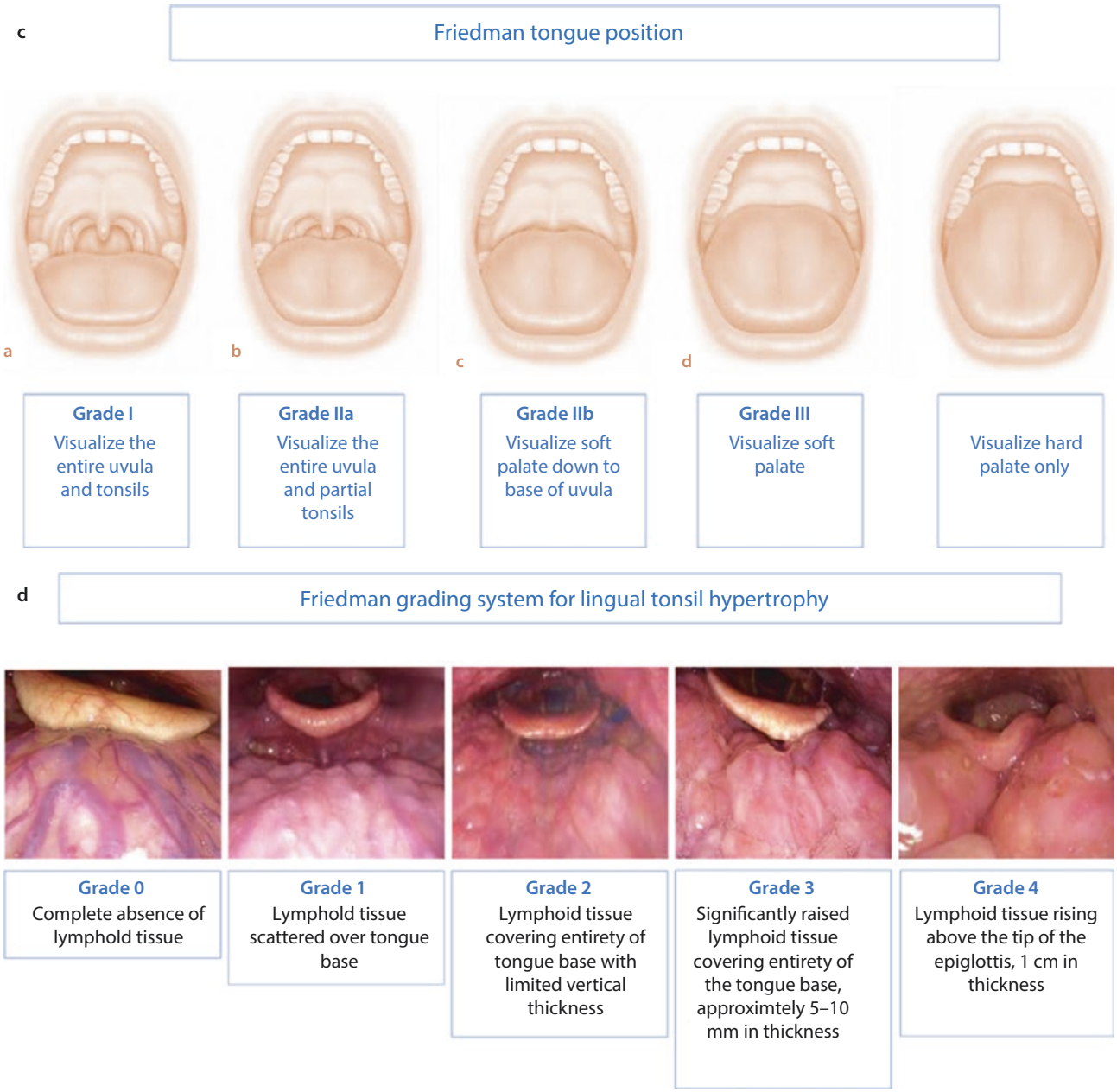


Fig. 18.3 (continued)

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18.7 Treatment Algorithm

Surgical treatment of the hypopharynx consists of procedures designed to prevent sleep-related tongue obstruction. The majority of patients choose surgery due to intolerance of nonsurgical treatments such as CPAP. Goals of surgery and anticipated surgical outcomes should be discussed prior to surgical intervention. Informed consent must be obtained, and patients should be educated regarding the potential risks and benefits of hypopharyngeal surgery.

18.8 Procedures

18.8.1 Transoral Robotic Surgery

Transoral robotic surgery (TORS) for OSA has rapidly gained acceptance among sleep surgeons as a part of a multilevel approach in highly selected patients. Since the first publication of TORS for OSA in 2009, numerous publications representing over 800 patients have been reported. However, due to procedure morbidity and advent of hypoglossal nerve stimulation, the num-

ber of TORS procedures for OSA has more recently declined [13].

Candidates for TORS are patients who have been diagnosed with moderate-to-severe OSA and have failed conservative therapy with weight loss and CPAP. The typical patient has a body mass index (BMI) < 30. As part of the clinical exam, the surgeon will have assessed the airway and selected patients with a Friedman tongue position of 3 or less without significant retrognathia [12]. TORS requires organization and efficiency from a multispecialty team. The team includes the surgeon, anesthesiologist, and a surgical technician who has dedicated time to train for robotics cases and a bedside assistant familiar with TORS. As shown in Fig. 18.8, there is a basic instrument setup that is standard for TORS, and familiarity of the team with this set is essential.

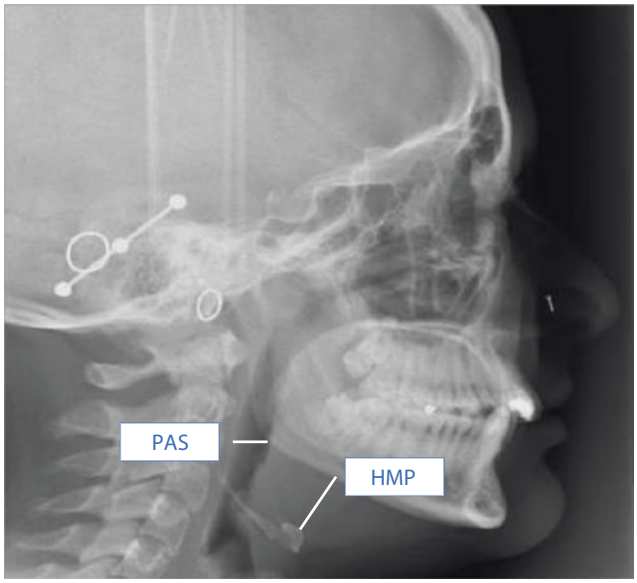

Fig. 18.4 Cephalometric characteristics on lateral radiograph: Posterior airway space (PAS), hyoid to mandibular plane distance (HMP)

18.8.2 Radiofrequency Ablation (RFA)

Radiofrequency (RF) tongue reduction is an outpatient procedure often performed under local anesthesia for volumetric reduction in tongue base tissue. It is particularly helpful for patients without lymphoid hypertrophy who present with a muscular tongue base. An insulated probe is used to deliver radiofrequency energy at 465 KHz. The resultant frictional heat causes tissue injury and results in tongue volume reduction via coagulation necrosis and healing by scar. RFA is often an adjunctive procedure, performed along with other hypopharyngeal airway surgical procedures.

Prospective studies of RF tongue reduction have demonstrated a significant improvement in respiratory disturbance index without a change in speech or swallowing [9]. Surgical risks include superficial tongue ulceration, persistent odynophagia, and infection [10, 11].

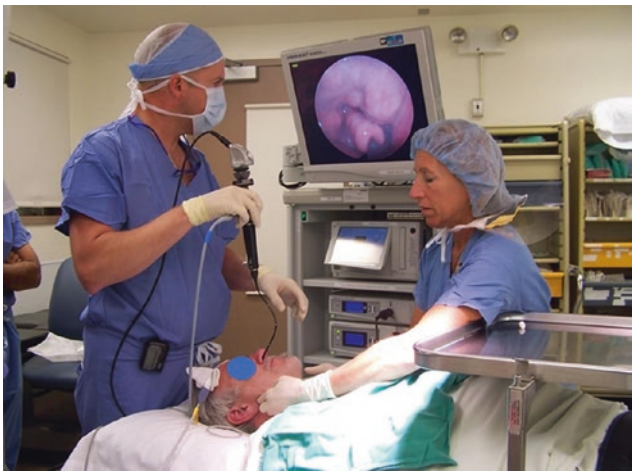

Fig. 18.6 DISE operating room setup

Fig. 18.5 VOTE classification which characterizes both the direction and degree of collapse at the level of the velum (V), oropharynx (O), tongue base (T), and epiglottis (E)

Drug-induced sleep endoscopy: VOTE classification

	DIRECTION		
LEVEL	AP	LATERAL	CONCENTRIC
Velum			
Oropharynx			
Tongue Base			
Epiglottis			

Degree of obstruction: 0 – No obstruction, 1 – partial obstruction, 2 – complex obstruction, X – not observed

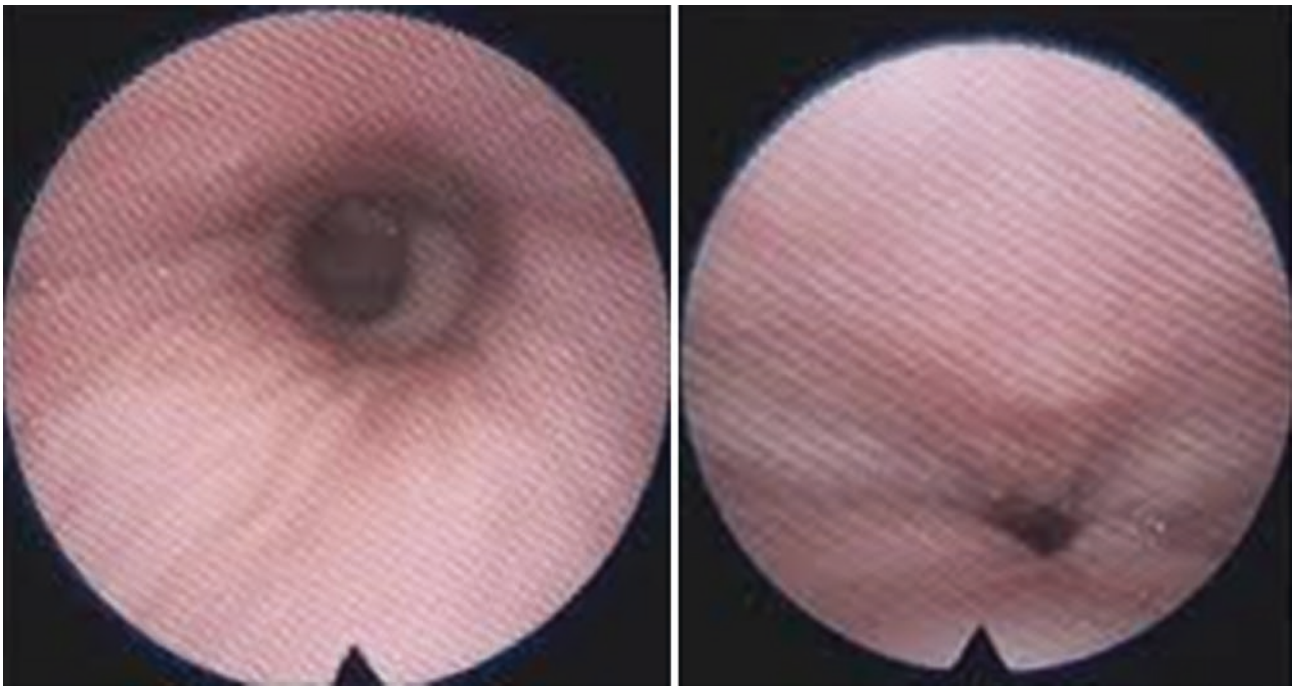

Fig. 18.7 Circumferential collapse at velum identified on DISE

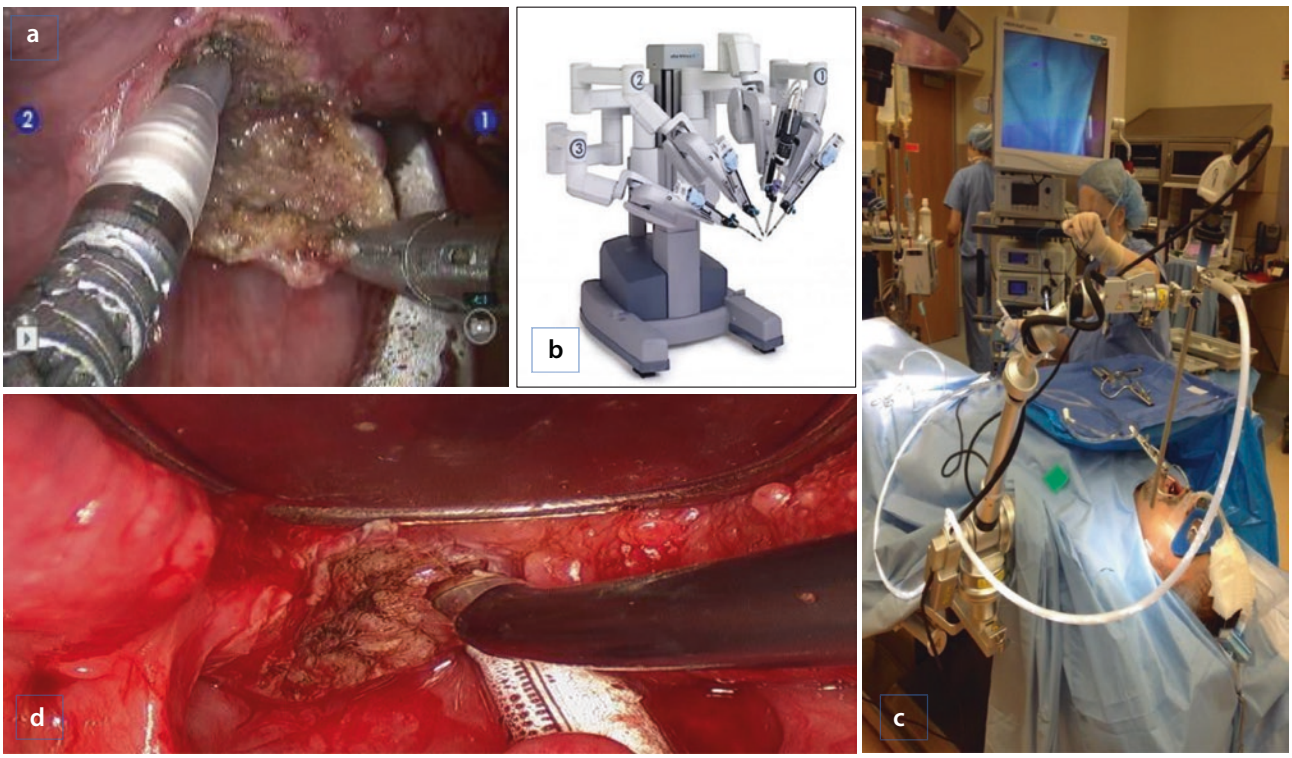

Fig. 18.8 Transoral robotic surgery (TORS): **a** TORS lingual tonsillectomy, **b** DaVinci robot, **c** CELL operating room setup, **d** CELL technique

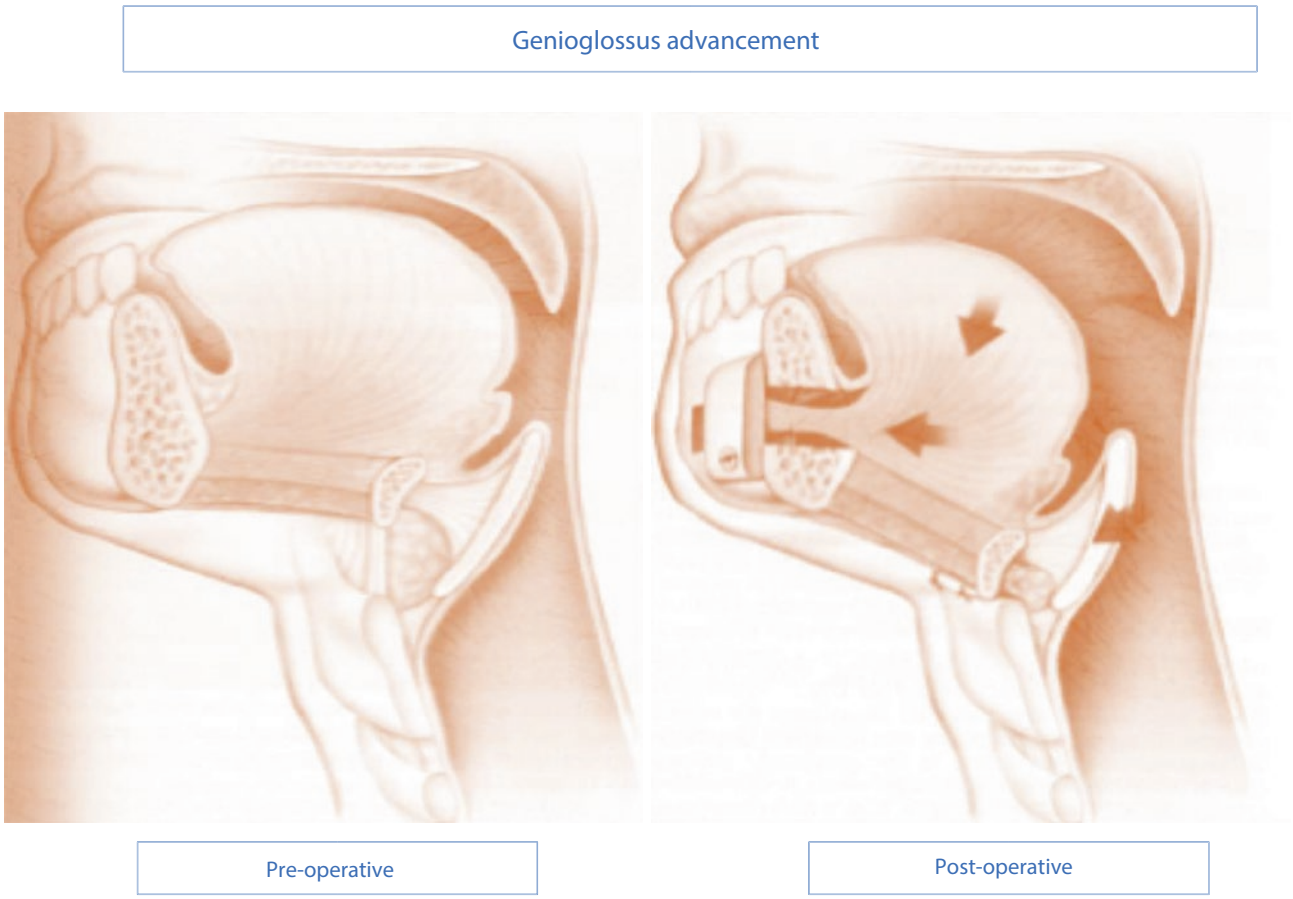

Fig. 18.9 Genioglossus advancement involves movement of the genial tubercle with genioglossus insertion forward, to place tension on the tongue musculature and limit posterior displacement during sleep

18.8.3 Genioglossus Advancement

The genioglossus advancement procedure involves a bicortical osteotomy inclusive of the genial tubercle with its genioglossus muscle insertion anteriorly to place tension on the tongue musculature and limit posterior displacement during sleep, Fig. 18.9. A rectangular osteotomy along the symphysis of the mandible is made intraorally. The rectangular segment is then advanced and either rotated or secured with titanium microplates to prevent retraction. Typically, genioglossus advancement is performed in conjunction with other sleep apnea surgical procedures such as palatal repositioning procedures and hyoid advancement to maximize success. Like RFA, genioglossus advancement is helpful for patients with a large muscular tongue. Variable surgical success with genioglossus advancement procedures, ranging from 20% to 70%, highlights the difficulty in accurately predicting success. Anatomic factors, body habitus, and OSA severity have been demonstrated to influence surgical success. Potential risks associated with genioglossus advancement include infection, hematoma, mandibular fracture, and paresthesia of the lower teeth.

18.8.4 Tongue Base Suspension

Like genioglossus advancement, tongue base suspension seeks to reduce tongue collapsibility during sleep, Fig. 18.10. Via either an intra-oral or submental incision, a suspension suture is brought from an anchor screw on the inner surface of the mandible to the base of the tongue. This is then tightened such that a hammock effect for the tongue is created. This short, relatively simple procedure is often performed in conjunction with palatal repositioning procedures. Variable success rates ranging from 20% to 82% have been noted. Risks of the procedure include infection, injury to tooth roots, and possible detachment of the anchor screw.

18.8.5 Hyoid Suspension

Hyoid advancement is performed by either advancing the hyoid bone in an anterior-inferior (thyrohyoidpexy) or anterior-superior direction (hyoidmandibulopexy), and is often performed in conjunction with genioglossus

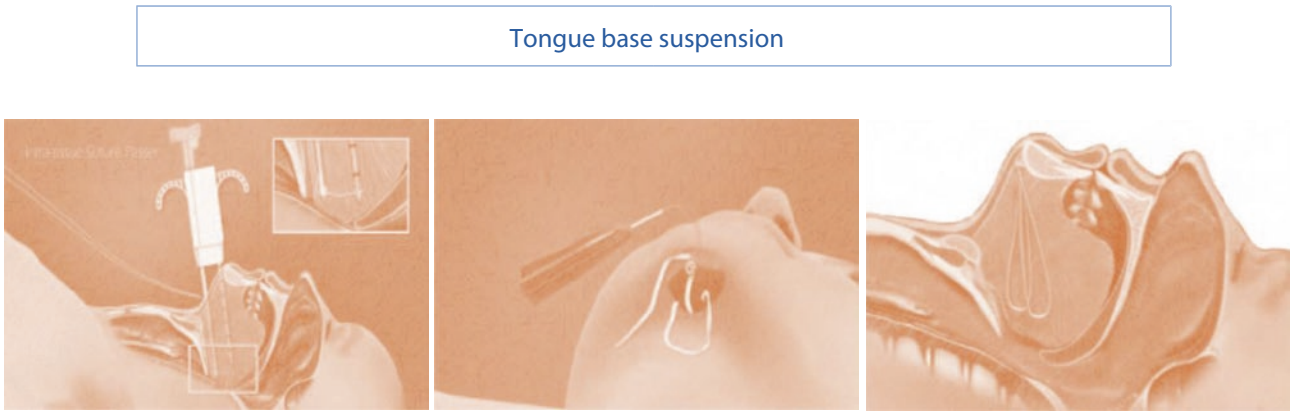

Fig. 18.10 Tongue base suspension procedure

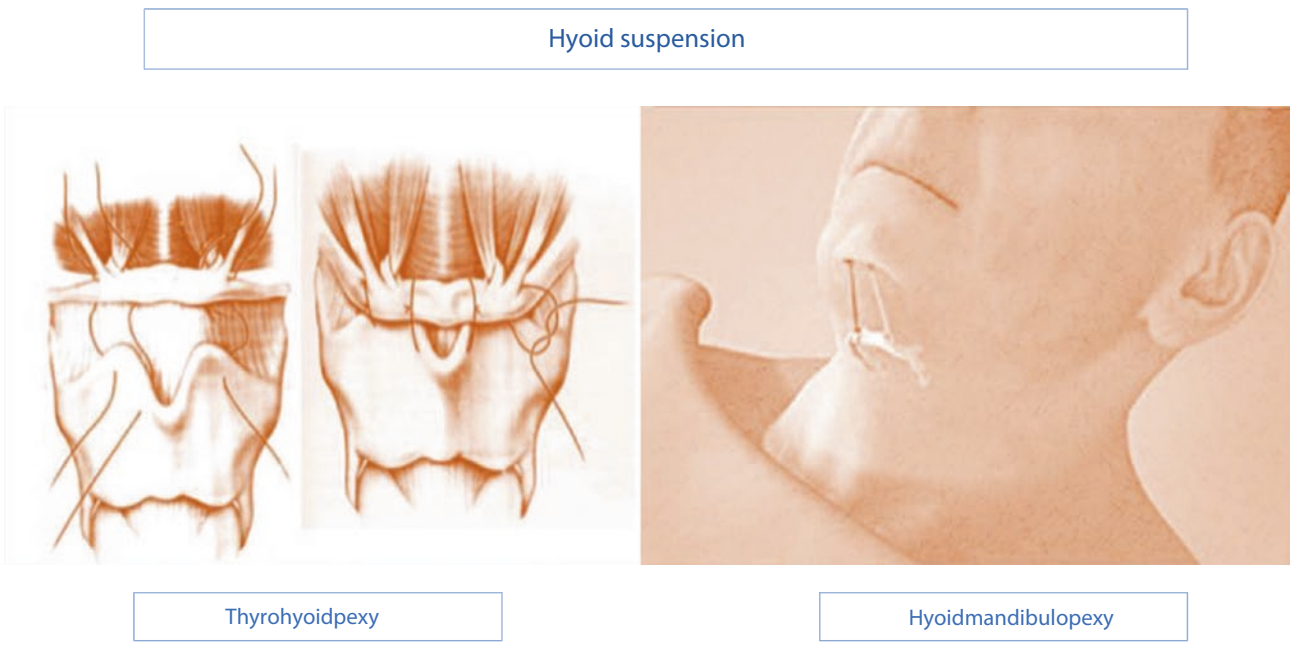

Fig. 18.11 Hyoid suspension procedures

advancement and/or with palatal repositioning procedures for obstructive sleep apnea, Fig. 18.11. The thyrohyoidpexy procedure advances the hyoid bone over the thyroid lamina which is then secured with permanent sutures placed through the superior portion of the thyroid cartilage. Anterior repositioning of the hyoid bone by attaching it to the thyroid cartilage expands the airway. While there is variable success with hyoid advancement, the procedure is generally well tolerated with surgical risks limited to infection, seroma formation, and dysphagia.

The hyoid can also be suspended to the posterior surface of the mandible, which pulls the tongue base in an anterior-superior direction. The hyoid bone is fixed to the mandible with anchor sutures placed through a submental incision. This is a minimally invasive technique and works well for patients with a large muscular tongue base without lymphoid hypertrophy.

Suspension procedures have been shown to be modestly effective when performed in isolation; the best results are obtained when performed in conjunction with palatal surgery.

18.8.6 Maxillomandibular Advancement (MMA)

Maxillomandibular advancement increases the retropalatal and retrolingual airway by advancing the maxilla and mandible through Le Fort I maxillary and sagittal-split

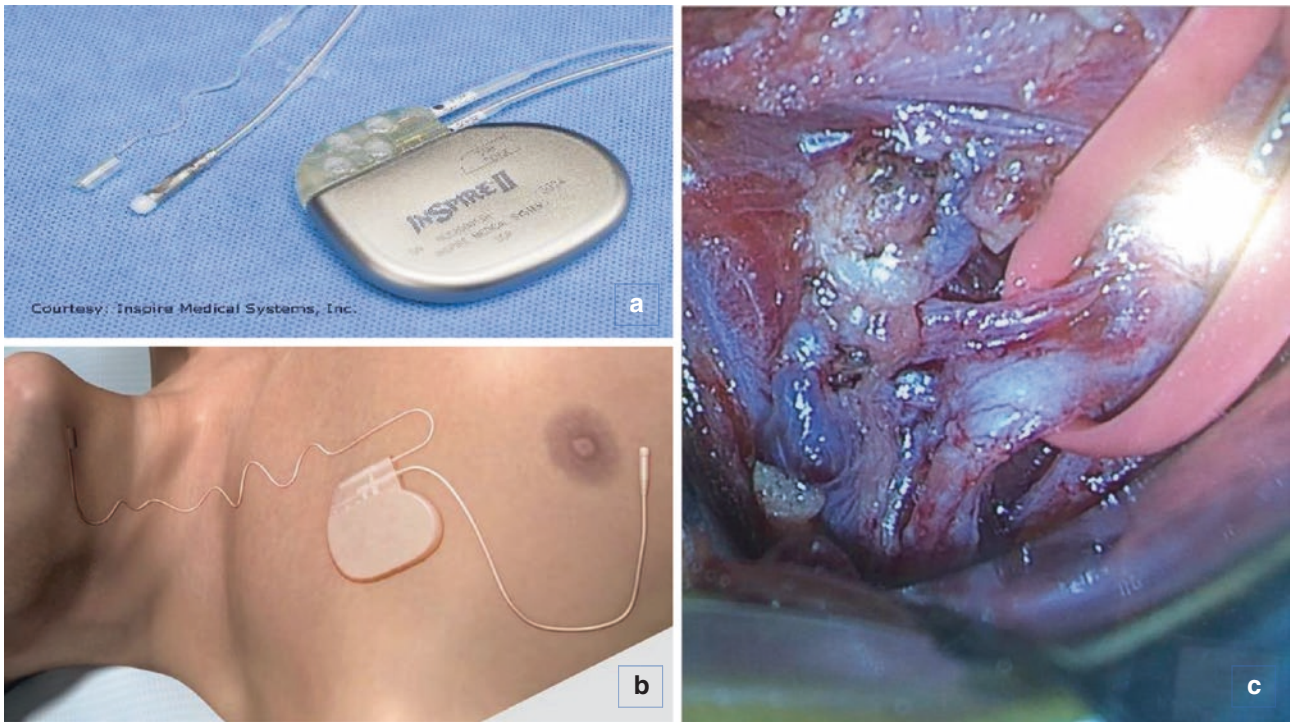

Fig. 18.12 Inspire hypoglossal nerve stimulator: **a** Inspire pulse generator, sensing lead, and stimulating lead, **b** Inspire layout, **c** intraoperative image of hypoglossal nerve dissection with red vessel loop around inclusion branches of the nerve

mandibular osteotomies. Typically, this procedure is performed when other surgical interventions have been unsuccessful. Potential complications include malocclusion, nonunion, or malunion temporomandibular joint problems and nerve paresthesia. Despite the risks and relative morbidity of the procedure, the success rate of this procedure has been reported to be between 80% and 90%.

18.8.7 Hypoglossal Nerve Stimulators

HNS therapy has been shown to significantly reduce AHI in moderate-to-severe OSA patients with strict inclusion criteria including BMI <32, an AHI between 15 and 65, and favorable pattern of upper airway obstruction during DISE. The hypoglossal nerve stimulator system involves three main components: the implantable pulse generator (IPG), the sensing lead, and the stimulator lead, Fig. 18.12. The IPG is surgically implanted into an infraclavicular subcutaneous pocket superficial to the pectoralis major and produces electrical impulses. The sensing and stimulation leads are tunneled subcutaneously from the IPG, respectively, to the lower ribs and hypoglossal nerve. Closed-loop stimulating systems produce impulses with inhalation through the IPG to the hypoglossal nerve via a tripolar electrode that wraps around the nerve resulting in opening of the retroglossal airway as well as the retropalatal airway due to mechanical coupling of the palatoglossal and genioglossus musculature, Fig. 18.13.

18.9 Future Directions

Increasingly, neurostimulation treatment strategies are viewed as a preferred alternative to bony or soft tissue surgical interventions for treatment of OSA in CPAP-intolerant patients. These procedures have a lower risk profile and are associated with significantly reduced postoperative pain and recovery time. While strict inclusion criteria and cost are currently the primary barriers for increasing adoption of hypoglossal nerve stimulation, novel stimulation strategies are being designed and invigorating the field of sleep medicine. Additional clinical trials are underway assessing the efficacy of external HNS stimulation devices.

Patient selection is an area of active research. It is now widely recognized that surgeons must address both the anatomic and nonanatomical parameters such as loop gain (a measure of ventilatory stability) and arousal threshold using a physiology-based model to better predict outcomes after upper airway surgery for obstructive sleep apnea.

Additionally, recent research has indicated that efferent motor pathways may be stimulated through recruitment of reflex afferent input to respiratory and upper airway motor control centers. Compared with direct unilateral hypoglossal nerve stimulation, recent studies in animal models has suggested esophageal distention, electrical auricular stimulation, sciatic nerve stimulation, and pulsed nasal insufflation of heated, humidified

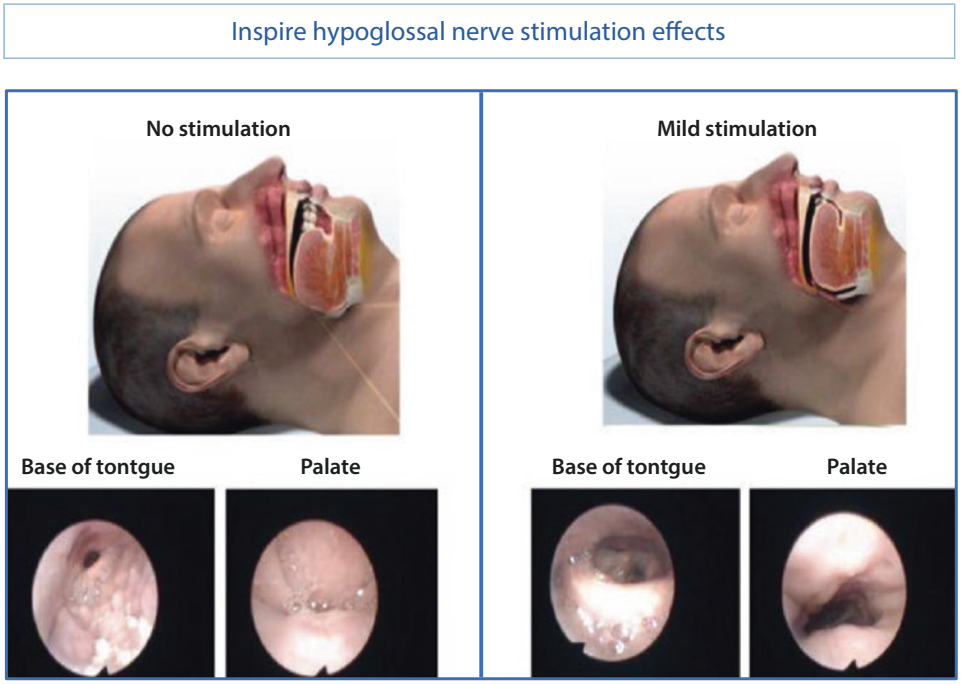

Fig. 18.13 Effects of hypoglossal nerve stimulation. (Courtesy of Inspire Medical Systems)

air may activate respiratory brainstem motor nuclei to initiate a more coordinated brainstem response involving several cranial nerves and upper airway muscles to improve airway patency during sleep. The field of neurostimulation for treatment of OSA is exciting and nascent as promising new approaches for activating efferent and afferent motor pathways are currently in early stage development.

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Management of Obstructive Sleep Apnea (OSA) in Craniofacial Patients

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Contents

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19.1 Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome

Many screening tools and questionnaires have been described for the evaluation of pediatric patients with suspicion of obstructive sleep apnea (OSA). These screening tools could not be relied upon in patients with craniofacial syndromes since they commonly present with other manifestations such as cognitive and hearing deficits and ocular deficits, among others.

Advanced imaging has shown to be powerful in the assessment of structural abnormalities and in helping to locate the anatomical location of the obstruction along the upper airway. Many software tools incorporate measuring tools to assess the airway, providing three-dimensional measurements.

Polysomnography has proven to be the gold standard for diagnosis of OSA in pediatric population. However, other alternatives have been proposed in the past for screening and diagnosis of this condition.

19.1.1 Imaging

Plain film radiographs including lateral cephalometric radiographs, AP/PA cephalometric radiographs, and panorex radiographs have been used in the past as an efficient and inexpensive diagnostic tool in the assessment of dentofacial deformities since they are readily available at orthodontists and oral surgeons' offices. These X-rays are used to diagnose bony anatomy, position of the maxilla and mandible, length of the soft palate, and position of the hyoid bone. The major disadvantage of pain films is the inability to study soft tissues.

Computer tomography (CT) and cone-beam CT (CBCT) significantly improve the soft tissue contrast and detail. In addition, 3D studies aid in examining the airway in all pains as compared to plain films allowing for precise measurement of minimal cross sectional area and volumetric assessment of the airway. CT scans also provide with detailed anatomical information of the obstruction site, this is relevant in planning upper airway surgery when necessary. However, controversy still exists in the usefulness of 3D imaging in awake non-supine patients, since the true anatomy of asleep patient cannot be assessed accurately.

Comparison of CBCT before and after surgery determines the difference in airway volume. However, no guidelines have been established regarding minimal cross sectional area and airway volume to diagnose OSA based on these findings.

Magnetic resonance imaging with compared to CBCT and CT scans offers various advantages including lack of ionized radiation and better analysis of the soft tissue structures. The lack of ionized radiation makes MRI the imaging technique in children with OSA syndrome.

The application of these imagine technique in craniofacial patients can be challenging to the lack of cooperation by the patient. Sedation may be required for the patient to tolerate these studies without moving while acquiring the imaging. On the other hand, obtaining the images with the patient sedated in a supine position may replicate the airway size and position during sleep, but this is still controversial and no advantages have been proved.

19.2 Role of Sleep Upper Airway Endoscopy in the Diagnosis of OSA

Sleep endoscopy has been used since 1991 to examine upper airway during pharmacologically induced sleep. Even though the goal standard up to date remains polysomnographic studies, other studies such as sleep endoscopy aid in identifying the level and degree of upper airway collapse. This diagnostic tool can aid in the development of a treatment plan guided toward obstruction.

Multiple grading systems have been developed to grade the severity of OSA. Berchard et al. described a system that focuses on five anatomical sites including nose and nasopharynx (N), palatine plane, uvula or tonsils (P), tongue (T), larynx (L), and hypopharynx (H). Furthermore, the obstruction was categorized as partial (1) or complete (2) for each one of the above. So, if the patient has partial obstruction at the nose and tonsils and complete obstruction at the tongue, this would be labeled as N1P1T2 and would be assigned a value of 4 (1 + 1 + 2). A score range of 0–2 was associated with mild OSA, 3–4 with moderate, and more than 4 with severe.

19.2.1 Mandibular Deficiency

19.2.1.1 Pierre Robin Sequence

Pierre Robin Sequence (PRS) is a congenital malformation occurring in 1 in 30,000 live births. This condition is characterized by the triad of severe mandibular hypoplasia, glossoptosis, and cleft palate. Many of these patients present with airway obstruction.

Sher et al. described four types of airway obstruction in patients with PRS based on flexible fiberoptic nasopharyngoscopy findings. The most common form, Type I, is defined by obstruction due to posterior movement of the tongue against the posterior pharyngeal wall. Type II obstruction is due to posterior and superior displacement of the tongue, causing obstruction from the tongue, the velum, and the pharyngeal wall in the

superior oropharynx. Type III obstruction is caused by prolapse of the medial pharyngeal wall. Type IV obstruction is due to constriction of the pharynx circumferentially by lateral pharyngeal walls and the tongue.

Syndromes presenting along with this condition include Stickler syndrome, Nager syndrome, Treacher Collins, and velocardiofacial syndrome. Al-Samakri et al. showed up to 60% of Pierre Robin sequence cases present in combination with other syndromes; 80 to 90% of these patients also present with cleft lip and palate.

The prevalence of OSA in this population ranges from 46% to 100% across the literature. This is due to upper airway obstruction related to micrognathia, posterior position of the tongue, and cleft palate.

Management of OSA in patients with Pierre Robin sequence involves treating the upper airway by treating cleft palate, stabilizing the pharyngeal wall, and widening the hypopharynx. Emergent tracheostomy is necessary in cases where airway is severely affected. Other necessary treatments include mandibular distraction and application of oral devices to widen the palate and the upper airway.

19.2.1.2 Craniofacial Microsomia

Craniofacial microsomia is condition characterized by structures derived from the first and second branchial arches, including maxillomandibular complex, facial nerves, ears, and soft tissue. The incidence of craniofacial microsomia ranges from 1 in 3500 to 1 in 20,000 live births in the literature and it is the second most common congenital facial defect, after cleft lip and palate. Craniofacial microsomia can be unilateral, known as hemifacial microsomia, or bilateral.

The mandible is commonly affected in craniofacial microsomia. Mandibular and maxillary hypoplasia, along with adenotonsillar hypertrophy and glossoptosis are important contributing factors for obstructive sleep disorders in this patient population.

The orbits, mandible, ears, nerve, soft tissue (O.M.E.N.S) classification has been widely used to describe the different anatomical variations and severity of this condition. Later, the word Plus was added to the classification to describe any alterations outside of the craniomaxillofacial complex. The OMENS-Plus score is calculated grading each anatomical abnormality from 0 to 3. Where 0 is normal, 1 is abnormal size, 2 is abnormal position, and 3 for the combination of both. Pruzansky graded the mandible, and this classification was later modified by Kaban. Mandibular grading assesses the mandibular ramus and condyle [0 = normal; 1 = small mandible, short ramus; 2 = abnormally shaped ramus and condyle] with (a) glenoid fossa anatomically acceptable compared to the contralateral side, (b) temporomandibular joint (TMJ) displaced anteriorly, medially, or inferiorly with hypoplastic condyle; and 3 = complete absence of ramus and fossa.

The prevalence of OSA in patient with craniofacial microsomia varies significantly in the literature from 7% to 67% taking into consideration the severity of the anatomical defects as well as the definition of OSA in the different studies.

19.2.2 Mid-Face Deficiency

19.2.2.1 Crouzon's Syndrome

Crouzon's syndrome is an autosomal dominant condition. It is considered a syndromic craniosynostosis with birth prevalence of 1 in 60,000. This condition can present as an isolated entity or in combination with other malformations. Important clinical characteristics of this condition include bilateral coronal craniosynostosis, exorbitism with hypertelorism, and maxillary hypoplasia, among other variations. The combination of skull and maxillary growth disturbance often results in increased intracranial pressure and obstructive sleep apnea; 40 to 85% of these patients are diagnosed with obstructive sleep apnea at some point and the severity is usually determined by the anatomy of the airway and the degree of mid-face deficiency.

Treatment of obstructive sleep apnea for these patients is different from treatment of adults with just OSA. Children with Crouzon's syndrome often present severe mid-face deficiency, increased scleral show, and other craniofacial deformities. These deformities and skeletal deficiencies causing obstructive sleep apnea must be address simultaneously. Many surgical options have been proposed for treatment of OSA in children with Crouzon's syndrome depending on the severity of the case. Severe emergent cases may require tracheostomy for immediate airway protection. Other treatment modalities include LeFort III osteotomies for severe mid-face deficiency, LeFort I osteotomies, distraction osteogenesis (DO), and orthodontic/orthognathic surgery. Conservative methods such as CPAP application have been recommended for less severe cases.

19.2.2.2 Apert Syndrome

Apert syndrome is a rare condition that affects 1 in every 65,000 births. It is an autosomal dominant transmitted disorder that results in the abnormal development of the skull and face due to the premature closure of sutures, primarily the coronal sutures resulting in brachycephaly and turricephaly (Omar Breik).

This syndrome is characterized by craniosynostosis, mid-facial hypoplasia, syndactyly of the hands and feet, and other malformations of the limbs (Omar Breik). In this syndrome, the maxilla is also involved in the synostosis. Therefore, up to 75% of these patients present with cleft palate or bifid uvula.

The causes of OSA in patients with Apert syndrome are similar to those mentioned in Crouzon syndrome. OSA

is often multi-level problem in these children. However, since mid-face deficiency is the primary cause for airway constriction, monoblock or LeFort III advancement with distraction is usually the treatment of choice (Doerga).

19.2.3 Both Mid-Face and Mandibular Deficiency

19.2.3.1 Treacher Collins Syndrome

Treacher Collins syndrome is an autosomal dominant condition that affects 1 in 25,000 to 1 in 50,000 births. This craniofacial syndrome is characterized by craniomaxillofacial soft tissue and skeletal hypoplasia of the first and second branchial arches. The major features include mandibular micrognathia, conductive hearing loss, malar deficiency, and down-slanting eyes and in 33% of the cases with cleft palate.

The treatment of this patient population is carried through the different growth phases. First, to support the airway when the child is born, then during early childhood to support feeding and speech, and definitive treatment is performed when growth has been completed to address the facial defects.

19.2.3.2 Goldenhar Syndrome

Goldenhar syndrome is a congenital disorder that affects the first and second branchial arches and it affects 1 of 5600 live births. Unlike microfial microsomia, Goldenhar syndrome also affects ears, eyes, and vertebrae. Cardiac and neurologic conditions are associated with this disorder; however, they are not necessary for the final diagnosis.

Obstructive sleep apnea affects 11.6% of patients with Goldenhar syndrome. The etiology of the obstructions is multifactorial, including anatomical and neurological alterations. Unlike patients with hemifacial microsomia, patients with this syndrome show increase in CO_2. This additional finding may correlate with dysfunction of the regulation of respiration during sleep caused by neurological impairment.

Treatment of OSA in patients with Goldenhar syndrome like in other syndromes varies depending on the severity of OSA as well as the anatomical area in the obstruction. Severe cases require more aggressive interventions such as tracheostomy to protect the airway and improve oxygenation levels while definitive treatment is performed.

19.3 Surgical Correction

In the newborn emergency setting, tongue–lip adhesion and/or tracheostomy may be required to secure and maintain the airway in craniofacial syndromes with profound airway obstruction and respiratory distress. Tracheostomy is a bypass procedure that can be used as a temporary measure to maintain the airway while other procedures to improve the airway are planned and performed.

These immediate procedures, performed shortly after birth to address the airway, are then revised. Once facial growth is adequate and life-threatening obstruction is treated, release of the tongue–lip adhesion and a reversal of tracheostomy are performed.

Tonsillectomy/adenoidectomy and palatal procedures such as uvulopalatopharyngoplasty (UPPP) address obstructions of the oropharynx. Adenotonsillectomy is the procedure of choice for surgical management of OSA in children.

Procedures such as hyoid suspension, partial glossectomy, and radiofrequency ablation of the tongue base are soft tissue procedures that will address obstruction of the hypopharynx.

Timing of surgery can be during active phases of growth or after growth cessation.

It is possible that surgery performed during active phases of growth is susceptible to relapse and there is need for additional surgical procedures including repeat of the surgical procedure to obtain the desired functional result.

Ilizarov described his concept of distraction osteogenesis (DO) for limb reconstruction in 1952 and McCarthy described the application of DO to the human craniomaxillofacial skeleton in 1989. As described, the principle includes a surgical osteotomy after subperiosteal dissection, a latency period of 4–5 days, a distraction period of a rate of sometimes greater than 1.0 mm per day, and then a consolidation period. Commonly used distraction protocols are based on a clinical goal of 20% overcorrection. The overall treatment time for distraction of the craniofacial skeleton can be less than 3 months. Successful maxillary, mid-face, zygomatic, orbital, mandibular, and cranial bone distraction have all been described.

Distraction osteogenesis is a viable treatment option for obstructive sleep apnea in craniofacial syndromic patients. Depending on the anatomic location of the obstruction, the patient may be a candidate for mandibular or mid-face distraction (see ◘ Fig. 19.1). Virtual surgical planning based on medical grade computed tomography scans or cone-beam CT scans (CBCT) can be helpful to predict the surgical design.

Mid-face distraction osteogenesis can be performed intraorally or extraorally with a rigid external distraction (RED) device. With proper patient selection, RED can be utilized from age 5 through adulthood (see ◘ Fig. 19.2). Similarly, mandibular distraction can be performed with an internal or external device.

Unilateral or bilateral costochondral graft (CCG) reconstruction of the mandible can be an option for craniofacial patients with severe mandibular deficiency involving the ramus and condyle unit (see ◘ Fig. 19.3). It is recommended that patients who receive mandibular reconstruction with either distraction osteogenesis

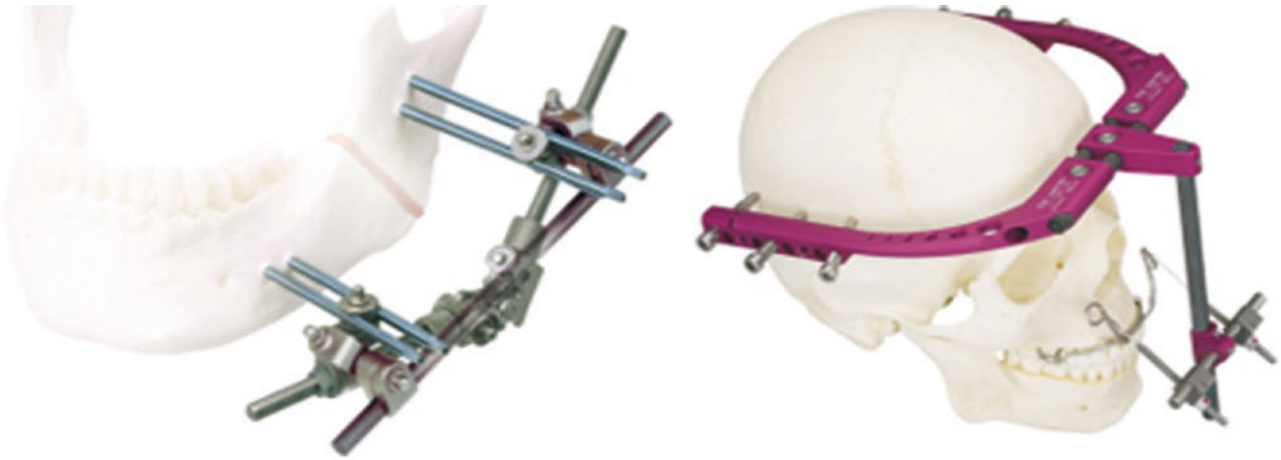

Fig. 19.1 Graphic description of extra-oral mandibular distractor (left) and rigid external distractor (RED) device (right)

Fig. 19.2 Mid-face deficiency in teenager patient with history of cleft lip and palate treated with distraction osteogenesis with rigid external distractor (RED) device (left). Postoperative lateral cephalogram with RED device (right)

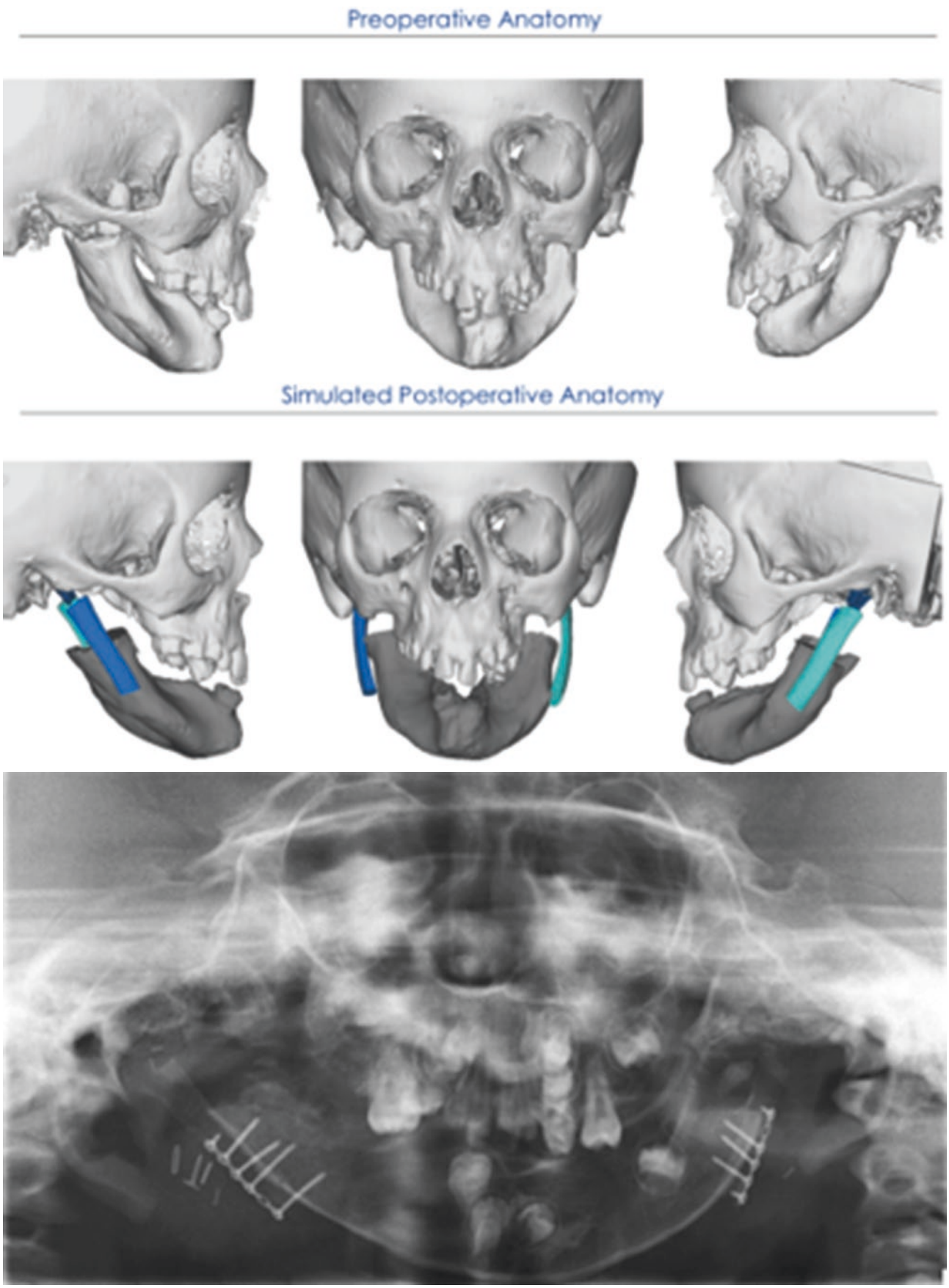

Fig. 19.3 Patient with Pierre Robin Sequence and severe mandibular hypoplasia treated with bilateral condylectomies and costochondral graft. Top picture shows virtual surgical planning and bottom pictures postoperative panoramic X-ray

or costochondral graft follow a similar protocol as has been described by Kaban et al. for the management of TMJ ankylosis in children, which includes the following:

1. Lining of joint with temporalis fascia
2. Rigid fixation
3. Early mobilization of jaw
   (a) if DO is used to reconstruct, mobilize day of surgery
   (b) if CCG is used, early mobilization with minimal maxillomandibular fixation (not to exceed 10 days)
4. Aggressive physiotherapy

Among the many therapies offered for OSA, maxillomandibular advancement (MMA) is recognized as a powerful technique for relieving upper airway obstruction. This orthognathic surgical procedure generally involves LeFort I maxillary and sagittal split mandibular osteotomies (see Fig. 19.4). It is possible that in the craniofacial patient, a sagittal split osteotomy would be insufficient to allow for the large mandibular discrepancy that can oftentimes be greater than 1 cm. In these patients, extra-oral inverted L osteotomies with bone graft augmentation may be necessary (see Fig. 19.5).

MMA surgery will simultaneously enlarge the pharyngeal airway dimension at the nasopharynx, oropharynx, and hypopharynx. The facial skeletal framework is expanded, with beneficial airway effects based on improved positioning of the pharyngeal soft tissues and the tongue. In addition to maxillomandibular advancement, an advancement genioplasty can be performed with a simple anterior mandibular horizontal osteotomy (AMHO) and this technique will allow to advance the genioglossus attachment further opening the airway (see Fig. 19.6).

However, genioglossus advancement may compromise facial aesthetics by over-projecting the chin prominence. A modification of the AMHO genioplasty was described by Heggie et al. with a design involving a rotational repositioning that allows for advancement of the genioglossus attachments while avoiding excessive projection of pogonion.

In the genial tubercle advancement, a rectangular bone window of the anterior mandible that incorporates the genial tubercle is osteotomized. The bone fragment is advanced, rotated 90 degrees, and stabilized. The bony advancement stretches the genioglossus muscle and addresses hypopharyngeal soft tissue obstruction. During this operation, the chin point is not changed, and it can be used in patients with an orthognathic profile without a negative impact on facial aesthetics.

Surgical treatment planning for correction of OSA in patients with craniofacial syndromes will often be a component of a multimodality approach.

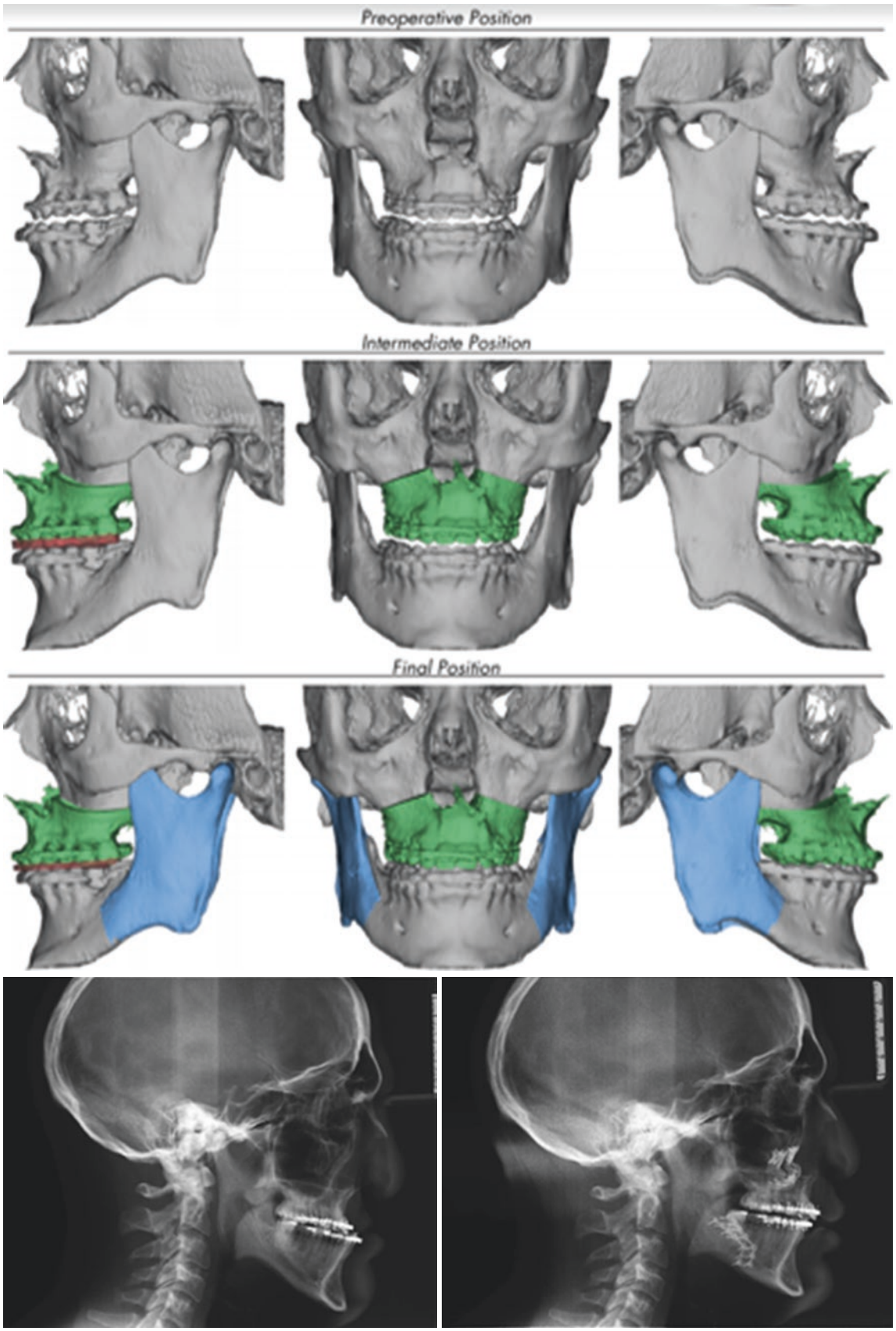

Fig. 19.4 MMA case in patient with history of cleft lip and palate. Virtual surgical planning with predictive movements (top). Preoperative lateral cephalogram (lower left) shows the anteroposterior deficiency of the maxilla. Postoperative lateral cephalogram with MMA and hardware in place

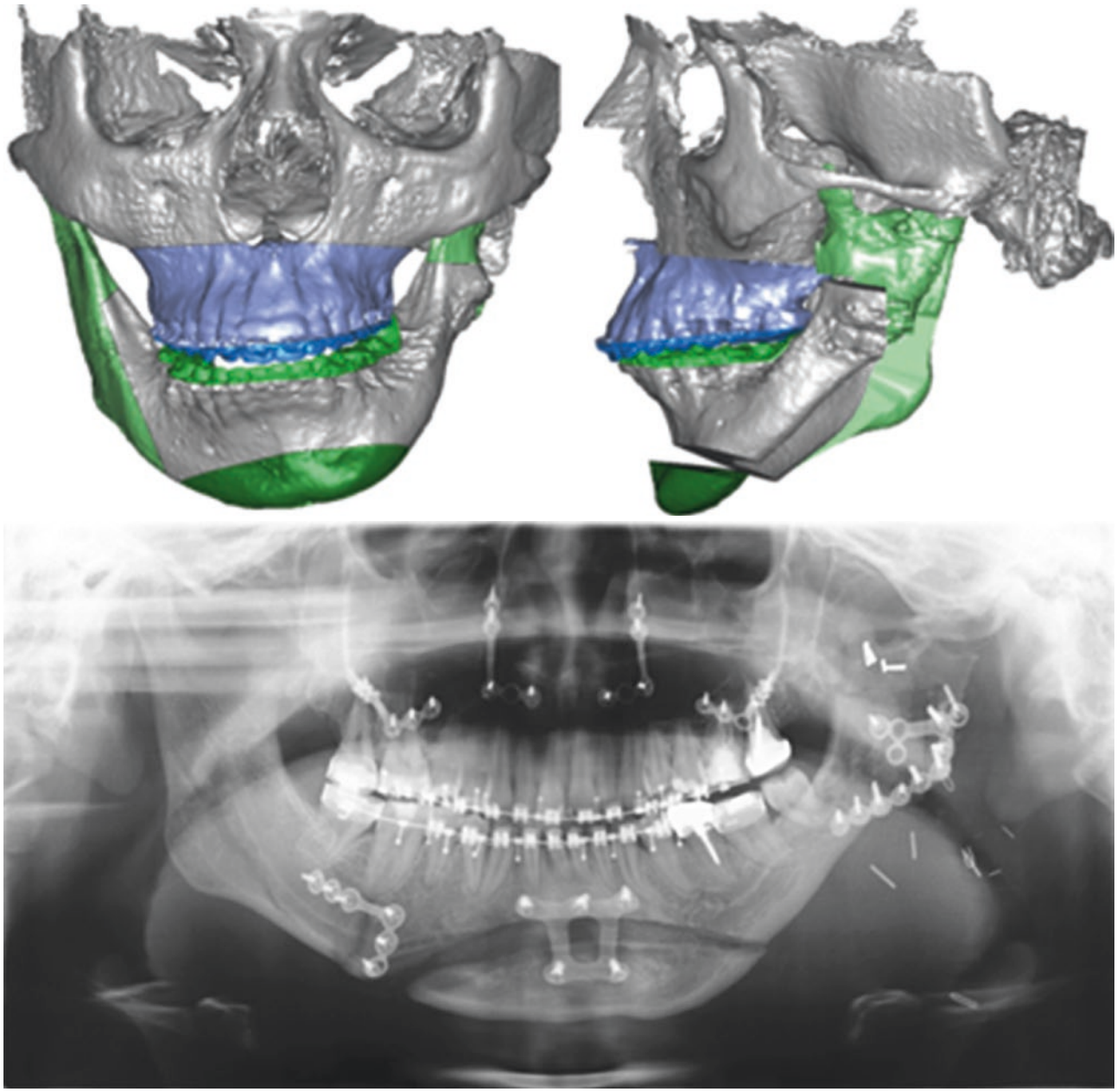

Fig. 19.5 Severe facial asymmetry case. Virtual surgical planning for correction of facial asymmetry with LeFort I osteotomy, right BSSO and left inverted L osteotomy (top). Postsurgical panoramic X-ray with asymmetry corrected (bottom)

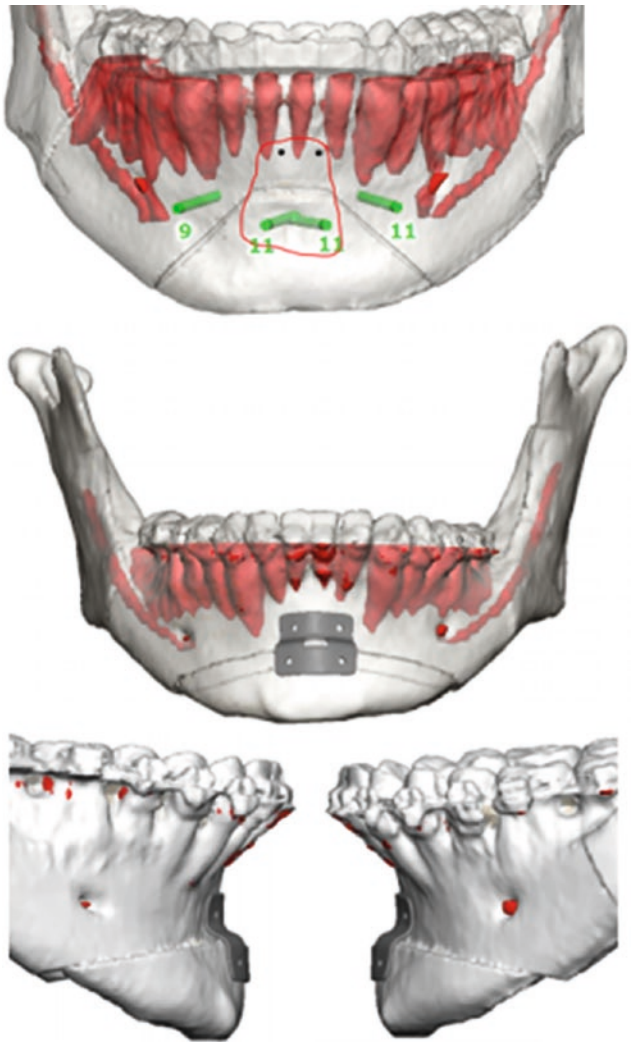

Fig. 19.6 Anterior mandibular osteotomy with the aid of virtual planning the exact location of the genial tubercles. This allows to plan the osteotomies incorporating the muscle attachment for advancement and the fabrication of patient-specific plates for rigid fixation

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Miniscrew-Assisted Maxillary Expansion Techniques for Treatment of Obstructive Sleep Apnea

Audrey Jung-Sun Yoon, Stanley Yung-Chuan Liu, and Christian Guilleminault

Contents

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20.1 Introduction

An important element of obstructive sleep apnea (OSA) pathophysiology involves risk factors created by the maxillofacial and oropharyngeal anatomy [1]. Maxillary constriction is associated with alterations in tongue posture resulting in retroglossal airway narrowing which is one of the defining characteristics of OSA [1]. Maxillary constriction with a high arch palate appears to be one of reasons for high nasal airway resistance and this transverse deficiency of the maxilla is associated as a potential contributor to the development of OSA [2].

In the pediatric population with patent sutural junctions, rapid maxillary expansion (RME) corrects the transverse discrepancy of the hypoplastic maxilla by bony expansion along the mid-palatal and circummaxillary sutures. Overall, there is an increased nasal cavity volume and resultant decrease in nasal airflow resistance [3]. Expansion also widens the distance between dilator muscles of the upper airway. The soft tissue sequela of maxillary expansion is allowing the tongue to protrude forward and upward, thus expanding the posterior pharyngeal airway space [4]. Therefore, RME is an effective treatment for OSA in patients with maxillary constriction [5]. In a 12-year follow-up study, Pirelli has confirmed stable, long-term results post-RME treatment for pediatric OSA [6].

In children, maxillary widening often does not require surgical intervention since the mid-palatal suture is still patent; the maxilla is widened simply by placing a RME appliance in the palate anchored to the teeth. Biologically, fusion of the mid-palatal suture occurs during the early teens and often coincides with the pubertal growth spurt [7]. Any attempts to expand the maxilla after the sutures have fused often incorporate surgical osteotomy to facilitate expansion. Even so, the expanders still exert lateralizing forces on the dentition, and expansion forces tend to concentrate more on the supporting dental segments rather than the mid-palate or nasal floor. In 1964, a study by Krebs on orthopedic transverse modification demonstrated that expansion is 50% skeletal and 50% dental in younger children while the percentage dramatically changes to 35% skeletal and 65% dental in adolescents [8]. Over the years, there have been many RME design modifications to minimize dental side effects while maximizing skeletal expansion.

With the introduction of temporary skeletal attachments, bone miniscrews can be placed in the maxilla to secure a RME appliance such that it can directly apply forces to the maxilla, effectively bypassing teeth as anchor units. This creates new avenues of maxillary expansion even if no teeth are present and avoids any undesirable tooth movement. This new group of miniscrew-anchored maxillary expanders can attain greater physiologic suture expansion, minimize negative dentoalveolar iatrogenic effects, and achieve maximum nasal expansion as compared to conventional RME [9, 10].

Even with creative efforts to transfer lateral forces of RME expansion screw directly to the maxillary bone, the success of suture split and skeletal expansion without osteotomy is not always predictable in the adult OSA population. To increase the success of maxillary skeletal expansion, Yoon, Liu, and Guilleminault have developed the "Distraction Osteogenesis Maxillary Expansion (DOME) protocol for adult OSA patients which integrates minimally invasive osteotomies with miniscrew-anchored maxillary RME [11]. This establishes much more predictable skeletal expansion results and more importantly reliable OSA improvements [12].

In this chapter, various miniscrew-anchored RME expansion designs and techniques for the treatment of OSA are introduced with or without corticotomy in order to achieve maximum improvement of OSA.

20.2 Different Designs of Miniscrew-Assisted/Bone-Anchored Rapid Maxillary Expanders

There are numerous designs of bone-anchored expanders that can be applied in OSA maxillary expansion cases: bar type, multiple miniscrew-supported expander, acrylic supporting design, hybrid design, customized "specific to patient" design (Fig. 20.1).

The advantages and disadvantages of each design type are listed in Table 20.1.

There are reports that some of these expanders are sufficient to overcome sutural tension without surgery and can be a practical alternative to surgically assisted rapid palatal expansion in specific cases especially for teenagers and young adults. However, the most reliable and effective technique is still introducing site-specific osteotomies with a bone-anchored expander to minimize iatrogenic dental and periodontal damage in the older adult OSA population [13, 14].

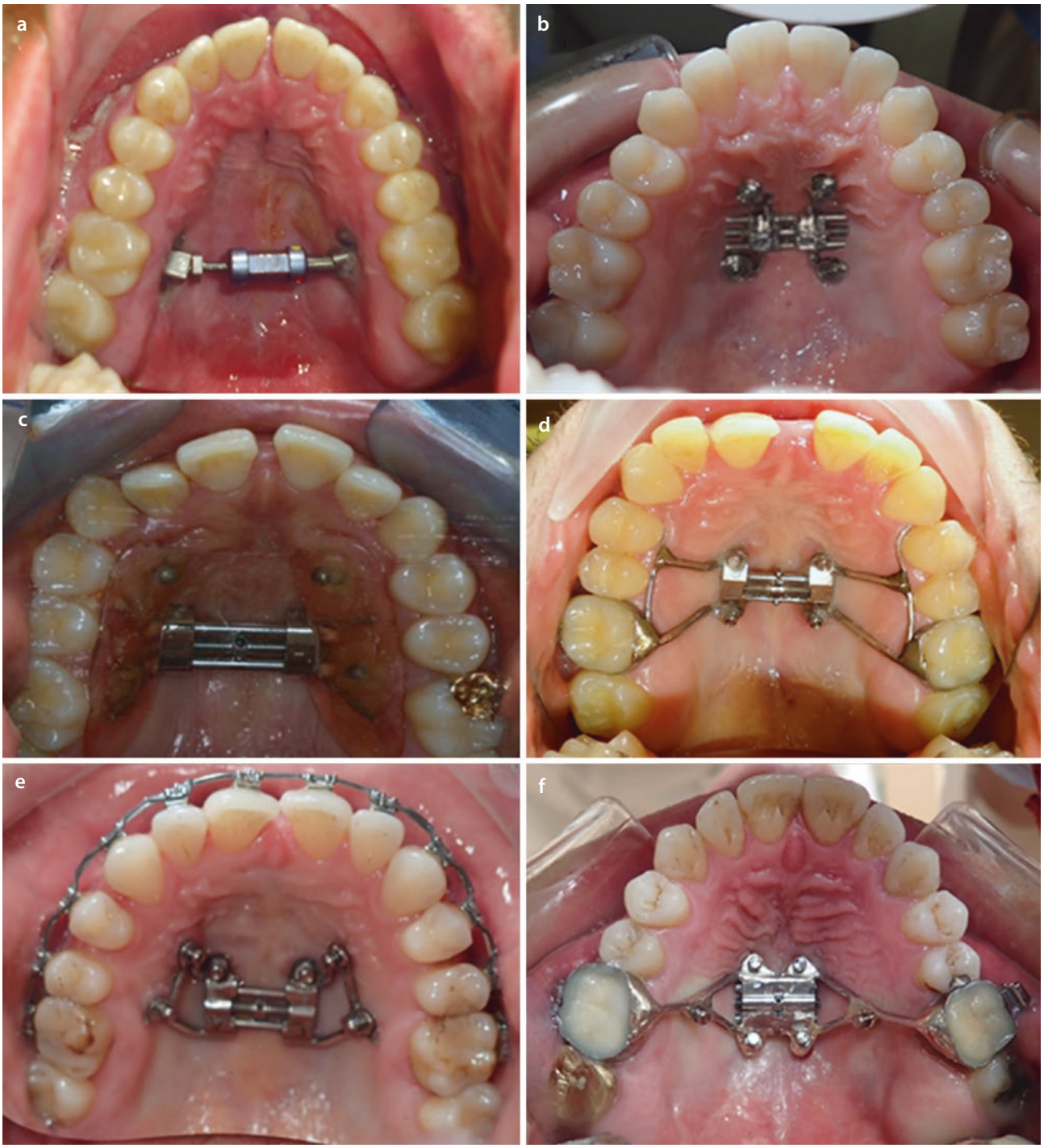

Fig. 20.1 Different types of miniscrew-assisted maxillary expander. **a** Bar type. **b** Multiple miniscrew-supported expander. **c** Acrylic supporting design. **d** Hybrid design (combination of tooth- and bone-anchored expander). **e** & **f** Customized designs; any custom combination of numbers of bone screws, location of screws, and different design of jackscrews specific to the shape and thickness of palate

20.3 Installation of Miniscrew-Assisted Maxillary Expanders

The surgeon usually places the miniscrews or certain types of expanders such as the bar-type expander (Fig. 20.2) [15].

The RME device is traditionally placed by the orthodontist prior to surgery. Different designs have varying protocols for installation depending on the type of expander. The following are examples of designs which are usually placed by orthodontist (Figs. 20.3 and 20.4).

Table 20.1 The advantages and disadvantages of each design type of miniscrew-assisted maxillary expanders

Design	Advantage	Disadvantage
Bar type – *KLS Martin RPE* – *TPD (Transpalatal distractor)*	– Pre-fabricated design – Same day appointment process (no lab work necessary) – Can be inserted in areas with very narrow palate	– Technique-sensitive: Screw needs to be placed at a non-favorable angle – Difficult to position – must be parallel to expansion vector to avoid asymmetric expansion – Only single point contact on each side resulting in more concentrated forces during expansion
Multiple miniscrew-supported expander	– Can choose design with miniscrew anchors on the side of palate or secured on roof of palate – Can be custom-made to individual – No teeth involvement to avoid any dental periodontal damage	– High cost of fabrication – High cost of components – Multiple piece fabrication – Process requires multiple appointments to complete
Acrylic base reinforced with four miniscrews – *C-expander*	– Cost-effective – Fabricated in-house – Easy refabrication for second expander – Can be custom-made to individual – Able to insert very narrow palate – No teeth involvement to avoid any dental periodontal damage	– Technique-sensitive – Difficult to remove miniscrews after use – Potential for food trap, especially surface area junction between acrylic and contact with surface of palatal tissue – Multiple appointment process
Hybrid design: 2 molar bands + 2 miniscrews – *Hybrid hyrax (Ludwig Design)*	– Easy to insert and remove miniscrews – Reduced inventory – Can be custom-made to individual – Easy refabrication for second expander – High success rate of mid-palatal suture splits on young adults	– High cost of fabrication – High cost of components – Multiple piece fabrication – Multiple appointment process – Too bulky to insert into very narrow palate
Hybrid design: 2 molar bands + 4 miniscrews – *MSE (maxillary skeletal expander)*	– Pre-fabricated design of jackscrew including miniscrew insertion holes – Easy to insert and remove miniscrews – Simple technique – High success rate of mid-palatal suture splits on young adults	– High cost of fabrication – High cost of component – Multiple appointment process – Jackscrews are too big for very narrow palate – Location of mini-screw insertion into bone are limited and dictated by the pre-fabricated holes of expander – Once miniscrew anchorage fails, it is hard to change the location – Miniscrews often penetrate through nasal cavity (oroantral fistula)

20.4 Distraction Osteogenesis Maxillary Expansion (DOME) Protocol

20.4.1 Custom Design and Installation of Expanders

All pre-fabricated miniscrew-retained expanders have an inherent limitation of where the insertion points of the miniscrews can be located. Optimizing the placement of one miniscrew may compromise the other miniscrews due to irregular bone thickness and density throughout

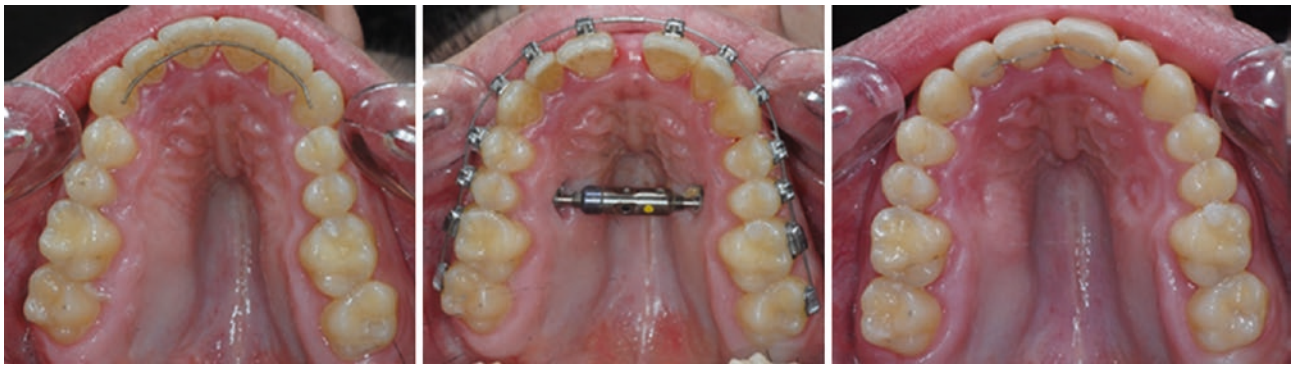

Fig. 20.2 Bar-type expander: transpalatal distractor (TPD). (*Courtesy of Dr. Bart Vande Vannet*)

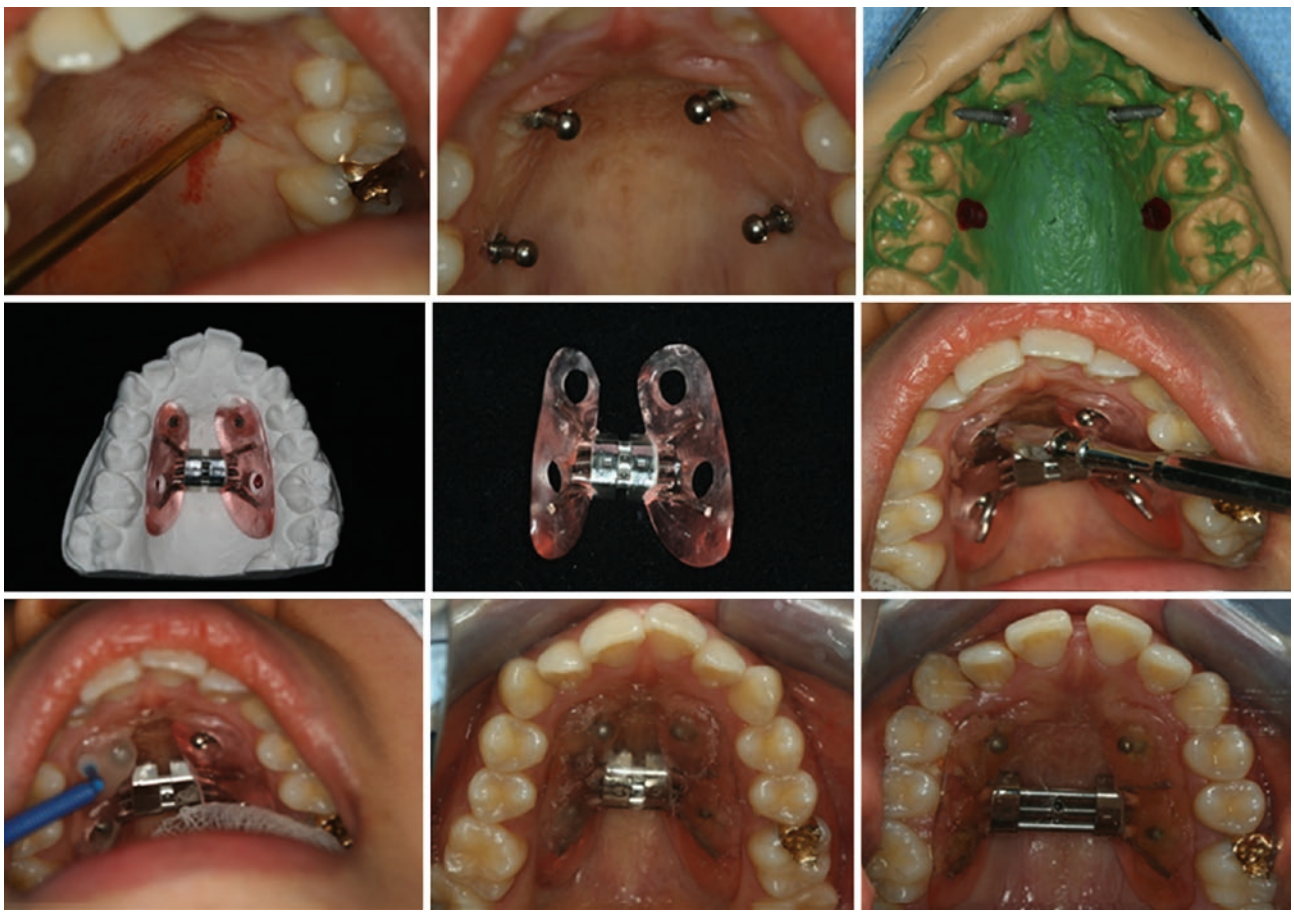

Fig. 20.3 Fabrication, insertion of acrylic-base expander (C-implant), and expansion result. (*Courtesy of Dr. Seong-Hun Kim*). Suture separation was achieved without osteotomy at patient's age of 17

the palate. To increase the success rate of expansion and decrease potential complications (e.g., miniscrew loosening, miniscrew perforation through the maxillary sinus, etc.), the anatomic structures (especially bone thickness at various locations of the palate) should be carefully mapped using 3-D cone-beam computed tomography (CBCT) imaging to generate a custom-fabricated expander design. The density and thickness of palatal bone information gained from CBCT data provide information to identify optimal positions for placement of screws, deciding on the proper length of screws to use, mapping suture location, and evaluating status of sutural fusion. Recommended placement of miniscrews would be as close to the mid-palatal suture as possible with the caveat that sufficient bone thickness needs to be present. Also, bicortical engagement of the

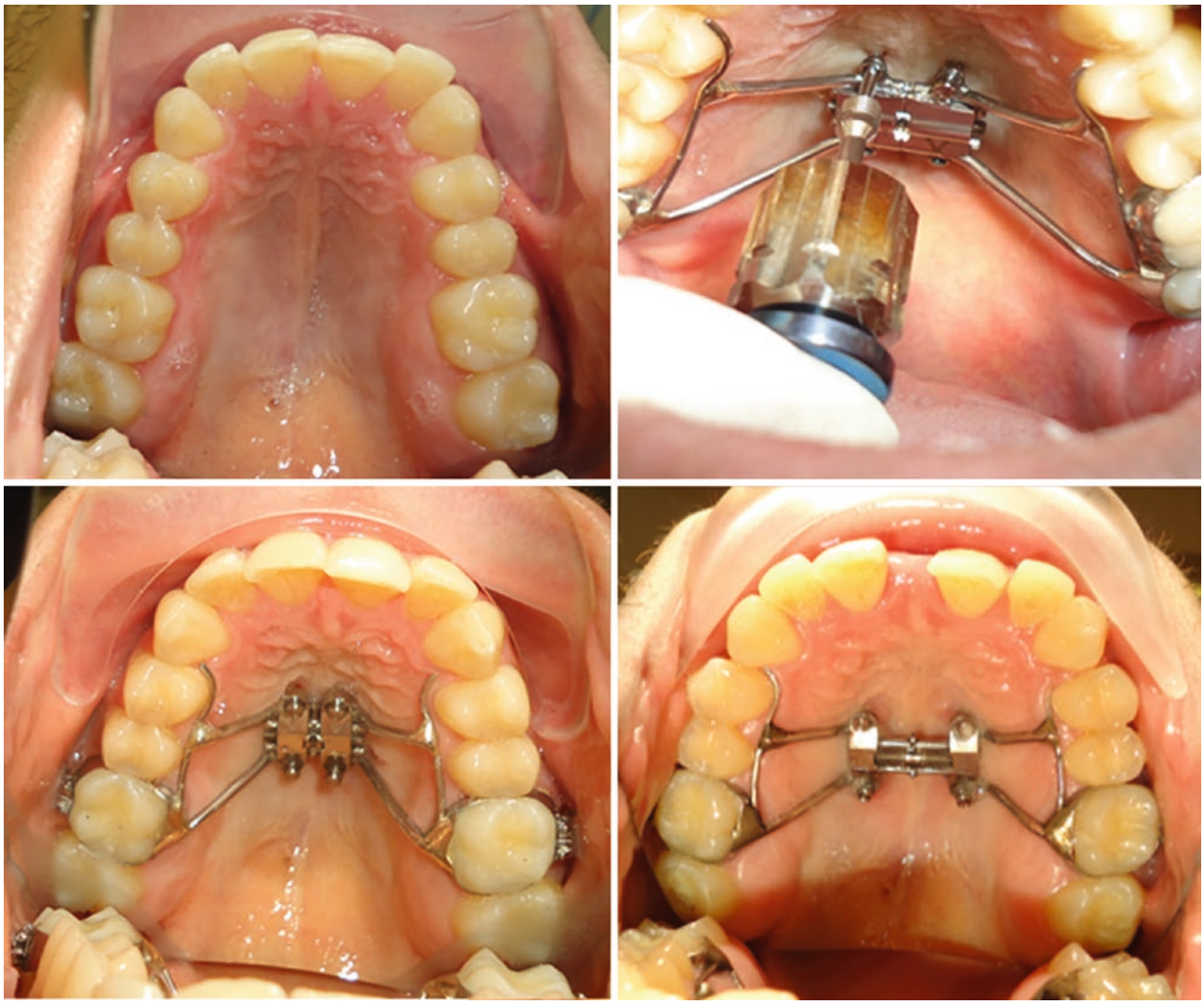

Fig. 20.4 Installation of a hybrid-design expander (MSE) and expansion result. Suture separation was achieved without osteotomy at patient's age of 16

palatal roof should be ideal [16] using an optimal screw length as measured on CT so as not to create an oroantral fistula and avoid root damage. The orthodontist and surgeon need to work closely and plan together (Fig. 20.5).

20.4.2 Surgical Technique (DOME)

DOME begins with a limited osteotomy at the Le-Fort I level without down-fracturing. Additional auxiliary osteotomy at the midline of the maxilla may also be performed using a piezo-electric saw and wedge. As the suture opens, a dental central diastema develops immediately (Fig. 20.6). The expander screw is turned to verify that screw threads are intact; symmetric and easy separation of the maxilla bilaterally should be observed. Patients with less severe OSA can be discharged on the day of surgery, while individuals presenting with more severe OSA should be monitored overnight as a safety precaution. The patient may resume a regular diet within a week.

20.4.3 Activation of Expander and Orthodontic Treatment

The expander device is activated by turning some type of axial screw, usually at an expansion rate of 0.125–0.25 mm per day. On average, a total of 8–12 mm maxil-

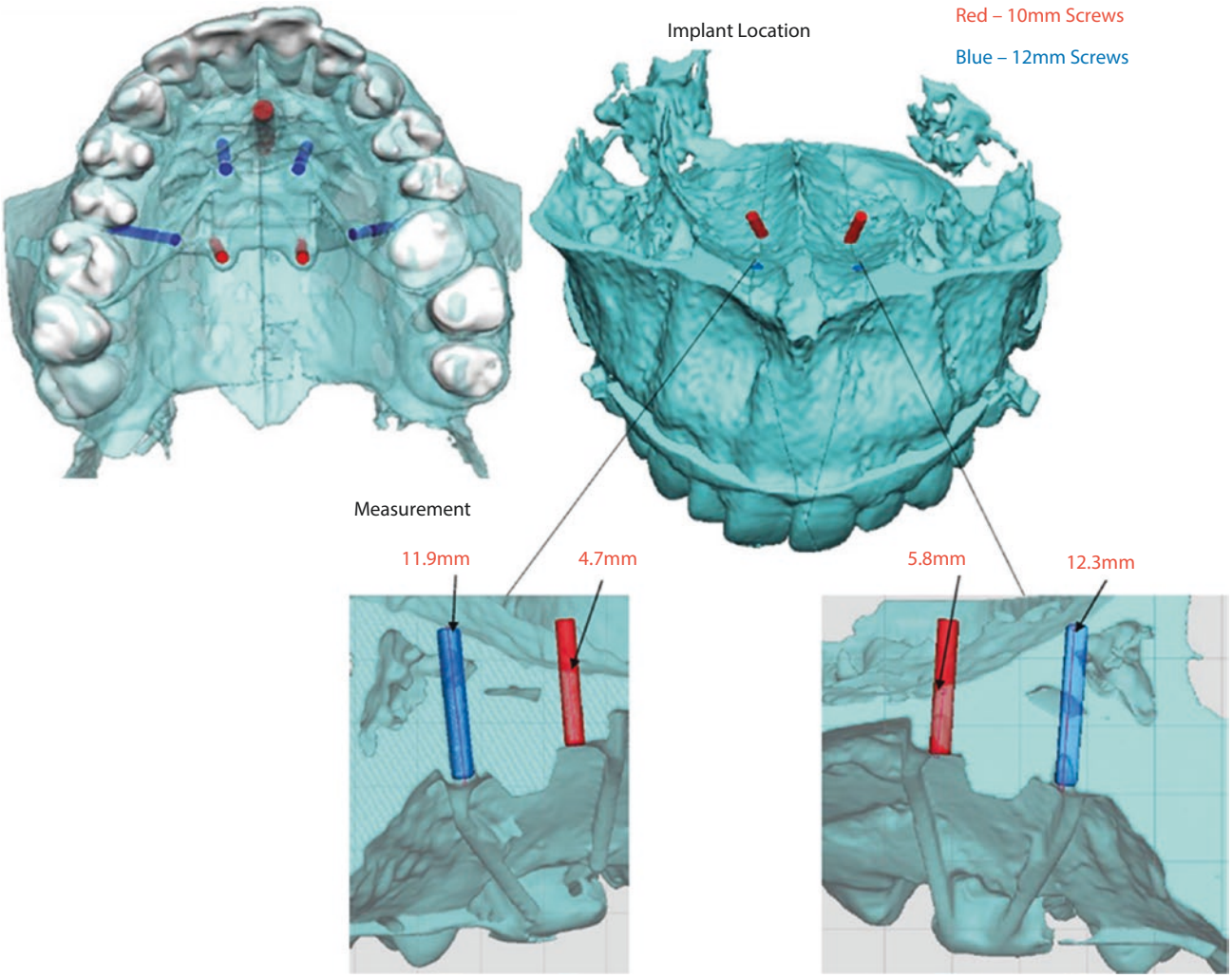

Fig. 20.5 DOME virtual planning. Using 3-D technology, custom-fabricated miniscrew-assisted maxillary expander is designed and ideal sites and length of miniscrews are identified to achieve optimal results and minimize side effects and failure for OSA

lary expansion should be achieved to resolve obstructive sleep apnea medical conditions. Once the planned expansion is completed, orthodontic treatment is initiated to close the existing diastema and expand lower arch to achieve normal occlusion.

20.4.4 Consolidation Phase

Typically, the consolidation phase is 3 months [15, 17, 18] for typical craniofacial distraction osteogenesis, but the ideal recommended consolidation period is 6–8 months in order to allow maximal bone fill and minimize relapse. The miniscrew-assisted rapid palatal expander technique does not interfere with tooth movement and allows the expander to remain in place while the orthodontist guides the teeth into proper position to correct the occlusion. Longer consolidation period with the device in place increases greater long-term stability of the skeletal expansion.

20.4.5 Determining the Amount of Expansion

Defining the amount of necessary skeletal expansion for improvement of OSA has not yet established. Typical orthodontic measurements are based on arch width differences between maxillary and mandibular intermolar width. However, in order to achieve the greatest possible skeletal maxillary expansion for OSA improvement, the most important areas of consider-

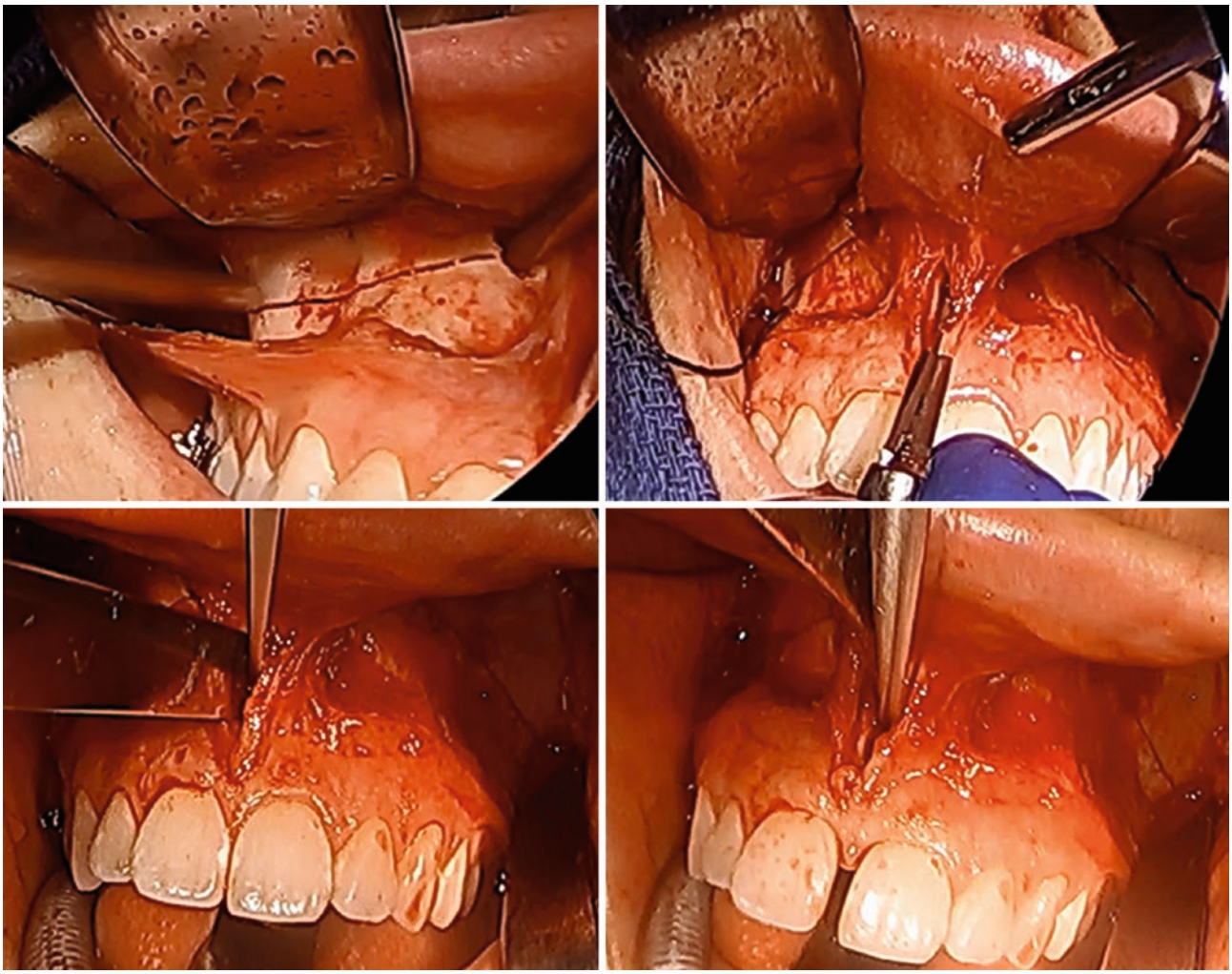

Fig. 20.6 Limited Le-Fort I and mid-sagittal split without down-fracture. (Courtesy of Dr. Stanley Liu)

ation are the width of nasal floor and palatal floor. Depending on location of screws and application of forces, dentoalveolar response and teeth angulation changes after expansion are quite different than conventional tooth-anchored expander. For example, if you use only miniscrews on floor of palate close to midpalatal sutures, palatal crown tipping of molars will occur after expansion, which is the opposite phenomenon to tooth-anchored maxillary expander. Therefore, the design of expander, location of screws, and basal bone width and angulation all need to be considered. The orthodontist needs to evaluate the skeletal and dental relationships in the transverse plane using dental casts and/or coronal cross-section views of 3-D images.

In many cases of constricted maxilla, the compensated lower teeth are more lingually inclined, camouflaging the maxillary constriction. Uprighting the posterior teeth to a normal inclination over the supporting basal alveolar bone needs to be calculated for both maxillary and mandibular dentition. In some cases, uprighting of the lower posterior teeth first (i.e., mandibular dental decompensation) can be helpful.

20.4.6 Retention and Relapse

Following the active phase of any expansion, a retainer is needed even after bone fill seems complete. The expansion must be maintained passively by fixed or removable appliance to aid in transverse retention.

20.5 Case Result of DOME

Figures 20.7, 20.8, 20.9, and 20.10 illustrate pre-DOME and post-DOME results. For most patients using a proper DOME technique, 8–9 mm expansion of

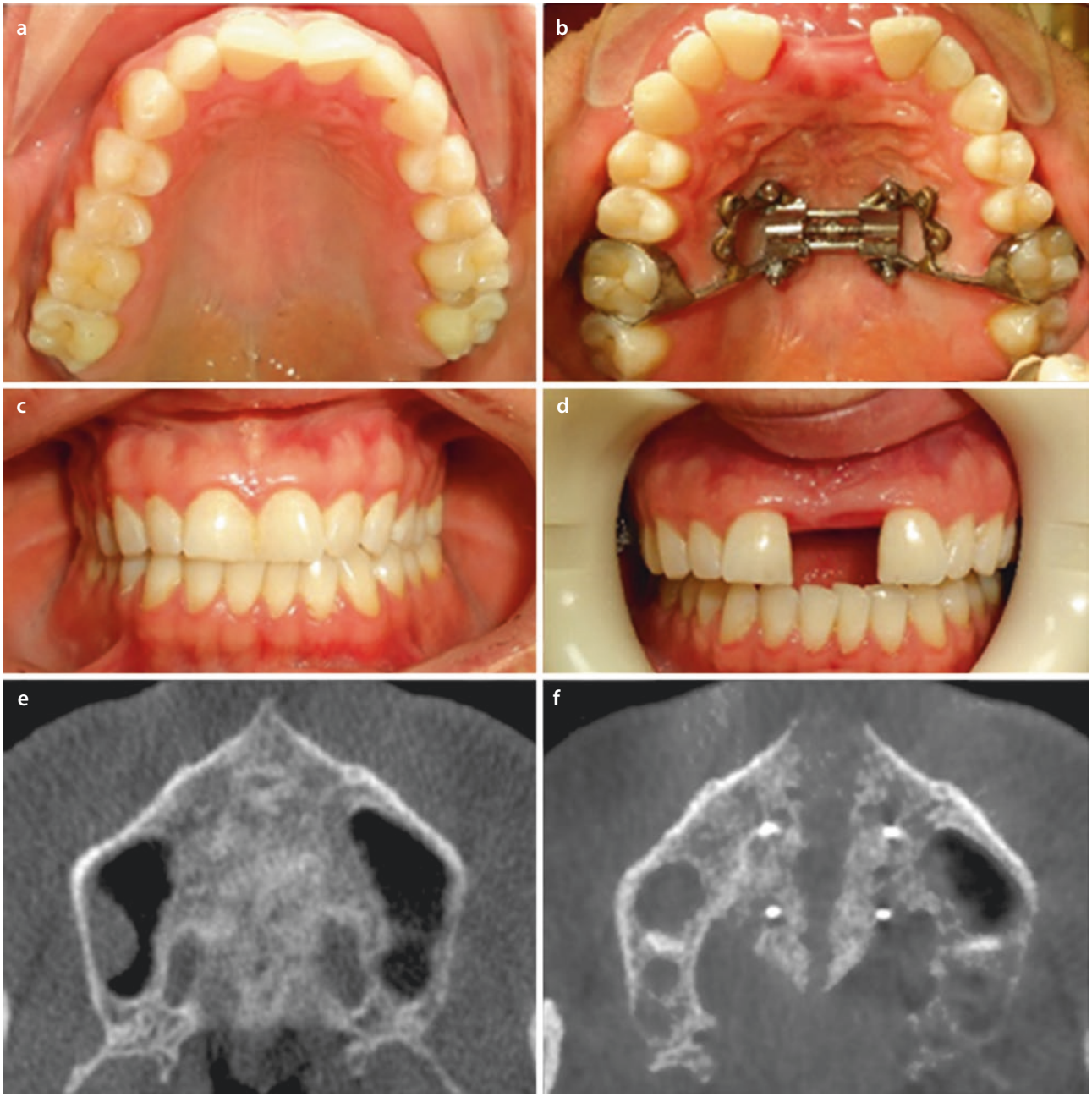

Fig. 20.7 Pre-DOME (left) and post-DOME (right). **a** Pre-DOME occlusal view. **b** Post-DOME occlusal view. 14 mm diastema presented after 9 mm of jackscrew expansion. **c** Pre-DOME frontal view **d** Post-DOME frontal view. Note 14 mm diastema **e** Pre-DOME transverse view of palate of CBCT f) Post-DOME transverse view of palate of CBCT. Note 12 mm expansion at anterior nasal spine, 4.5 mm expansion at posterior nasal spine after 9 mm of transpalatal expansion at jackscrew level

the appliance jackscrew equates to approximately 10–14 mm dental diastema present. This patient presented 14 mm diastema, 12 mm expansion at anterior nasal spine, 4.5 mm expansion at posterior nasal spine, and 8 mm expansion on nasal floor after 9 mm of transpalatal expansion at jackscrew level. Internasal width, intermolar width, and internal nasal valve are all significantly increased after DOME. This patient's Apnea Hypopnea Index dropped from 13.8 to 4, Nasal Obstruction Symptom Evaluation Scale dropped from 17 to 3, and Epworth Sleepiness Scale improved from 23 to 6 after DOME procedure.

20.6 Discussion

There are many studies that show maxillary expansion as an effective treatment modality for OSA in children [5, 6, 19]; however, there are very limited published data available for adult OSA population [20].

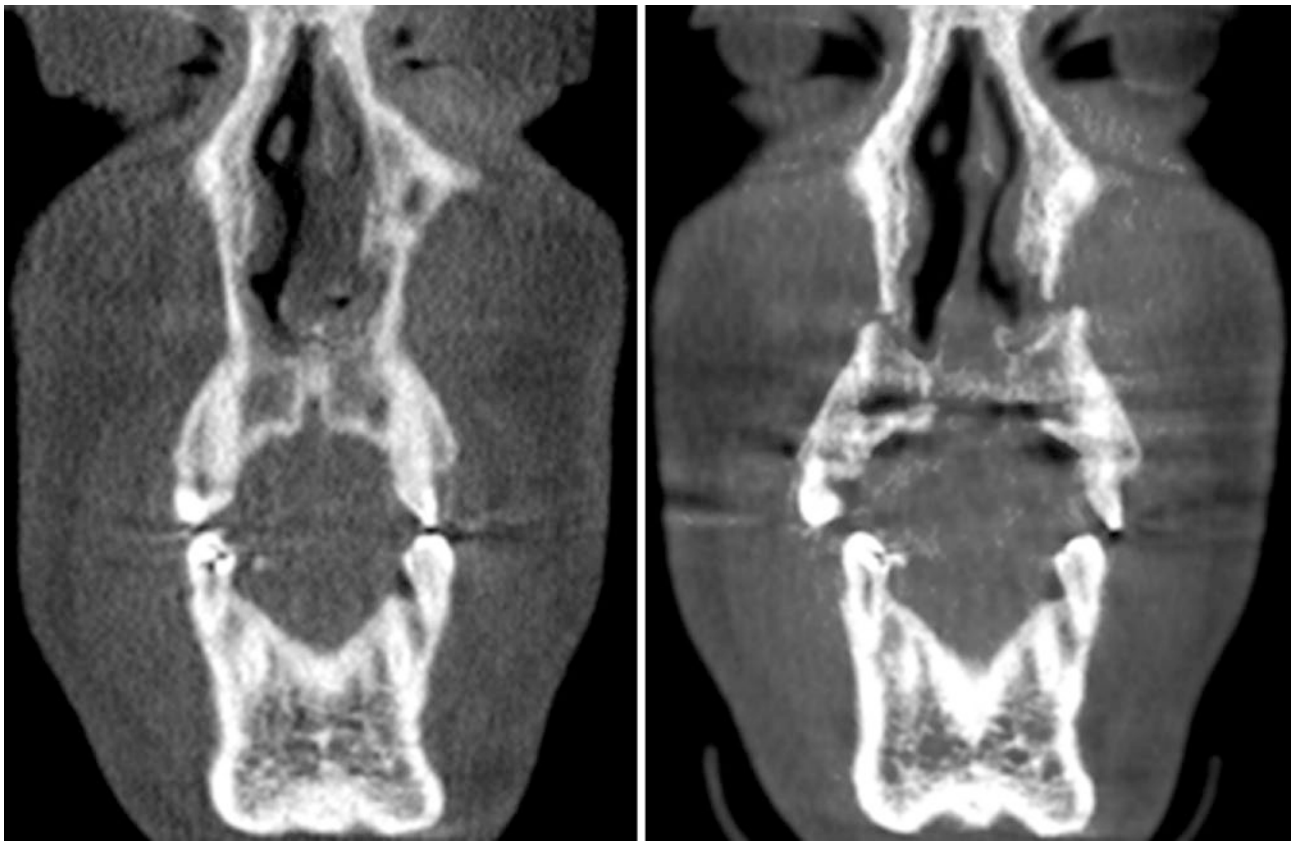

Fig. 20.8 Pre-DOME (left) and post-DOME (right): coronal view at nasopalatine canal level. Nasal floor was expanded 8 mm and nasal cavity became more patent after expansion

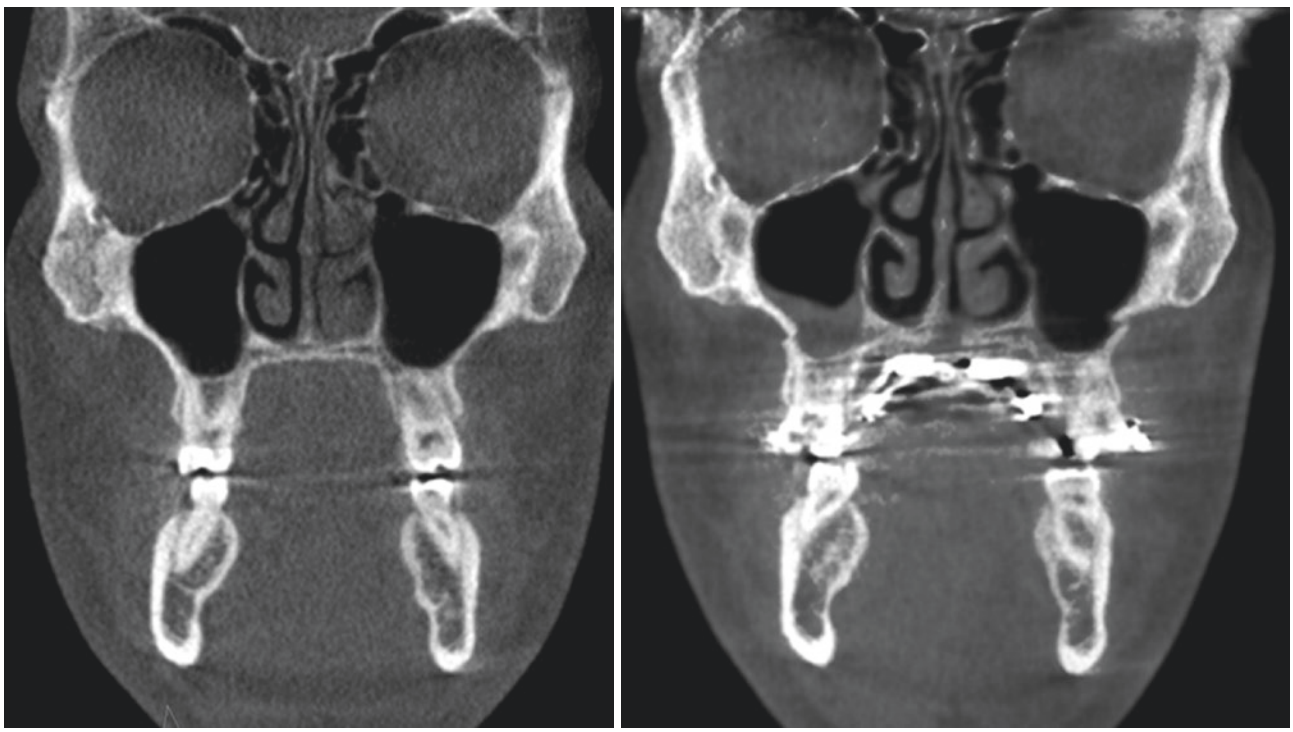

Fig. 20.9 Pre-DOME (left) and post-DOME (right): coronal view at palatal cusp of first molar level. Nasal floor was expanded 6 mm and nasal cavity become more patent after expansion

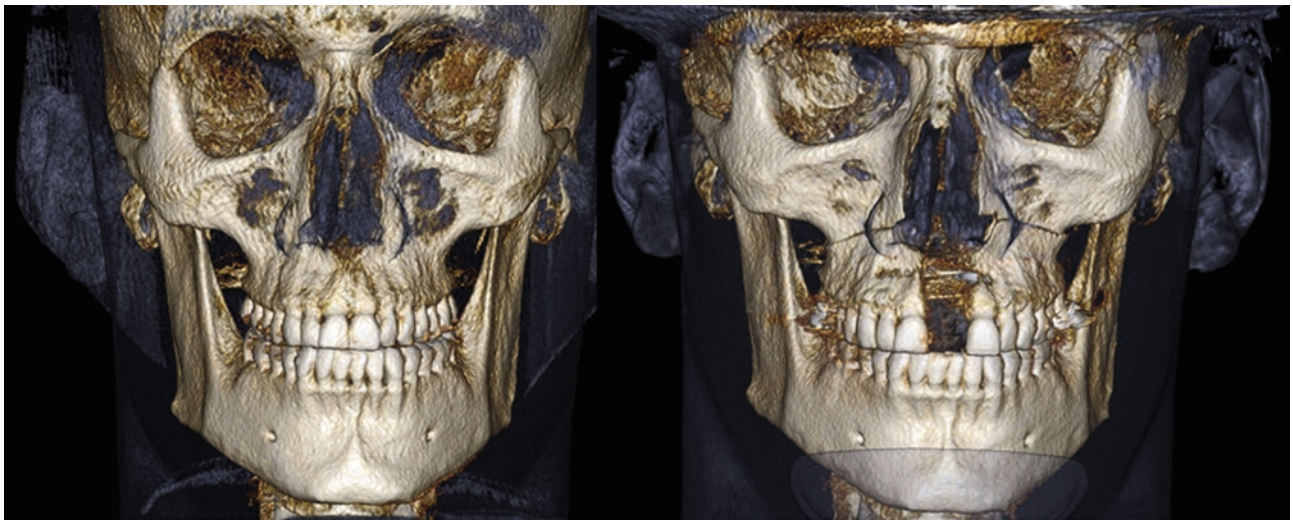

Fig. 20.10 Pre-DOME (left) and post-DOME (right): 3-D surface rendering from CBCT

Orthodontic miniscrews, which are used for absolute orthodontic anchorage, have been incorporated into bone-anchored maxillary expander designs (i.e., fixed maxillary expander attached directly to palatal bone using miniscrews) and clinical research has attempted to validate the theory that direct transfer of expansion energy to the palatal bone should result in greater skeletal expansion rather than alveolar bone bending [9]. Miniscrew-assisted palatal expanders allow for greater physiologic suture expansion, reduces negative dentoalveolar effects, achieves the maximum nasal and oral cavity volume compared to conventional RME [9, 10], and contribute to more predictable stable management of OSA. Recently, randomized-controlled trial showed that significantly higher post-expansion nasal airflow values for bone-anchored maxillary expander (hybrid type, average age 10.2 years) compared with tooth-anchored expander (average age 9.7 years) [21]. Many studies have demonstrated that miniscrew-assisted maxillary expander can be a better treatment option than conventional tooth-anchored maxillary expander for increasing skeletal expansion and airway volume without osteotomy but these patient's age were mostly for late teenagers [22] and has not been studied yet in the OSA patient pool.

The separation of sutures using DOME becomes much more predictable and reliable in adult OSA patients, thus the author advocates the continued augmentation of minimal osteotomy during maxillary expansion using miniscrew-assisted RME appliances for older population of OSA. It still remains to be determined whether mandibular expansion is possible although there are some individual case reports [23]. Skeleton-borne maxillary expansion using palatal miniscrews offers a new treatment alternative for a multidisciplinary approach to adult sleep apnea syndrome.

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Orthognathic Surgical Considerations for Obstructive Sleep Apnea

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Contents

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In 1978, Bell and Epker [1] recognized that preoperative orthodontic treatment helps improve the outcome of orthognathic surgery. Bell et al. [2, 3], Epker and Fish [4], and Profitt and White [5] all concluded improved stability and outcomes could be achieved via close cooperation between maxillofacial surgeons and orthodontists. Although orthognathic surgery was initially confined to the treatment of sagittal discrepancy before 1975, its scope gradually widened thereafter to include treatment of transverse discrepancy and various forms of skeletal discrepancies. In the 1990s, rigid fixation was generally used to achieve precise surgical results and to reduce patient discomfort (e.g., typically 6–8 weeks of intermaxillary fixation characterized by a liquid diet, the inability to brush teeth, and reported psychological complaints similar to claustrophobia).

In 1985, Wolford et al. [6] published the Surgical Treatment Objective (STO), which predicted outcome of orthognathic surgery. Profitt et al. [7, 8] constructed a treatment plan based on a hierarchy of stability of outcomes of orthognathic surgical procedures, rendering it possible to obtain a more stable outcome [9, 10]. In patients with severe skeletal discrepancy, skeletal improvements result in an improved aesthetic outcome with better functionality and stability. Prior to the advent of orthognathic surgery, practitioners attempted to resolve malocclusion using a compensatory treatment; however, the patient and the practitioner were less satisfied with the treatment outcome because of suboptimal aesthetic improvements. With the development of orthognathic surgical methods, it is possible to overcome the limitations of compensatory treatment and relatively easier to eliminate skeletal discrepancies. However, orthognathic surgery significantly changes the anteroposterior or vertical position of the maxilla or the mandible. This skeletal modification inevitably induces alterations in the soft tissue that may lead to changes in the upper airway space.

In the early 1950s, Drs. King [11] and Brodie [12] separately reported that the nasopharynx's anteroposterior size is nearly fully formed in the first and second years of life. In 1976, Handelman and Osborne [13] suggested the growth of the nasopharynx is complete at 18 years of age, but noted growth patterns differ according to sex. In adults, structural changes do not occur in the upper airway space after maturation and there is no structural change in the airway space except for specific pathological conditions or a long-term aging effect [14, 15]. Clinicians should consider these inevitable changes in the airway space when performing orthognathic surgery [16].

It is well known that the upper airway space and skeletal movement of the maxilla and mandible interact closely with each other [17]. Therefore, orthognathic surgery, including maxillomandibular advancement (MMA), is one of the effective treatments for obstructive sleep apnea (OSA) in severe skeletal Class II patients with sleep apnea. MMA is a relatively straightforward intervention for Caucasian patients who have a large nose and a retruded mandible, whereas the procedure is more difficult to perform for Asian patients who have a small nose and flat facial profile. Because orthognathic surgery improves the upper airway space and causes aesthetic changes, both race and facial pattern need to be considered.

It is also important to note that positional changes of the hyoid bone and tongue in concert with mandibular movement are also closely related to the spatial change in the upper airway [18, 19]. The upper airway space includes the nasal and oral cavity and consists of the nasopharynx, the posterior region of the nose, posterosuperior region of the soft palate, oropharynx, posterior region of the mouth and mandible, hypopharynx, and the third and fourth cervical vertebrae regions. The upper airway space is surrounded by hard tissues such as the maxilla, mandible, palatine bone, vomer, and cervical vertebrae. The muscles consist of the tongue and soft palate. The mucosa originates from the oral, nasal, and laryngopharyngeal cavity.

In mandibular prognathism and a skeletal class III malocclusion, mandibular setback is performed to resolve the skeletal discrepancy. However, space reduction may cause snoring and OSA in some patients [20].

In most studies that report change in the upper airway after mandibular setback, results consistently demonstrate that the upper airway space is reduced immediately after surgery. However, it remains controversial whether the reduced space recovers due to physiological adaptation [21, 22], remains reduced after the surgery [23–27], or continues to decline when observed after a certain period of time [28, 29].

Because orthognathic surgery inevitably changes the position of the skeleton, more accurate and stable results should be obtained by accurately analyzing functional characteristics of the upper airway space, soft palate, uvula, the position of the hyoid bone, as well as achieving the aesthetic goal set forth in planning [9].

21.1 Assessment of the Posterior Airway Space

Because the upper airway space cannot be directly visualized, it can be challenging to evaluate. Various imaging modalities have been used to evaluate the upper airway space, peripheral soft tissues, and skeletal structure, such as acoustic rhinometry, fluoroscopy, nasopharyngoscopy, magnetic resonance imaging (MRI), cephalometry, and tomography, among others [30]. Each method has its inherent advantages and disadvantages; thus, the selected method of imaging should be based on the goal of the assessment.

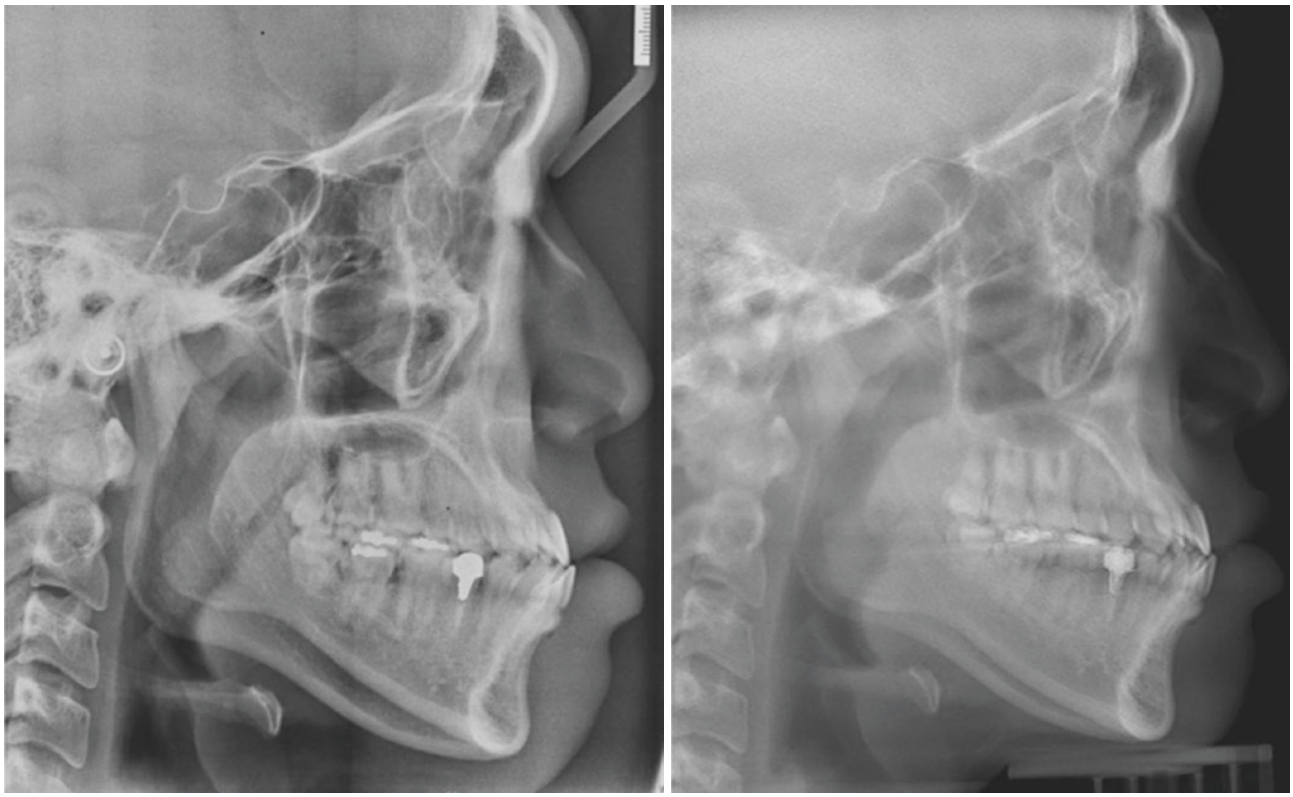


Fig. 21.1 Comparison of the lateral cephalogram and cephalogram extracted from cone beam computed tomography

The results from cephalometric radiography are commonly used as data for establishing the orthodontic treatment plan, whereas most studies of the upper airway employ cephalometric measurements. However, cephalometric radiography is obtained by projecting a three-dimensional (3D) structure in two dimensions (2D) and presents a disadvantage in accurately elucidating size and complexity of the upper airway. Various imaging modalities have been used to evaluate the upper airway space, peripheral soft tissues, and skeletal structure, such as acoustic rhinometry, fluoroscopy, nasopharyngoscopy, magnetic resonance imaging (MRI), cephalometry, and tomography, among others [30]. Each method has inherent advantages and disadvantages and the selection of imaging modality should be based on the goal of the analysis.

Cone-beam computed tomography (CBCT) has recently been used widely and can acquire the 3D volumes of all structures in the maxillofacial complexes. The 3D volume data can be reconstructed into a more detailed image by converting it into a multiplanar reconstruction image using commercially available 3D imaging software, which facilitates measurements of not only the soft tissue and the upper airway space, but also the skeletal structure, all in 3D [24].

The 3D raw image data reconstruction allows visualization of a multilayered cross-section, and this 2D image of the pharynx can be evaluated in all directions (most common of which are sagittal, coronal, and axial; see Fig. 21.1). Various commercially available imaging software programs are capable of observing the upper airway space from various angles. Unlike hard and soft tissues, the void space of the upper airway allows for a sharper and clearer spatial analysis. Specific tools can be used to distinguish tissues of different densities. Software capable of using transparency allows observation of the hard tissue covered by the soft tissues. A linear measurement tool is also available, allowing measurement of height, width, and depth of the entire pharynx (Fig. 21.2).

Owing to variation in the conditions at the time of image acquisition, images obtained from the CBCT are not always acquired using a consistent head position. Therefore, the patient's 3D image needs to be realigned with the reference plane to facilitate image analysis (a process similar to that in lateral cephalometric image analysis). This means the Frankfort horizontal plane should be parallel to the axial plane and the midsagittal plane should coincide with the patient's midline, and, in the same way, the coronal plane should contact the lower margins of the orbit (Figs. 21.3 and 21.4). If an asymmetry is detected, the reorientation process should be carefully performed. This virtual position allows for appropriate head rotation, which helps to

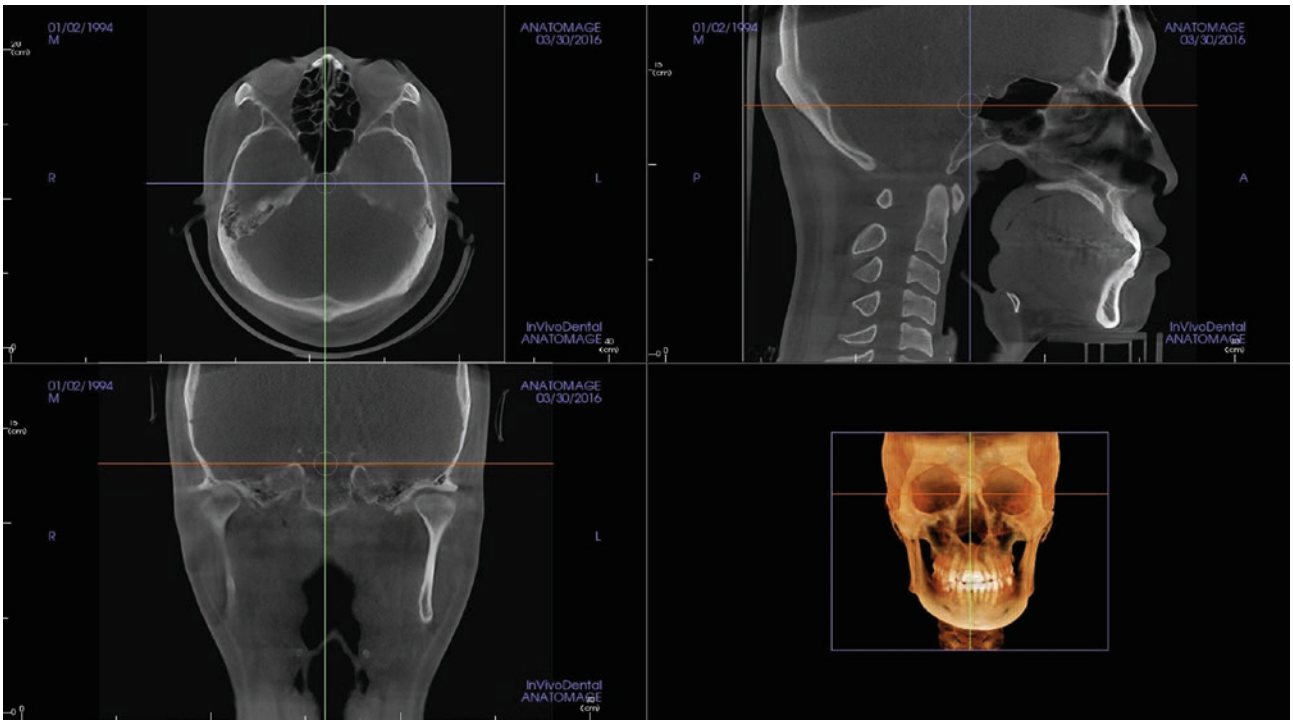


Fig. 21.2 3D image to multiplanar reconstruction image

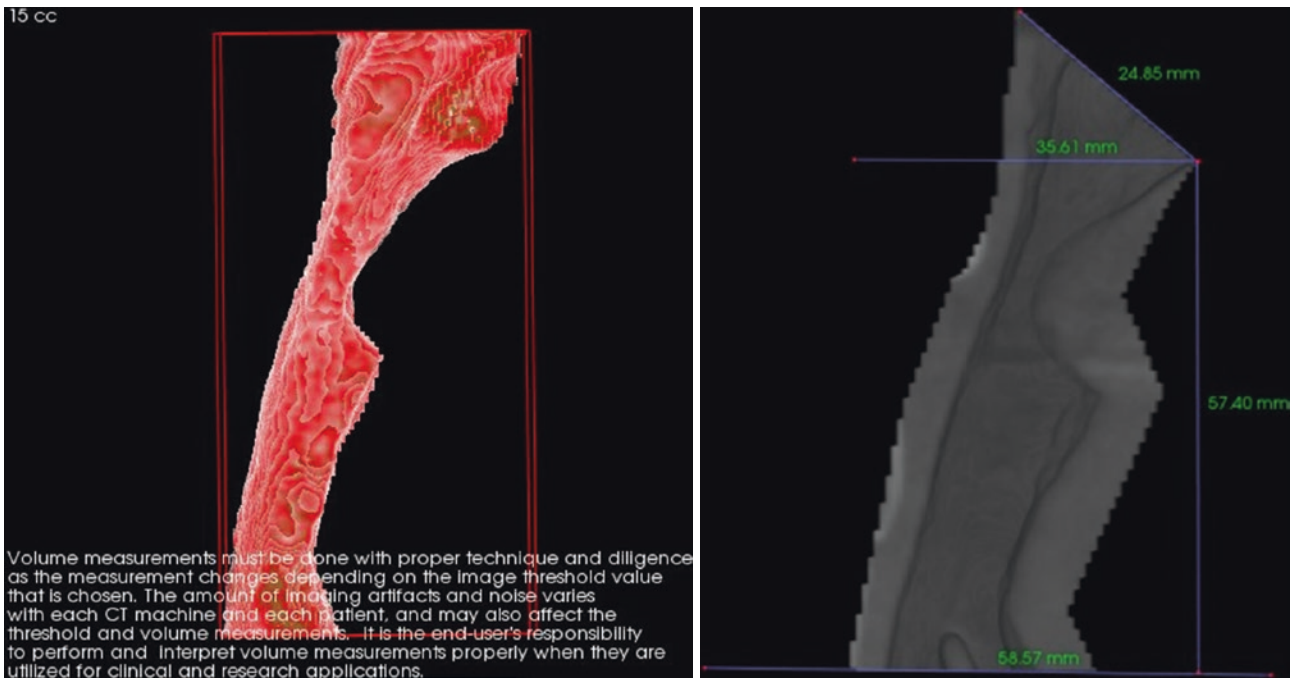


Fig. 21.3 Establish the boundaries for pharyngeal airway

ensure that structures present bilaterally correspond with each other [31].

To accurately compare and analyze the airway space before and after treatment, the head posture of the CBCT should be reconstructed with reproducibility, and the upper airway space needs to be evaluated in each section using a tool for evaluating the airway space. Because CBCT provides information in 3D, clinicians may effectively evaluate the airway space and surrounding structures and analyze the narrowest areas and volume of the

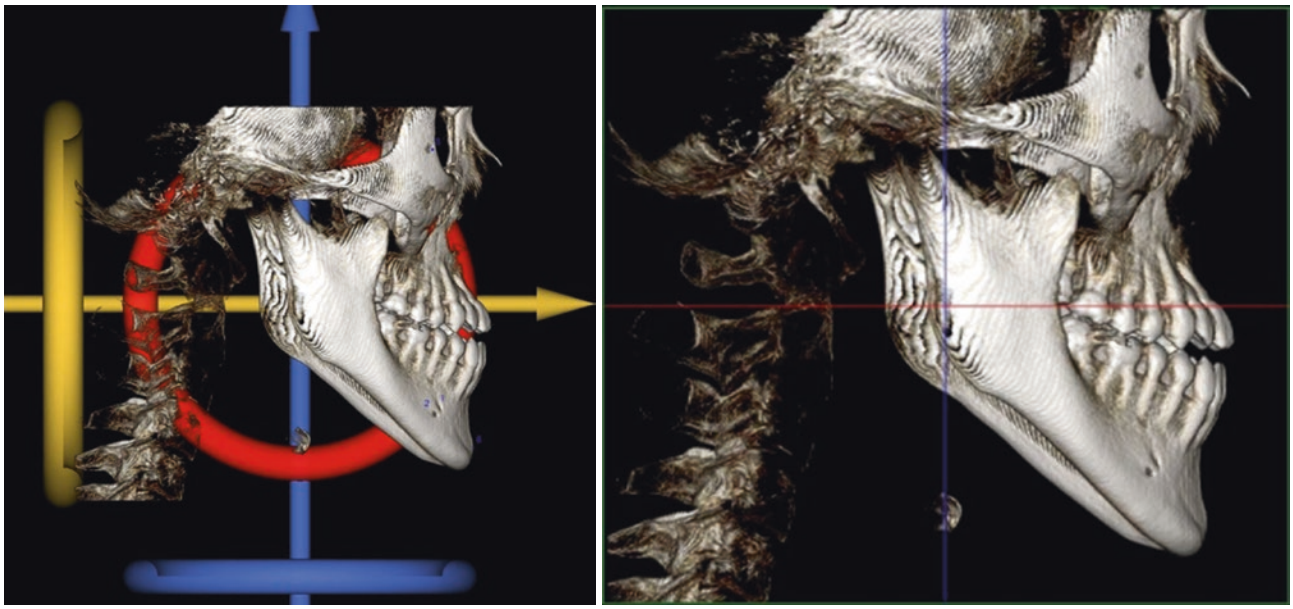


Fig. 21.4 Adjustment of head orientation

nasopharynx, oropharynx, and hypopharynx, which is the smallest portion in the anteroposterior direction and lateral pharyngeal dimension in patients with OSA syndrome (OSAS). Furthermore, it is relatively straightforward to evaluate changes before treatment, providing a baseline from which the patient's response to treatment may be gauged over time by superimposition of subsequent follow-up images [32].

21.2 Visualization of the Airway Space for Volumetric Analysis

To evaluate the upper airway space, CBCT images were reconstructed using InVivo5 (Anatomage, San Jose, CA, USA) in the form of DICOM files. After creating the 3D image of the entire raw dataset, Invivo5 facilitates an assessment of the specific airway region of interest with tools to separate and delete opaque areas corresponding to skin and bone (using the "Remove" function of the volume render of the segmentation tool). The upper border may be set as the plane across the superior part of the atlas; the lower border may be set as the plane crossing the lowermost point of the fourth cervical vertebra. Anteroposterior walls may be defined as the anatomical border of the pharynx. After adjusting opacity threshold to −750 with the fine tuning bar, the final 3D image of each pharyngeal cavity was constructed using the volume rendering function [31] (Fig. 21.5).

The 3D reconstructed image allows a detailed comparative evaluation of the pattern of the airway space. For example, the airway space of non-OSAS group has a more rounded or rectangular shape, while that of the patients with OSAS has a more elliptical or concave shape [33]. Class III skeletal relationships are characterized by a wider and flatter anteroposterior direction [31], and, with advancing age, the airway space widens laterally to become more elliptical [34]. Airflow resistance is directly related to both the size and shape of the airway. While the airway space may be large in some cases, its winding path may detrimentally impact airflow with significant resistance, ultimately impacting respiratory function. Reconstructed 3D images may be exported as an STL file and used to analyze the airway airflow using the finite-element study.

21.3 Airway Space Change and Stability Related to Orthognathic Surgery

21.3.1 Mandibular Setback and Bimaxillary Surgery

Skeletal Class III malocclusion may be accompanied by the combination of a prominent mandible and retracted maxilla or mandibular prognathism alone [19, 26, 35, 36]. To improve the skeletal discrepancy, orthognathic surgery (mandibular setback or MMA) is recommended to improve masticatory function and aesthetics [17]. In orthognathic surgeries involving mandibular setback, both preoperative and postoperative evaluation of the airway space may be performed by CBCT. The advantage of CBCT is that the airway space may be reconstructed in 3D to provide a detailed visualization for analysis (Figs. 21.6 and 21.7).

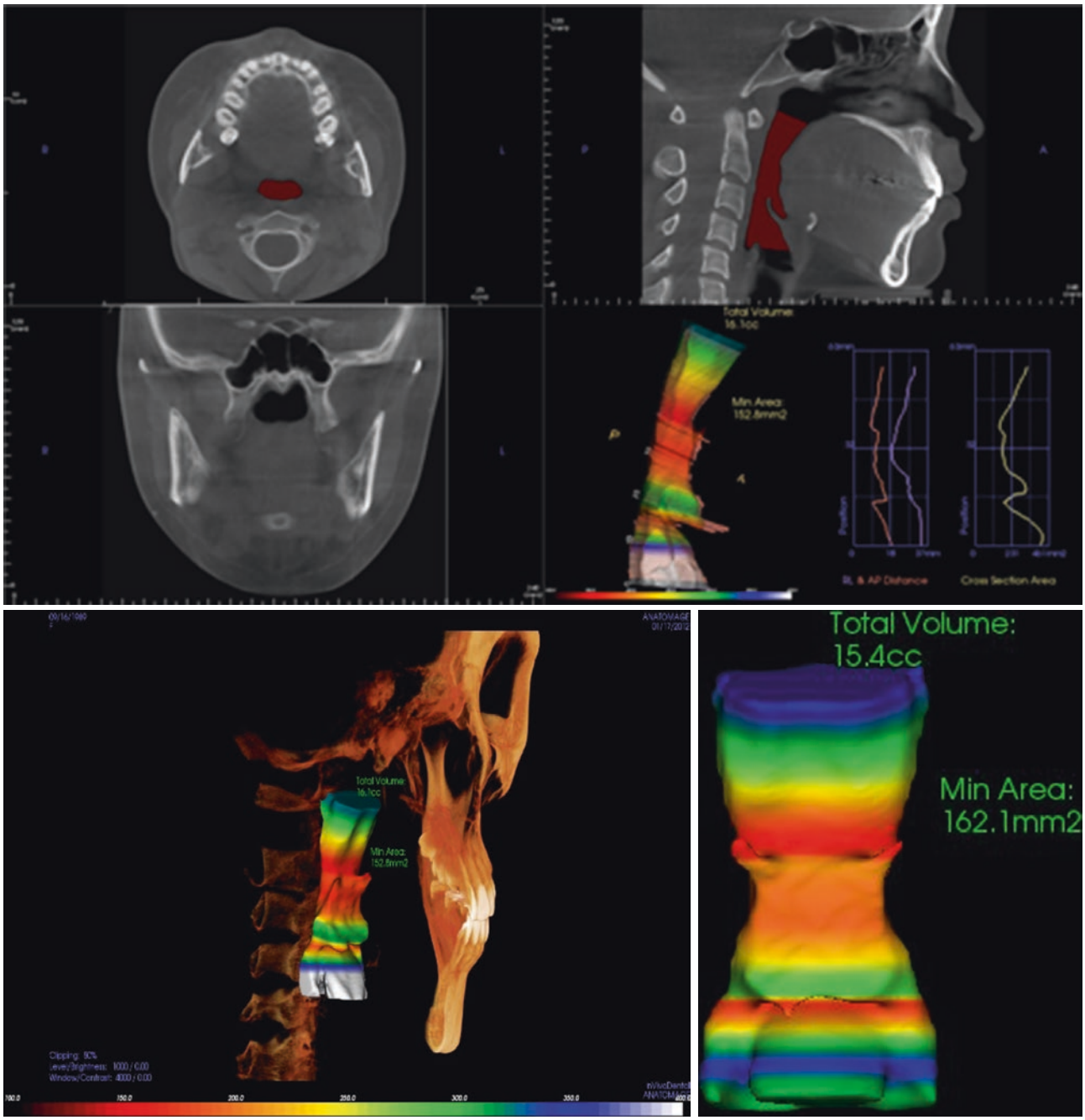


Fig. 21.5 3D image construction process of the pharyngeal cavity

Several studies report the correlation between the change in airway space and orthognathic surgery [22, 23, 27, 28, 37–48]. Mandibular setback surgery for a skeletal class III malocclusion may cause stenosis of the upper airway space immediately after surgery because it repositions the tongue posteriorly as the mandible moves backward (Fig. 21.6). Some cases have reported the occurrence of OSA due to a reduction in volume of the retrolingual and hypopharyngeal airway after mandibular setback and change in position of the hyoid bone [17]. When the mandible is retracted, the hyoid bone is repositioned downward, and a posterior displacement of the tongue and soft palate occurs. This movement eventually results in the narrowing of the upper airway space in the anteroposterior and lateral directions [49]. In addition, relative mean negative pressure decreases, as does pharyngeal airway volume.

In contrast, Wenzel et al. [48] found decreased nasopharyngeal volume following mandibular setback,

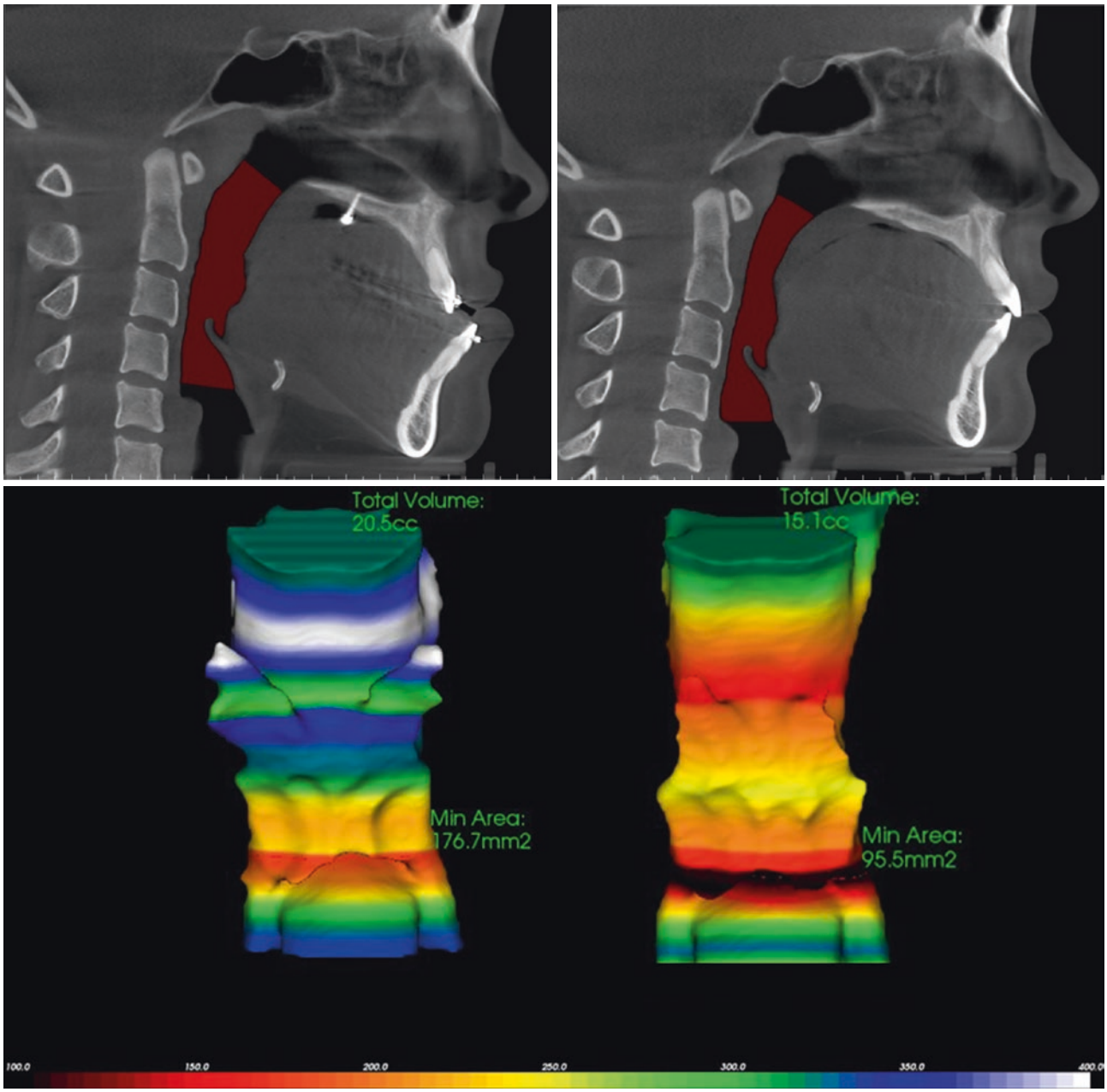


Fig. 21.6 Mandibular setback in this case led to a reduction in total volume of 4.9 cc and a reduction in minimum area of 81.2 mm

which remained reduced over a long period of time. If maxillary advancement is performed with mandibular setback, the narrowing of the airway can decrease. In 2008, Degerliyurtet al [37]. showed the comparative airway changes between bimaxillary surgery and mandibular setback and suggested that bimaxillary surgery could prevent airway stenosis. To some extent, the increased volume of the airway at the nasopharyngeal level in bimaxillary surgery compensates for the effect at the hypopharyngeal level [41]. These reports suggest more effort is needed to maintain a constant pharyngeal airflow during the mandibular setback surgery [29]. Clearly, 3D imaging can adequately support airway assessments for this purpose.

Depending on the type of orthognathic surgery involving mandibular setback, there are conflicting reports on the degree of reduction and duration of retention in each region of the upper airway space. According to Park et al. [19], Tselnik and Pogrel [27], Hochban [25], Wenzel et al. [48], Holmberg et al. [39], Chung and Lee et al. [50], and Lee et al. [29], there was no significant decrease in the nasopharyngeal space; however, the width of oropharynx and hypopharynx decreased. In these studies, mandibular setback caused repositioning

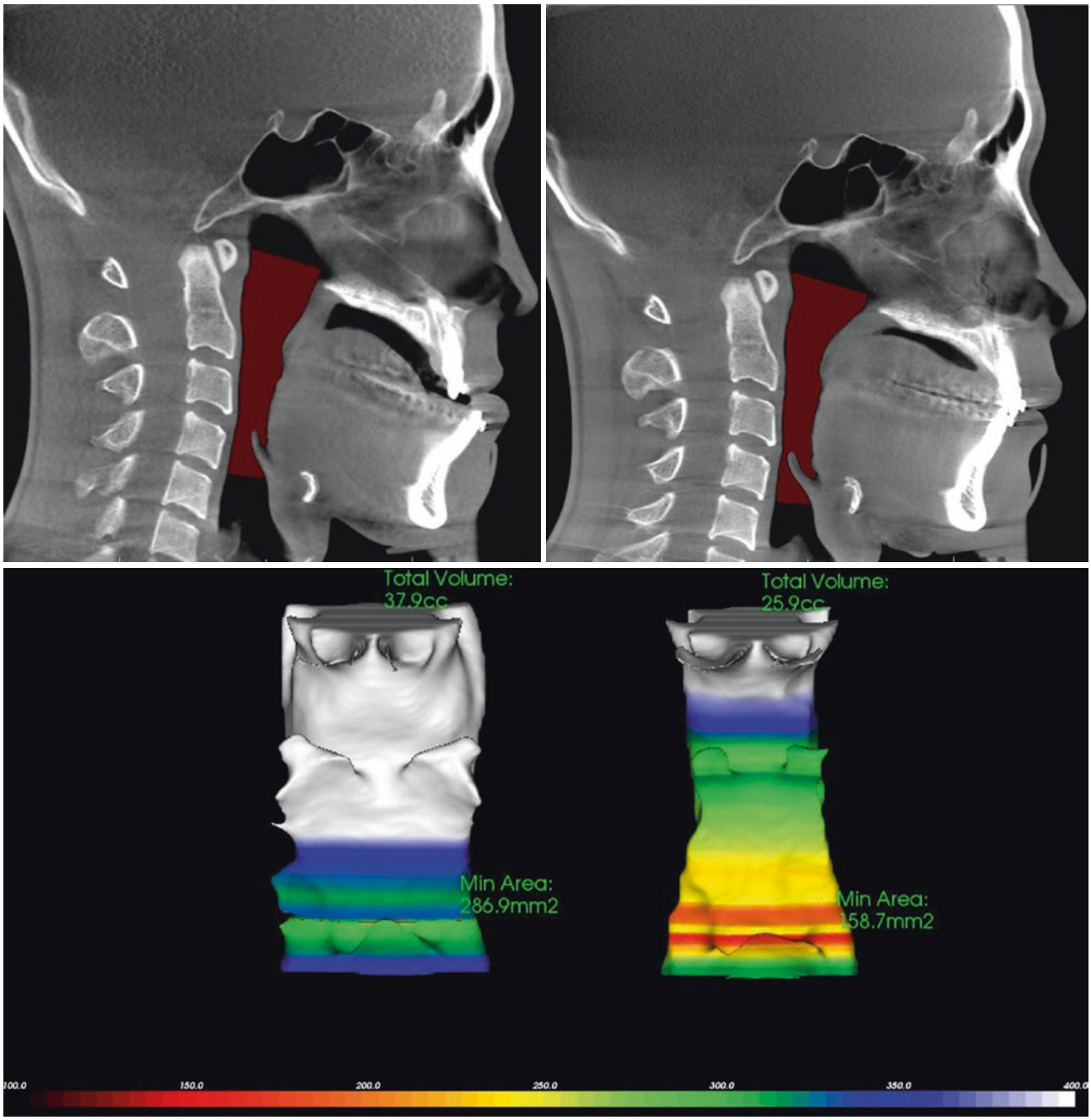


Fig. 21.7 Surgery involving both the maxilla and the mandible led to a 12-cc reduction in total volume and, a 128.2-mm reduction in minimum area

of the tongue in the posteroinferior direction at the skeletal position so that changes of the airway width were greater in the oropharynx and hypopharynx than in the nasopharynx.

In contrast to the previous studies that showed that the nasopharyngeal space remained almost unchanged during the mandibular setback, Kim et al. [16] and Wenzel et al. [48] reported a significant decrease in the nasopharynx after mandibular setback that remains reduced over time. In the study by Kim et al. [16], a reduction in the nasopharyngeal space was observed, but this reduction was smaller than that of the oropharynx and hypopharynx. There are conflicting results regarding nasopharyngeal space reduction. When the decrease in the three parts of the pharyngeal space were compared, reduction in the oropharynx was found to be most severe, followed by the hypopharynx and nasopharynx. Since the oropharynx is the closest to the posterior of the mandible and tongue, it is presumed to be the most affected by surgical mandibular movement.

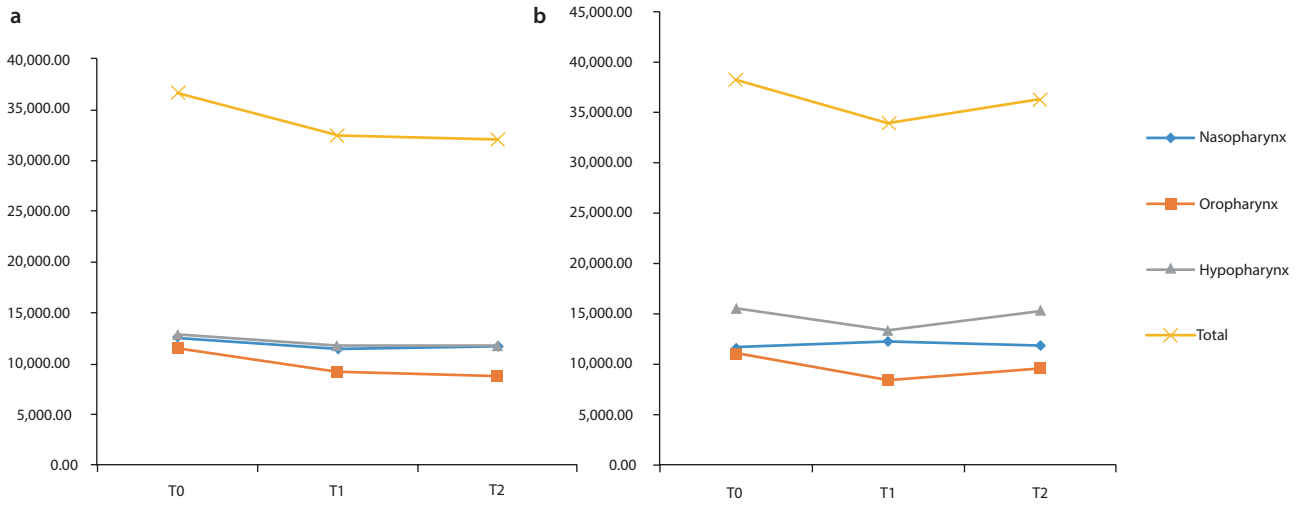


Fig. 21.8 Short- and long-term changes in the airway space after orthognathic surgery of Korean skeletal class III malocclusion patients. Changes in the airway volume of the group with only mandibular setback are shown in plot A; changes in the airway volume of the group with simultaneous maxillary advancement and mandibular setback are shown in plot B. X-axis: T0 (preoperative), T1 (4.6 months after surgery), T2 (1.4 years after surgery). Y- axis: airway volume. The Y-axis (air volume) was measured in cubic millimeters (mm^3) at the X-axis (baseline, T1, T2)

Similar results were obtained in a comparison between mandibular setback only and bimaxillary surgery with maxillary forward movement. Samman et al. [26] reported a decrease in the oropharyngeal and hypopharyngeal space and Cakarne et al. [35] found an increase in nasopharyngeal space. Chen et al. [36] reported changes in the upper airway space in both short- and long-term follow-ups post-mandibular setback and bimaxillary surgery. The mandibular setback group showed a significant decrease in the widths of the nasopharynx and hypopharynx in both short- and long-term follow-ups. After bimaxillary surgery, width of the nasopharynx increased, and oropharynx and hypopharynx widths decreased during short-term follow-up only. However, long-term follow-up showed no significant spatial changes in the structures. In most cases, maxillary advancement was performed simultaneously with bimaxillary surgery, resulting in a decrease in the amount of mandibular setback. Therefore, the duration of the decrease in the airway is not significant when compared to that of mandibular setback only.

It has been reported that the airway changes after orthognathic surgery are sustained for long periods of time and that a reduction in airway volume also persists. In addition, some research shows that the airway changes occur temporarily during tissue re-adaptation. Enacar et al. [24] reported postoperative changes in the oropharyngeal space, reporting that the reduced area of the oropharyngeal airway persisted for more than 18 months. Studies by Kim et al. [16], Tselnik et al. [27], and Hochban et al. [25] reported postoperative oropharyngeal width decreased and adapted to the reduced dimensions during the follow-up period. Significant reductions in the hypopharyngeal space after surgery suggest a functional readjustment of the hyoid bone, tongue muscle, and neck muscle, leading to changes in the airway space [51] (Fig. 21.8).

Few studies report the application of 3D upper airway images, and even fewer report the relationship between changes of the upper airway and post-surgical stability. A study conducted by Park et al. [19] at Pusan National University Dental Hospital (PNUDH) employed 3D CBCT to evaluate how the upper airway changed after orthognathic surgery in patients with skeletal Class III deformities and to analyze the relationship between the changes in the upper airway and post-surgical stability. A total of 36 adult subjects were included (23 men, 13 women; mean age 22.97 ± 3.01 years; range 19 to 29) who had been diagnosed with class III skeletal deformities and underwent surgical orthodontic treatment. As an alternative approach to the analysis of the anatomical characteristics of the upper airway, the anteroposterior length (APL), largest transverse width (LTW), and cross-sectional area (CSA) in five planes and in four volumes were calculated for all subjects (Fig. 21.9).

Patients were divided into groups by type of orthognathic surgery performed: group A (n = 20) underwent mandibular setback sagittal split ramus osteotomy (SSRO with rigid fixation), and group B (n = 16) underwent a LeFort I osteotomy with advancement and mandibular setback SSRO. A 3D CBCT examination was performed at three stages: T0 (before surgery), T1 (an average of 4.6 months after surgery), and T2 (an average of 1.4 years after surgery). While airway decreases were observed in both groups, the oropharyngeal and hypopharyngeal airways in group A showed signifi-

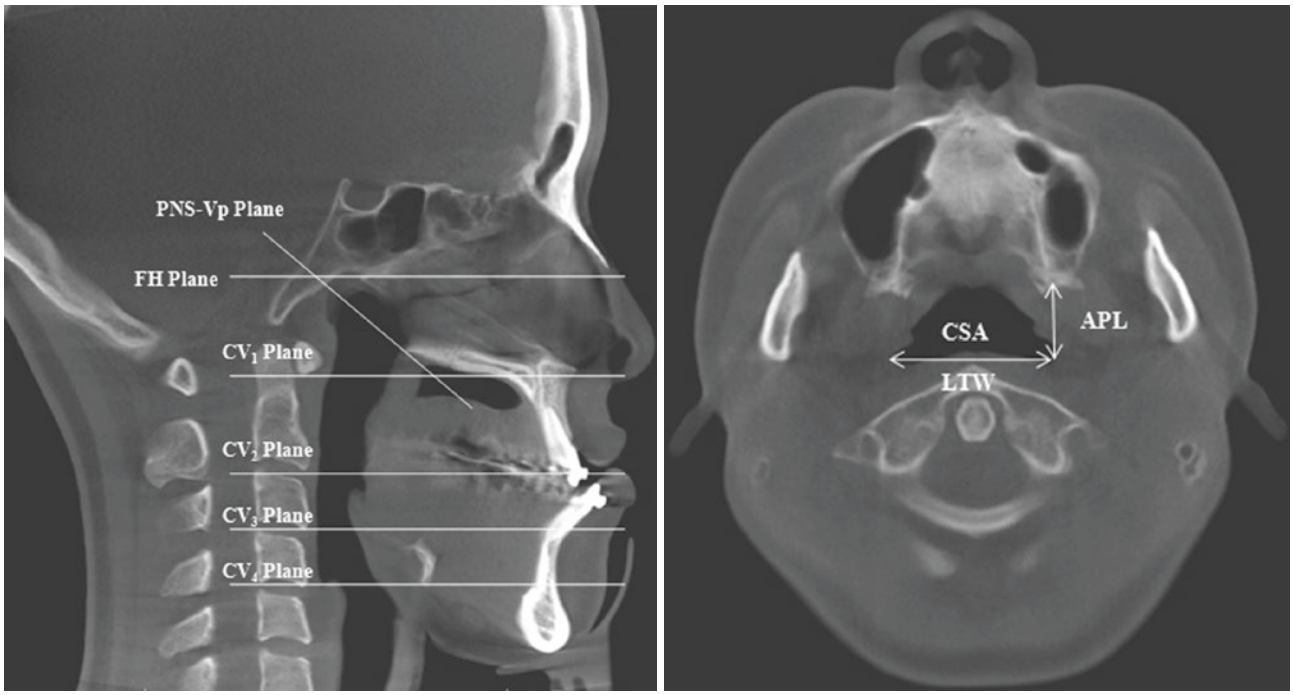


Fig. 21.9 Upper airway measurements. Five planes for upper airway measurement: PNS-Vp, CV_1, CV_2, CV_3, and CV_4 planes

cantly decreased volumes 4.6 months post-procedure ($P < 0.05$) and these diminished airways had not recovered 1.4 years post-surgery. Group B demonstrated post-surgical stability based on the APL of the hypopharyngeal. Group A, however, showed maxillary relapse based on the cross-sectional area of the nasopharynx correlated ($P < 0.05$).

Several studies have insisted that with bimaxillary surgery (compared with mandibular setback surgery), the reduction effect of the mandibular setback is moderate [26, 36, 37, 52]. Results reported by Park et al. are consistent with those findings; however, in patients who had excessive mandibular setback or who already showed signs of sleep apnea (such as obesity, excessive daytime sleepiness, and excessive snoring), other OSA interventions should be considered before orthognathic surgery is pursued [53]. Furthermore, a reduction in oropharyngeal and hypopharyngeal volume was observed 4.6 months after mandibular setback. This decreased volume did not recover until 1.4 years after surgery. The bimaxillary surgery group showed decreased volume of the oropharyngeal airway [19]. Long-term, 12-year follow-up showed a reduction in the hypopharyngeal airway, but the nasopharyngeal and oropharyngeal airways continued to decrease for 12 years [17].

In conclusion, mandibular setback movement reduces a part of the upper airway space in the short and long term [18]. A study by Kim et al. [16] that evaluated the amount of mandibular setback reported no significant changes in the upper airway space when the amount of retraction was less than 11 mm–12 mm; however, a significant change was observed when the amount of retraction was 12 mm or more. Thus, the amount of the mandibular setback is the factor that affects the degree of the change in the upper airway space. To clarify the regional airway changes and duration and the relationship between the amount of mandibular setback and change in the upper airway space, additional studies should be performed.

21.3.2 Vertical Movement of the Maxillomandibular Complex

Orthognathic surgery involving maxillary vertical correction is required in dolichocephalic patients with a skeletal class III malocclusion, represented by long face syndrome and a gummy smile. This skeletal discrepancy can be resolved through vertical repositioning of the maxilla and rotation and setback of the mandible, which is usually accompanied by occlusal plane rotation (Fig. 21.10).

Although surgery that involves occlusal plane rotation and vertical movement of the maxilla to relieve gummy smile is commonly performed, most studies on upper airway space change following orthognathic surgery are either on procedures performed only on the mandible or focused on the horizontal change in the maxilla. However, a 2008 study by Kim et al. [54] set out to observe changes in the vertical movement of the maxilla in 24 patients (9 men, 15 women) with a mean age of approximately

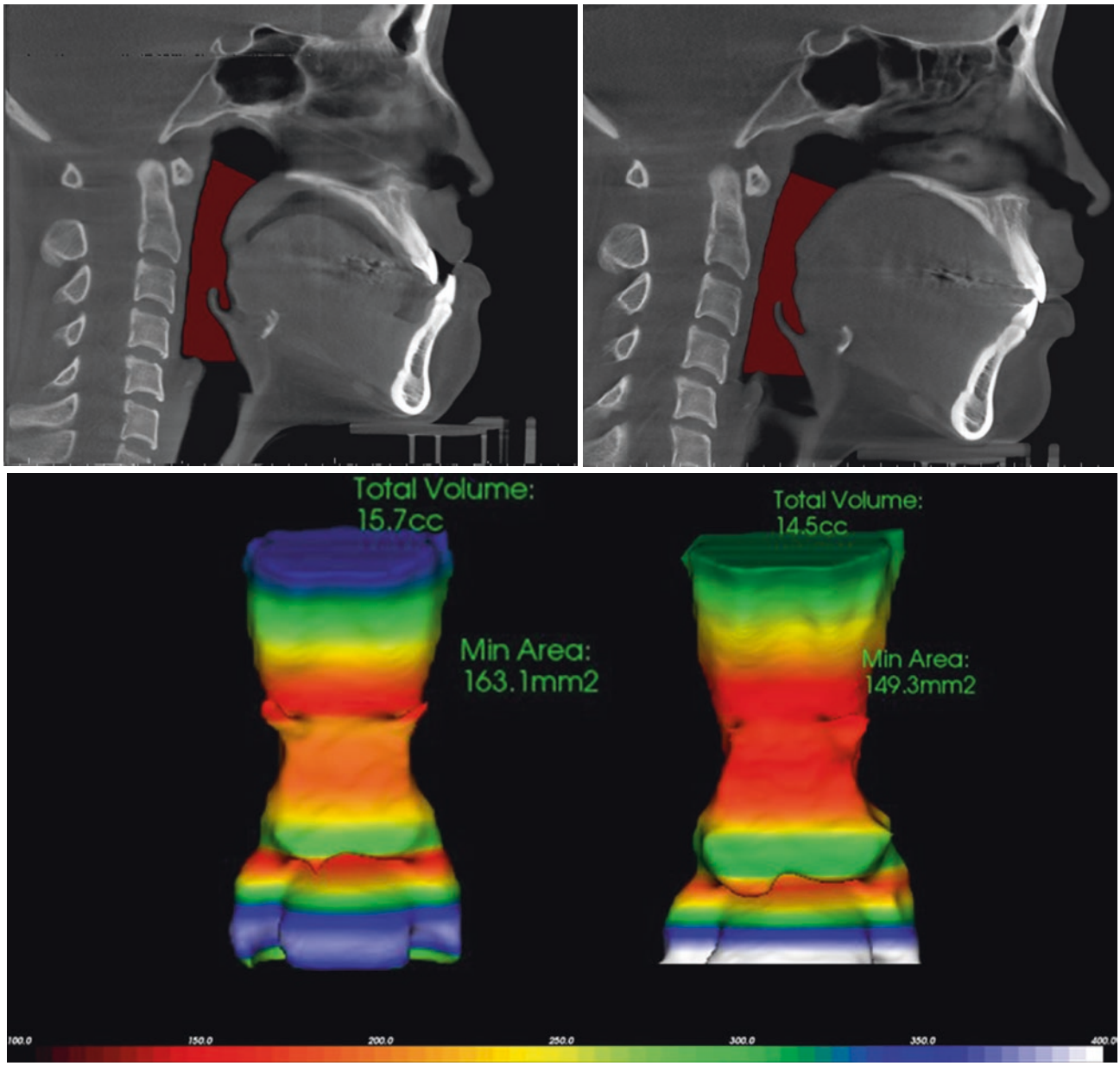
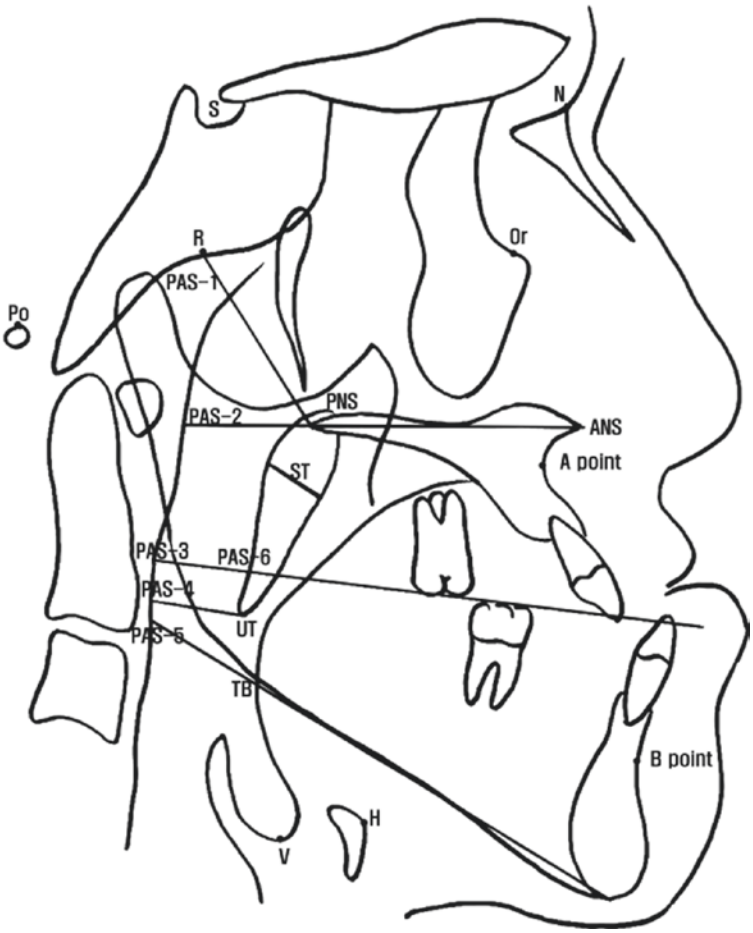


Fig. 21.10 Airway changes after orthognathic surgery with maxillary vertical movement (reduction in total volume of 1.2 cc and reduction in minimum area of 13.8 mm)

22 years who underwent preoperative orthodontic treatment and surgery involving vertical movement of the maxilla with a Le Fort I osteotomy and mandibular setback. Cephalometric radiographs were taken preoperatively (T0), postoperatively (T1, just or within 2 weeks after surgery), and at 6-month follow-up (T2). The radiographs were compared using a paired t-test to show differences in the change in upper airway width based on the vertical movement of the maxilla (Fig. 21.11).

The PAS(R) (starting point of the nasopharynx) observed to be decreased after surgery (T1) ($P < 0.01$), however, showed an increase at 6-month follow-up (T2). Swelling of the soft tissue from intubation during general anesthesia may cause the airway space to decrease in the short term, but soft tissues are known to quickly adapt and contract. The PAS region formed by the palatal plane (NL) showed a significant increase at T1 and T2 due to the vertical movement of the maxilla and the anterior movement component; however, longer term observation is necessary. PAS formed by the occlusal plane (OL) increased at T1 and T2, which may be attributable to the change of the location of the tongue and the soft palate. At T1, the soft palate increased in thickness, but at T2 was observed to be similar to the initial



1) Linear measurements

Pharyngeal airway space width

PAS (R): Distance-Pharyngeal airway space between PAS-1 and PNS

- PAS (NL): Distance between PAS-2 and PNS
- PAS (OL): Distance between PAS-3 and PAS-6
- PAS (UT): Distance between PAS-4 and UT
- PAS (ML): Distance between PAS-5 and TB

Skeletal change

- Vertical PNS: Distance PNS-FH plane
- Vertical ANS: Distance ANS-FH plane
- Horizontal PNS: Distance PNS-N perpendicular plane
- Horizontal ANS: Distance ANS-N perpendicular plane
- Horizontal B point: Distance B-point-S perpendicular plane

2) Angular measurements

- FH-uvular angulation: Angle between FH plane and PNS-UT

Fig. 21.11 Pharyngeal airway space points

thickness or decreased. The angle between the FH plane and the soft palate increased at T2 and appeared to be affected more by the anteroposterior movement of the maxilla rather than the vertical movement. This change likely results from a change in the maxillary anteroposterior position and the biological response to maintain the airway space rather than the muscular relaxation and constriction due to the maxillary vertical position change.

Regression analysis revealed the change in the width of the upper airway was not significantly related to maxillary vertical movement. Mean superior movement of the maxilla for bimaxillary surgery was observed to be 4.40 ± 1.14 mm, resulting in a non-significant change to the upper airway space. Therefore, biological adaptation can take place naturally [54].

However, there are factors that limit maxillary movement, which include the presence of anatomical structures, such as the nasal septum, and nasal breathing habits [55]. Vertical-only movement of the maxilla is rare in bimaxillary surgery; vertical movement coupled with advancement is more common. In this study, about 50% of the vertical maxillary movement was also involved in maxillary advancement [54]. More studies are needed to examine the changes resulting from the maxillary superior posterior rotation or the maxillary superior movement. In addition, further studies using 3D CT need to be done to understand the biological and functional aspects of the 3D changes.

21.3.3 Maxillomandibular Setback

Some patients have both maxillary and mandibular excess and a skeletal class III malocclusion with an acute nasolabial angle. To resolve this maxillomandibular skeletal problem, maxillary retraction can be an effec-

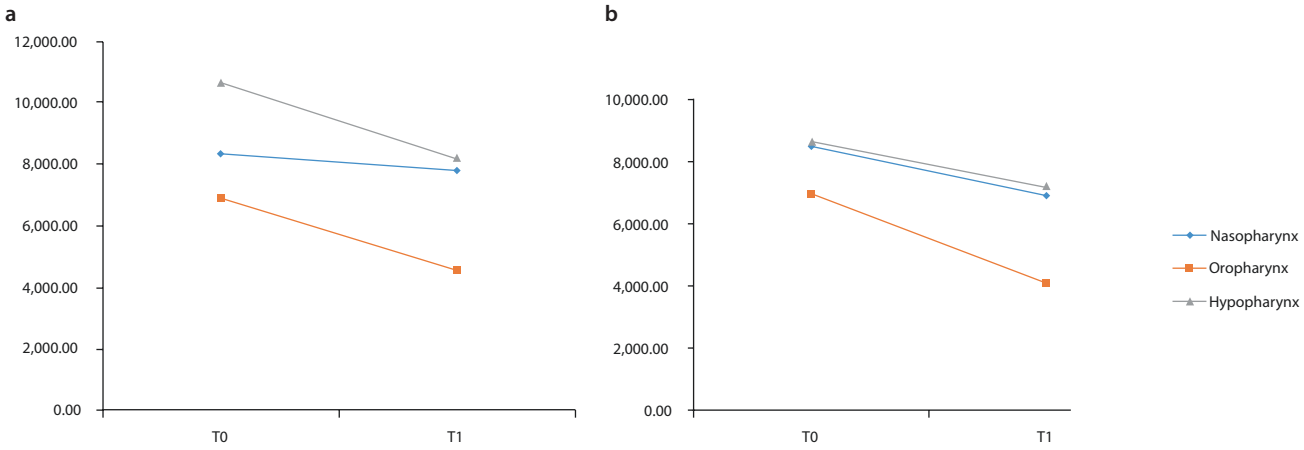


Fig. 21.12 Change in airway volume. Change in the airway volume with mandibular posterior movement of mandible **a**. Change in the airway volume with posterior movement of maxilla (clockwise rotation) before surgery (T0) and 6 months after surgery (T1) **b**. Y-axis volume (air volume) was measured in units of cubic millimeters (mm^3) at each time point on the X-axis (Baseline, T1)

tive treatment option. In addition, aesthetic results can be achieved by performing a clockwise rotation of the maxilla in patients with a recessed midface, flat occlusal plane, and prominent chin with an acute nasolabial angle [29]. Total maxillary setback surgery with or without clockwise rotation is often needed for the correction of a class III malocclusion in Asians.

When maxillomandibular rotation is required and both maxilla and mandible retraction are performed, narrowing of airway and a decrease in airway volume may occur. These types of maxillary movements can cause a concomitant decrease in the airway volume. The posterior movement of the maxilla can reduce the airway volume—it not only adversely affects the nasopharyngeal airway, but also increases the amount of posterior movement of the mandible.

Studies on the changes of the upper airway space after two-jaw surgeries that involve the posterior impaction or setback of the maxilla are rare [56]. A 2013 study by Lee et al. [29] used CBCT to observe changes in upper airway space volume in patients with class III skeletal deformities. Patients in group A (n = 24) underwent mandibular setback surgery. Patients in group B (n = 23) underwent bimaxillary surgery (mandibular setback surgery and maxillary setback Le Fort I osteotomy) (Fig. 21.12 and Table 21.1).

Mandibular setback movement appeared to significantly change the volumes of the oropharynx and hypopharynx, as well as a reduced volume of the APL, LTW, and CSA on the CV_1, CV_2, and CV_3 planes, respectively ($P < 0.05$). Significant reductions were also observed in the APL and CSA volume on the CV_4 plane ($P < 0.05$). Furthermore, group A's oropharyngeal region was observed to be significantly decreased vs. group B's ($P < 0.05$). These data appear consistent with those from other studies [13, 22, 27].

In group B, the upper airway volumes were narrowed by the maxillary and mandibular setback movement (APL on the CV_1, CV_2, CV_3, and CV_4 planes, the LTW on the PNS-Vp, CV_1, and CV_3 planes, and the CSA on the PNS-Vp, CV_1, CV_2, CV_3, and CV_4 planes decreased after the surgery ($P < 0.05$)). Maxillary movement is known to decrease airway volume by its deleterious effect on the nasopharyngeal airway and can also increase the extent of movement from the mandibular setback. Upper airway volumes (including the nasopharyngeal airway) were significantly decreased in group B in comparison to those in group A ($P < 0.01$). Additionally, APL, LTW, and CSA on the PNS-Vp plane were significantly different ($P < 0.05$) [29]. While movement from maxillary setback was shown to increase the extent of the movement from a mandibular setback, no patients developed OSA postoperatively. Special consideration and caution should be exercised when performing bimaxillary surgery on patients who also have a large anteroposterior discrepancy, particularly in skeletal class III patients with a maxillary protrusion [29] (Fig. 21.13).

21.3.4 Maxillomandibular Advancement (MMA)

Class II deformities with a mandibular deficiency may be treated with growth control or compromised treatment if they are mild, but severe cases require orthognathic surgery. In such cases, the type of surgery performed is mainly mandibular advancement rather than maxillary setback. Patients with mandibular retrognathism that require orthognathic surgery often already have snoring problems or OSA. In the 1970s, it was surmised that surgical protraction of the mandible would improve OSA

Table 21.1 Comparison of airway changes between groups (group A: mandibular setback surgery; group B: bimaxillary surgery); from Lee et al. [29] (2013)

	Group A $n = 24$		Group B $n = 23$		
	Mean	**SD**	**Mean**	**SD**	***P***
PNS-Vp plane					
APL (mm)	1.4	3.15	−2.42	6.49	0.008†
LTW (mm)	1.37	2.73	−4.23	9.35	0.004†
CSA (mm^2)	21.26	123.96	−272	144.86	0.0006
CV_1 plane					
APL	−2.51	2.17	−2.88	7.91	0.869
LTW	−2.99	5.17	−2.22	6.43	0.906
CSA	−74.42	79.56	−94.6	134.7	0.777
CV_2 plane					
APL	−3.39	2.61	−2.52	7.17	0.906
LTW	−5.32	4.98	−3.53	9.34	0.346
CSA	−112.59	85.97	−82.21	119.44	0.081
CV_3 plane					
APL	−2.84	2.64	2.61	4.44	0.715
LTW	−1.74	3.48	−1.59	3.63	0.841
CSA	−98	88.08	−52.53	101.19	0.138
CV_4 plane					
APL	−4.54	3.95	−2.11	2.78	0.944
LTW	−0.32	3.45	−1.58	5.16	0.154
CSA	−35.37	80.89	−45.62	113.34	0.925
Volume (mm^3)					
Nasopharynx	−555.03	2135.12	−1604.1	2616.34	0.675
Oropharynx	−2345.36	2897.33	−2856.16	2415.32	0.868
Hypopharynx	−2455.59	2244.85	−1313.34	2064.61	0.984

*$P < 0.05$
†$P < 0.01$ (Mann-Whitney U test)

by improving the retropalatal and retrolingual dimension of the airway during protraction.

In 1983, Powell et al. [57] reported the first case of mandibular advancement for the treatment of OSA, which was achieved by LeFort I and bilateral sagittal split osteotomies. Mandibular advancement causes the hyoid bone and tongue muscle to move forward because the hyoid bone is attached by geniohyoid, suprahyoid, anterior digastric, and mylohyoid muscles. The soft palate, tongue, and anterior pharyngeal tissues follow the movement of the chin. MMA causes an increase in the volume of the nasopharynx, oropharynx, and hypopharynx, resulting in an increase in the PAS [17]. In addition, the minimum and mean cross-sectional areas of the nasopharynx and the mean transverse diameter of the oropharynx have also been reported to increase with the procedure [58]. In conclusion, MMA is considered to be a safe and highly effective treatment for OSA [59].

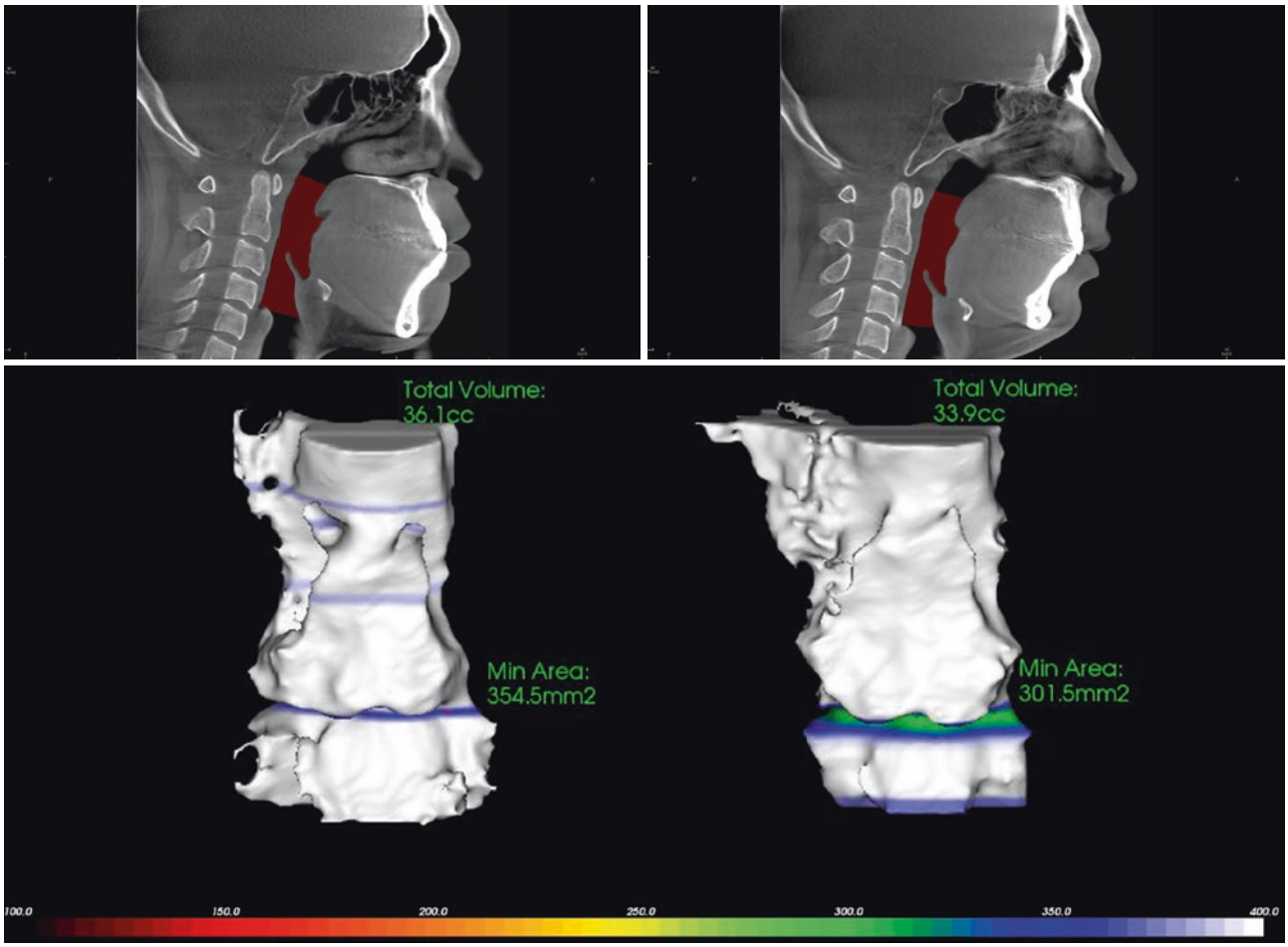


Fig. 21.13 Setback surgery involving both the maxilla and the mandible (reduction in total volume: 2.2 cc, reduction in minimum area: 53 mm)

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Individualized Treatment Planning for Asian Adult Patients with Obstructive Sleep Apnea Syndrome to Obtain Improvement of Respiratory Function and Facial Esthetics: Conventional Maxillomandibular Advancement (MMA) Versus Modified MMA with Segmental Osteotomy

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Contents

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22.1 Introduction

Obstructive sleep apnea syndrome (OSAS) is a sleep disorder with repetitive episodes of hypopnea and apnea induced by the upper airway collapse during inspiration [1–4].

According to age, symptoms, degree, and level of airway obstruction, several treatment modalities have been used for OSAS patients including change in life style, nasal continuous positive airway pressure (nCPAP), oral appliances, miniscrew-assisted rapid palatal expansion appliance, soft tissue procedures, conventional and modified maxillomandibular advancement (MMA) (Fig. 22.1) [2–8].

Among these treatment options, nCPAP is considered as the first line of treatment to overcome the negative pressure during inspiration. However, its compliance rate is less than 50%, especially in moderate-to-severe OSAS cases [4, 9]. Second, soft tissue surgical procedures such as uvulopalatopharyngoplasty (UPPP), tonsillectomy, hyoid suspension, and genioglossus advancement (GA) cannot fully correct narrowing of the pharyngeal airway and their success rates are known to be approximately 40–60% [4, 10]. Third, the MMA procedure with or without counterclockwise rotation can enlarge the facial skeletal framework and upper airway space, resulting in less airway collapse [4, 11–16]. The range of success rate is known to be 75–100% [4, 11–16]. In Caucasian patients, 9–12 mm of MMA has been recommended to achieve maximum expansion of the upper airway [17, 18]. However, since Asians have more protrusive lips and smaller nose compared to Caucasians, these amounts of MMA would be esthetically unfavorable for Asian OSAS patients [2–4, 19–23]. Therefore, several previous studies have proposed modifications of MMA with segmental osteotomy for obtaining improvement of the sleep function and facial esthetics for Asian OSAS patients [2–4, 16, 21–23]. The authors reported the concept of modified MMA surgery, which consists of MMA and segmental osteotomy with or without counterclockwise rotation (seg-MMA; Le Fort I osteotomy and advancement of the posterior segment of the maxilla, and anterior segmental osteotomy and total advancement of the mandible) [2–4].

However, according to the authors' knowledge, there have been a few studies that meticulously described how to plan the amount of advancement or how to set up differential treatment plan for either con-MMA or seg-MMA in Asian OSAS patients [16]. Therefore, the purpose of this study was to compare the effect of seg-MMA with or without counterclockwise rotation on improvement of the respiratory function and facial esthetics in adult OSAS patients with con-MMA procedure.

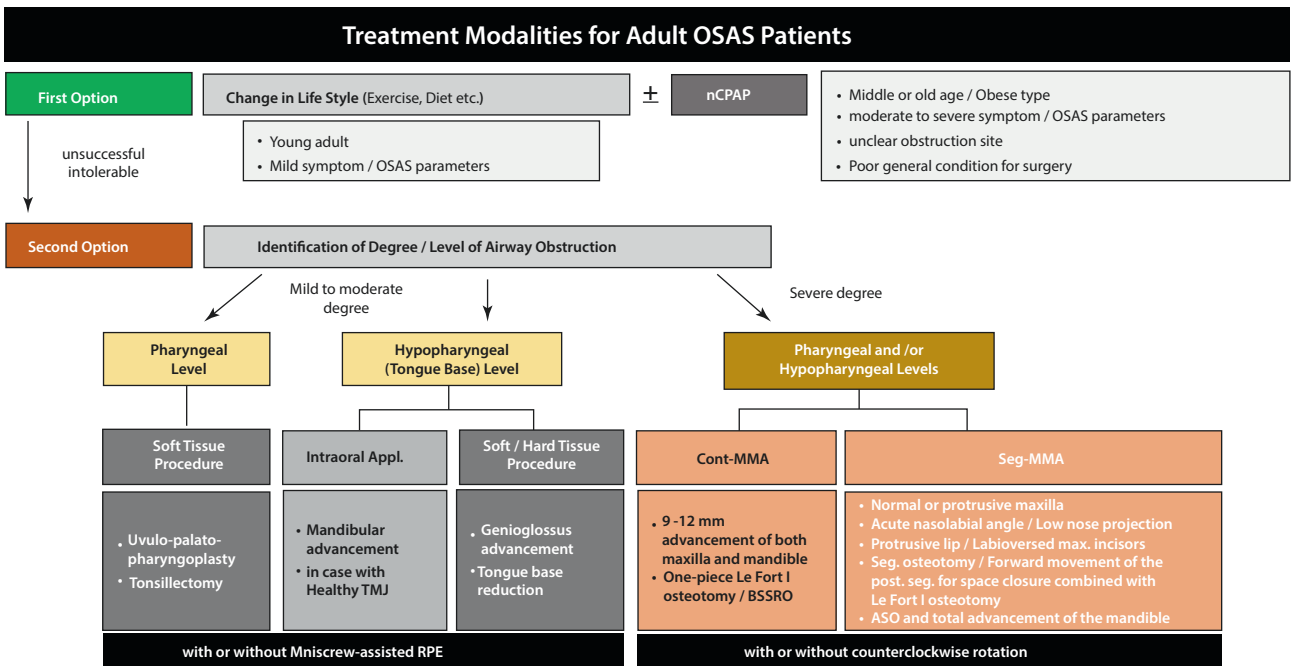


Fig. 22.1 The flowchart of treatment options for obstructive sleep apnea syndrome (OSAS). BMI body mass index, AHI apnea–hypopnea index, nCPAP nasal continuous positive airway pressure, Con-MMA conventional maxillomandibular advancement, Seg-MMA modified MMA with segmental osteotomy, TMJ temporomandibular joint, BSSRO bilateral sagittal split ramus osteotomy

22.2 Material and Methods

22.2.1 The Subjects Used in This Study

The subjects consisted of eight Korean adult OSAS patients (7 males and 1 female; mean age, 26-year-old), who underwent con-MMA or seg-MMA surgery at Seoul National University Dental Hospital from January 2009 to July 2015 by a single oral and maxillofacial surgeon (Choi JY). Two patients had previous surgery history such as septoplasty and UPPP, and other two patients became candidates of con-MMA or seg-MMA treatment due to nCPAP failure. Exclusion criteria were as follows: patients with genetic syndromes, psychological disease, or patients who were unable to comply with the scheduled follow-ups. This study was reviewed and approved by the Institutional Review Board of Seoul National University School of Dentistry (IRB Number S-D20150028).

22.2.2 Demographic Description of the Two Groups

The subjects were divided into two groups: con-MMA group [Le Fort I osteotomy in the maxilla, bilateral sagittal split ramus osteotomy (BSSRO) and genioglossus advancement (GA) in the mandible; n = 4] and seg-MMA group [Le Fort I osteotomy, segmental osteotomy and advancement of the posterior segment of the maxilla, and anterior segmental osteotomy (ASO), posterior movement of the anterior segment, and total advancement of the mandible, without GA; n = 4] (Table 22.1). During surgical procedures, both groups were treated with or without counterclockwise rotation of the maxillomandibular complex according to cephalometric analysis and treatment planning.

The GA procedure was performed only in patients belonged to the con-MMA group, not in patients of the seg-MMA group. The reason was to avoid unfavorable fracture of the mandible during simultaneous ASO and GA. In both groups, computer-aided design/computer-aided manufacturing (CAD/CAM)-made condylar jigs (Orapix Co, Ltd., Seoul, South Korea) were used to stabilize the condyle in the centric relation (CR) position during surgery [24].

22.2.3 Evaluation of the OSAS and Cephalometric Parameters Before and After Surgery

Four OSAS parameters from nocturnal polysomnogram [PSG; Body mass index (BMI, kg/m^2), apnea–hypopnea index (AHI, events/hour), respiratory disturbance index (RDI, events/hour), and lowest saturation rate (LSAT, %)] and five cephalometric measurements (SNA, SNB, FMA, U1 to FH, and nasolabial angle; Fig. 22.2) were investigated before (T1) and 6 months after surgery (T2) (Table 22.2).

The lateral cephalograms were taken at the T1 and T2 stages in the natural head position without any swallowing movement. These lateral cephalograms were traced and analyzed by a single operator (Hong SO) using the V-Ceph program (Version 5.5, CyberMed, Seoul, Korea).

All variables of eight patients were retraced and reassessed by the same operator (Hong SO) at a two-week interval. Since there were no significant differences between the first and second measurements, the first set of measurements was used. The Wilcoxon signed ranks test and Mann-Whitney U test were performed for statistical analysis.

22.3 Results

22.3.1 Success Rate

According to the success criteria for OSAS treatment by Sher et al. [25] (postoperative AHI <20 events/hour and >50% reduction compared with the preoperative AHI), overall success rate was 87.5%. (con-MMA group, n = 4/4 and seg-MMA group, n = 3/4, Table 22.2).

22.3.2 Comparison of the OSAS Parameter Measurements Between the T1 and T2 Stages in the Con-MMA and Seg-MMA Groups (Table 22.3)

There was the same trend of change in the OSAS parameter measurements from the T1 to T2 stages in the two groups as follows: Both groups showed decrease in AHI

Table 22.1 Demographic data for obstructive sleep apnea (OSAS) patients and their individualized surgical treatment plan

Group	Patient number	Age /sex	SNA (°)	SNB (°)	Overjet (mm)	Maxillary incisor showing (mm)	FMA (°)	U1 to FH (°)	Nasolabial angle(°)	Preoperative orthodontic treatment (month)	Operation
Group 1 (conventional MMA)	1	22Y/F	83.6	72.7	4.5	8.0	46.7	98.5	105.3	4	MMA: Mx (Total Imp 4 mm) and Mn (Adv 5 mm) GA (4 mm)
	2	23Y/M	75.6	69.9	3.5	3.0	42.1	101.6	105.4	None	MMA: Mx (Adv 7 mm; Ant Imp 3 mm) and Mn (Adv 8 mm) GA (5 mm)
	3	26Y/M	78.0	73.7	3.0	0	42.7	109.0	90.8	None	MMA: Mx (Total Elong 2 mm; Adv7 mm) and Mn (Adv 10 mm) GA (4 mm)
	4	28Y/M	76.8	71.2	5.0	2.0	22.9	102.3	109.0	None	MMA: Mx (Adv5 mm; Ant Imp 1 mm) and Mn (Adv 6 mm) GA (6 mm)
Group 2 (modified MMA)	5	40Y/M	85.7	82.1	2.5	1.0	27.7	111.6	98.8	3	Modified MMA: Mx (posterior segment Adv- 6 mm) and Mn (Adv 4.5 mm)
	6	27Y/M	85.1	79.3	3.5	3.0	23.6	99.9	87.8	10	Modified MMA: Mx (posterior segment Adv- 5 mm) and Mn (Adv 11.5 mm)
	7	21Y/M	78.5	73.6	3.5	2.0	32.8	117.2	94.0	0.5	Modified MMA: Mx (Ant Imp, 2 mm; post. Imp, 1.5 mm; posterior segment Adv 9 mm) and Mn (Adv 10.5 mm)
	8	20Y/M	74.7	71.6	5.0	4.0	36.1	113.4	80.0	5	Modified MMA: Mx (posterior segment Adv 5 mm) and Mn (Adv 5 mm)

MMA maxillomandibular advancement, *CCR* counterclockwise rotation, *Adv* advancement, *Imp* impaction, *Elong* elongation, *Ant* anterior, *Mx* maxilla, *Mn* mandibular, *GA* genioglossus advancement, *SNA* angle from sella to nasion to subspinale, *SNB* angle from sella to nasion to supramentale

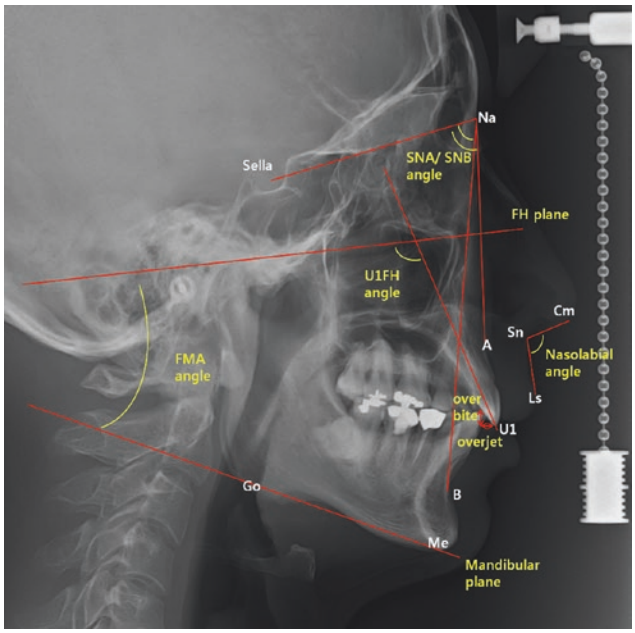


Fig. 22.2 Cephalometric parameters. SNA angle from Sella to nasion to point A, SNB angle from sella to nasion to point B, Nasolabial angle, angle formed by the columellar (Cm)–subnasale (Sn)-labial superioris (Ls), U1 to FH angle between the maxillary incisor and Frankfort horizontal plane, FMA, Frankfort horizontal plane to mandibular plane angle, Overjet sagittal distance between the maxillary incisor tip and the mandibular incisor tip, and overbite vertical distance between the maxillary incisor tip and the mandibular incisor tip

and RDI (AHI, 44.9 to 7.6 in the con-MMA group and 35.8 to 5.7 in the seg-MMA group; RDI, 50.7 to 11.0 in the con-MMA group and 41.6 to 10.8 in the seg-MMA group, Table 22.3) and increase in LAST (84.5% to 88.2% in the con-MMA group and 86.8% to 91.5% in the seg-MMA group, Table 22.3). However, both groups did not exhibit significant change in BMI (21.3 to 21.3 kg/m^2 in the con-MMA group and 21.9 to 21.6 kg/m^2 in the seg-MMA group, Table 22.3).

22.3.3 Comparison of the Amounts of Change in the OSAS Parameter Measurements Between the Con-MMA and Seg-MMA Groups During T1–T2 Stages (Table 22.3)

In terms of BMI, AHI, RDI, and LAST, there was no significant difference in the amounts of change between the two groups (all $P > 0.05$, Table 22.3).

22.3.4 Comparison of the Cephalometric Measurements Between T1 and T2 Stages in the Con-MMA and Seg-MMA Groups (Table 22.3)

There were some differences in the trend of change in the cephalometric measurements from the T1 to T2 stages between the two groups as follows: The con-MMA group showed a forward positioning of the maxilla and mandible (SNA, 78.5 to 82.5°; SNB, 71.9 to 76.4°, Table 22.3) and decrease in obtuse nasolabial angle (NLA, 102.6 to 98.9°, Table 22.3). However, the seg-MMA group exhibited that due to a forward movement of the posterior segment of the maxilla, and the anterior segmental osteotomy and total advancement of the mandible, there were no significant changes in the sagittal position of the maxilla and mandible (SNA, 81.0 to 80.8°; SNB, 76.7 to 77.6°, Table 22.3) and no exaggeration of the acuteness in nasolabial angle (NLA, 90.2 to 88.2°, Table 22.3). However, there was uprighting of the labioversed maxillary incisor in the seg-MMA group (U1 to FH, 110.5 to 104.4°, Table 22.3). Both groups showed some decrease in FMA (38.6 to 36.5° in the con-MMA group; 30.1 to 28.8° in the seg-MMA group, Table 22.3).

22.3.5 Comparison of the Amounts of Change in the Cephalometric Measurements Between the Con-MMA and Seg-MMA Groups During T1–T2 Stages (Table 22.3)

Although FMA, U1 to FH, and nasolabial angle did not show significant difference in the amounts of change between the two groups (all $P > 0.05$, Table 22.3), ΔSNA exhibited a marginal difference between the two groups (4.0° in the conv-MMA group vs. 0.2° in the seg-MMA group, $P = 0.057$, Table 22.3). In the seg-MMA group, there was no significant change in the sagittal position of the maxilla (SNA, 81.0 to 80.8°, Table 22.3) due to a forward movement of the posterior segment of the maxilla.

ΔSNB exhibited significant difference between the two groups (4.5° in the con-MMA group vs. 0.9° in the seg- MMA group, $P < 0.05$, Table 22.3). This difference occurred because the seg-MMA group had anterior segmental osteotomy and total advancement of the mandible, resulting in no significant change in the

Table 22.2 Changes in the OSAS parameters and cephalometric measurements before (T1) and 6 months after conventional MMA or modified MMA surgery (T2)

Group	Patient number	PSG results								Cephalometric measurements													
		BMI (kg/m^2)		AHI (n/h)		RDI (n/h)		LSAT (%)		SNA (°)		SNB (°)		Overjet (mm)		Maxillary incisor showing (mm)		FMA (°)		U1 to FH (°)		Nasolabial angle(°)	
		T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
Group 1 (conventional MMA)	1	18.8	17.3	39.8	2.1	53.3	2.6	82	91	83.6	84.5	72.7	75.8	4.5	1.8	8.0	2.5	46.7	43.9	98.5	99.0	105.3	116.4
	2	23.7	22.5	42.9	5.5	42.9	16.6	90	91	75.6	81.1	69.9	76.4	3.5	3.5	3.0	3.0	42.1	39.9	101.6	105.0	105.4	98.2
	3	21.5	21.6	53.5	7.6	55.8	13.9	87	91	78.0	85.5	73.7	79.9	3	3	0	3.0	42.7	42.9	109.0	104.8	90.8	85.4
	4	21.2	23.8	43.2	15.2	NA		79	80	76.8	78.9	71.2	73.6	5	5	2.0	3.0	22.9	19.2	102.3	107.7	109.0	95.6
Group 2 (modified MMA)	5	18.9	19.9	60	6	61	11	80	92	85.7	84.7	82.1	82.9	2.5	1.7	1.0	1.5	27.7	29.6	111.6	112.3	98.8	99.0
	6	22.9	22	61	6	64.5	16.9	89	92	85.1	83.5	79.3	79.2	3.5	2.8	3.0	3.0	23.6	23.3	99.9	93.8	87.8	82.7
	7	21.1	21.9	14.2	7.9	28.9	8.3	87	90	78.5	79.2	73.6	75.3	3.5	2	2.0	2.0	32.8	27.2	117.2	107.1	94.0	92.9
	8	24.7	22.7	7.9	2.7	11.9	6.8	91	92	74.7	75.8	71.6	73.1	5	3	4.0	3.5	36.1	35.2	113.4	104.2	80.0	78.1

MMA maxillomandibular advancement, *BMI* body mass index, *AHI* apnea–hypopnea index, *RDI* respiratory disturbance index, *LSAT* lowest saturation rate of O_2, *NA* not available

Table 22.3 Comparison of the polysomnogram (PSG) results and cephalometric measurements between modified MMA and MMA

		Group 1 (conventional MMA)					Group 2 (modified MMA)					Comparison of T1 between two groups	Comparison of T2 between two groups	Comparison of ΔT1 − T2 between two groups
		T1		T2		*p*-value[a]	T1		T2		*p*-value[a]	*p*-value[b]	*p*-value[b]	*p*-value[b]
		mean	SD	mean	SD		mean	SD	mean	SD				
PSG results	BMI (kg/m^2)	21.3	2.0	21.3	2.8	1.000	21.9	2.5	21.6	1.2	0.715	1.0000	1.0000	1.0000
	AHI (n/h)	44.9	6.0	7.6	5.6	0.068	35.8	28.7	5.7	2.2	0.068	1.0000	1.0000	1.0000
	RDI (n/h)	50.7	6.8	11.0	7.4	0.109	41.6	25.5	10.8	4.5	0.068	1.0000	1.0000	0.629
	LSAT (%)	84.5	4.9	88.3	5.5	0.066	86.8	4.8	91.5	1.0	0.066	0.4860	0.2000	0.886
Cephalometric measurements (Korean norm)	SNA (81.43° ± 3.10°)	78.5	3.5	82.5	3.1	0.068	81.0	5.3	80.8	4.1	0.715	0.4860	0.6860	**0.057**
	SNB (79.48° ± 2.85°)	71.9	1.7	76.4	2.6	0.068	76.7	4.9	77.6	4.3	0.144	0.2000	1.0000	**0.029***
	FMA (22.51° ± 3.80°)	38.6	10.7	36.5	11.6	0.144	30.1	5.5	28.8	5.0	0.465	0.3430	0.3430	0.686
	U1 to FH (116.0° ±5.60°)	102.9	4.4	104.1	3.7	0.465	110.5	7.5	104.4	7.8	0.144	0.2000	1.0000	0.114
	Nasolabial angle (91.11° ± 8.12°)	102.6	8.1	98.9	12.9	0.465	90.2	8.1	88.2	9.5	0.144	0.1140	0.3430	0.343

SD standard deviation, *MMA* maxillomandibular advancement, *BMI* body mass index, *AHI* apnea–hypopnea index, *RDI* respiratory disturbance index, *LSAT* lowest saturation rate of O_2, *SNA* sella to nasion to subspinale, *SNB* angle from sella to nasion to supramentale, *Nasolabial angle* angle formed by the columella-subnasale-labrale superioris, *U1 to FH* angle of the maxillary incisor inclination to Frankfort horizontal line (FH), *FMA* Frankfort mandibular plane angle

*$p < 0.05$

[a]Wilcoxon signed ranks test was performed

[b]Mann-Whitney test was performed

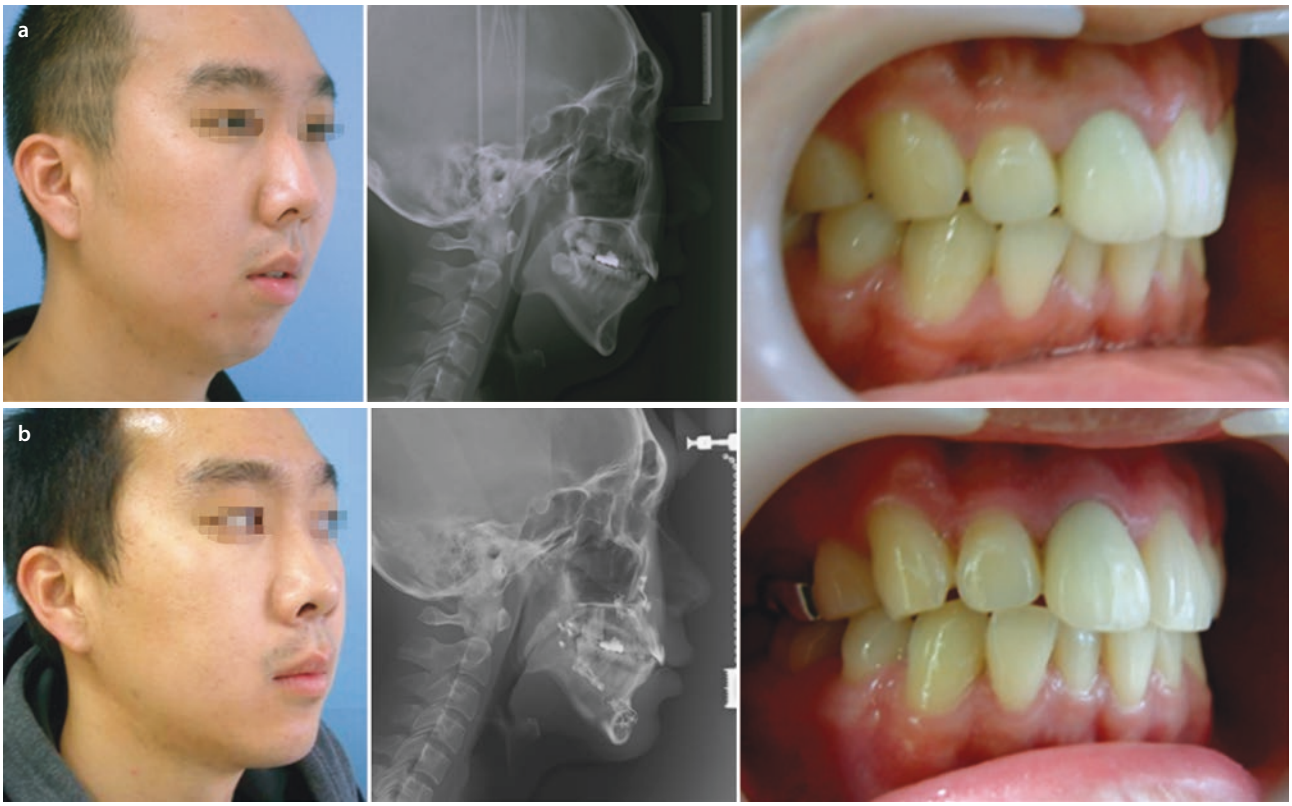


Fig. 22.3 Case 1 (patient number #2) treated with conventional maxillomandibular advancement (con-MMA), which consisted of Le Fort I osteotomy with counterclockwise rotation of the maxilla, bilateral sagittal split ramus osteotomy (BSSRO), and genioglossus advancement. **a** Initial status, **b** after con-MMA

sagittal position of the mandible (SNB, 76.7 to 77.6°, Table 22.3).

In summary, patients with relatively retrusive maxilla, obtuse nasolabial angle, and normal maxillary incisor inclination can be treated with con-MMA procedure. However, patients with relatively forward positioned maxilla, acute nasolabial angle, and labioversed maxillary incisors can be treated with seg-MMA procedure.

22.4 Cases

22.4.1 Case 1 (Patient #2): Con-MMA Procedure with Counterclockwise Rotation and GA

A nonobese OSAS patient (23-year-old male; BMI, 23.7; Table 22.2) was referred from the ENT department for MMA surgery. The results of initial PSG showed severe OSAS (AHI, 42.9 events/hour; RDI, 42.9 events/hour, Table 22.2). He had skeletal Class II facial pattern, relatively large nose, 3.5 mm overjet, and 3.0 mm maxillary incisal showing at the resting position (Fig. 22.3 and Table 22.2). Lateral cephalogram showed a hyperdivergent facial pattern, retrusive maxilla and mandible, and obtuse nasolabial angle (FMA, 42.1°; SNA, 75.6°, SNB, 69.9°; NLA, 105.4°; Fig. 22.3 and Table 22.2).

Con-MMA procedure with counterclockwise rotation of the maxilla to improve the steep occlusal plane [Le Fort I osteotomy (anterior impaction: 3 mm, posterior impaction: 0 mm, maxillary advancement, 7 mm) and BSSRO advancement of the mandible (mandibular advancement, 8 mm)] was planned (Table 22.1). The amount of maxillary advancement was determined by allowing the maxilla to stay in the normal range of cephalic measurements. In addition, 5.0 mm GA was performed to maximize improvement of the OSAS parameters and chin projection (Table 22.1 and Fig. 22.3).

There was significant improvement in the OSAS parameters (AHI, 42.9 to 5.5 events/hour; RDI, 42.9 to 16.6 events/hour, Table 22.2). Cephalometric measurement also showed significant increase in SNA (75.6 to 81.1°) and SNB (69.9 to 76.4°) and decrease in nasolabial angle (105.4 to 98.2°) (Fig. 22.3 and Table 22.2).

22.4.2 Case 2 (Patient #5): Seg-MMA Procedure

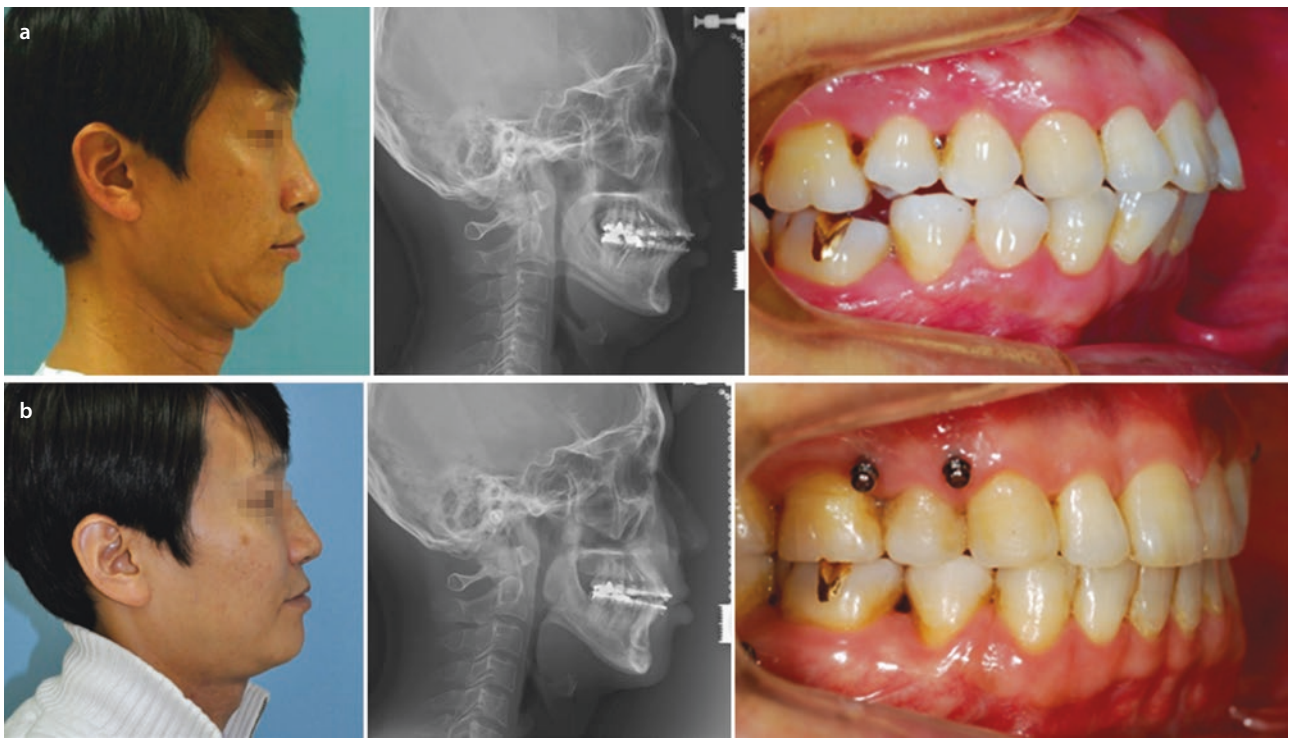
A nonobese patient (40-year-old male; BMI, 18.9; ◘ Table 22.2) was referred for OSAS treatment. Although nCPAP therapy was initially tried, the patient could not tolerate it. The initial PSG results showed severe OSAS (AHI, 60 events/hour; RDI, 61 events/hour; LSAT, 80%, ◘ Table 22.2). He had labioversed maxillary and mandible anterior teeth, skeletal Class II facial pattern, 1 mm maxillary incisor showing, and 2.5 mm overjet (◘ Fig. 22.4 and ◘ Table 22.2). Cephalometric measurement showed a relatively normodivergent facial pattern, protrusive maxilla and mandible, and slight obtuse nasolabial angle (FMA, 27.7°; SNA, 85.7°; SNB, 82.1°; NLA, 98.8°; ◘ Fig. 22.4 and ◘ Table 22.2).

Excessive maxillomandibular advancement in this patient would lead to bimaxillary protrusion and/or labioversion of the maxillary and mandibular incisors, resulting in unfavorable facial esthetics. Therefore, seg-MMA procedure without counterclockwise rotation was planned to maintain the original sagittal position of the anterior parts of maxilla and mandible. After the dental arches were aligned with presurgical orthodontic treatment (3 months), the posterior segment of the maxilla was advanced 6 mm to close the extraction space of the maxillary first premolars (◘ Table 22.1). The anterior segment of the mandible was moved backward to close the extraction space of the mandibular first premolars and the total mandible was advanced 4.5 mm (◘ Table 22.1). The CAD-CAM made condylar jigs were used to position the condylar segments in a stable position when fixating the plates and screws. The osteotomized segments were fixed by ligating stainless steel wires onto the brackets of the adjacent teeth.

There was significant improvement of OSAS parameters (AHI, 60 to 6 events/hour; RDI, 61 to 11 events/hour; LSAT, 80 to 92%, ◘ Table 22.2) without significant change in cephalometric measurement (SNA, 85.7 to 84.7°; SNB, 82.1 to 82.9°; nasolabial angle, 98.8 to 99°; ◘ Fig. 22.4 and ◘ Table 22.2).

22.4.3 Case 3 (Patient #7): Seg-MMA Procedure with Counterclockwise Rotation

A nonobese patient (21-year-old male; BMI, 21.1; ◘ Table 22.2) was referred due to the chief complaint of OSAS. The initial PSG results showed mild-to-moderate OSAS (AHI, 14.2 events/hour; RDI, 28.9 events/hour; LSAT, 87%, ◘ Table 22.2). He had normal maxillary incisal inclination, labioversed mandible anterior teeth, skeletal Class II facial pattern, large nose, short chin



◘ **Fig. 22.4** Case 2 (patient number 5) treated with modified MMA with segmental osteotomy (seg-MMA), which consisted of extraction of #14, #24, #34, #44, Le Fort I osteotomy and advancement of the posterior segment of the maxilla, and anterior segmental osteotomy and total advancement of the mandible. **a** Initial status, **b** after con-MMA

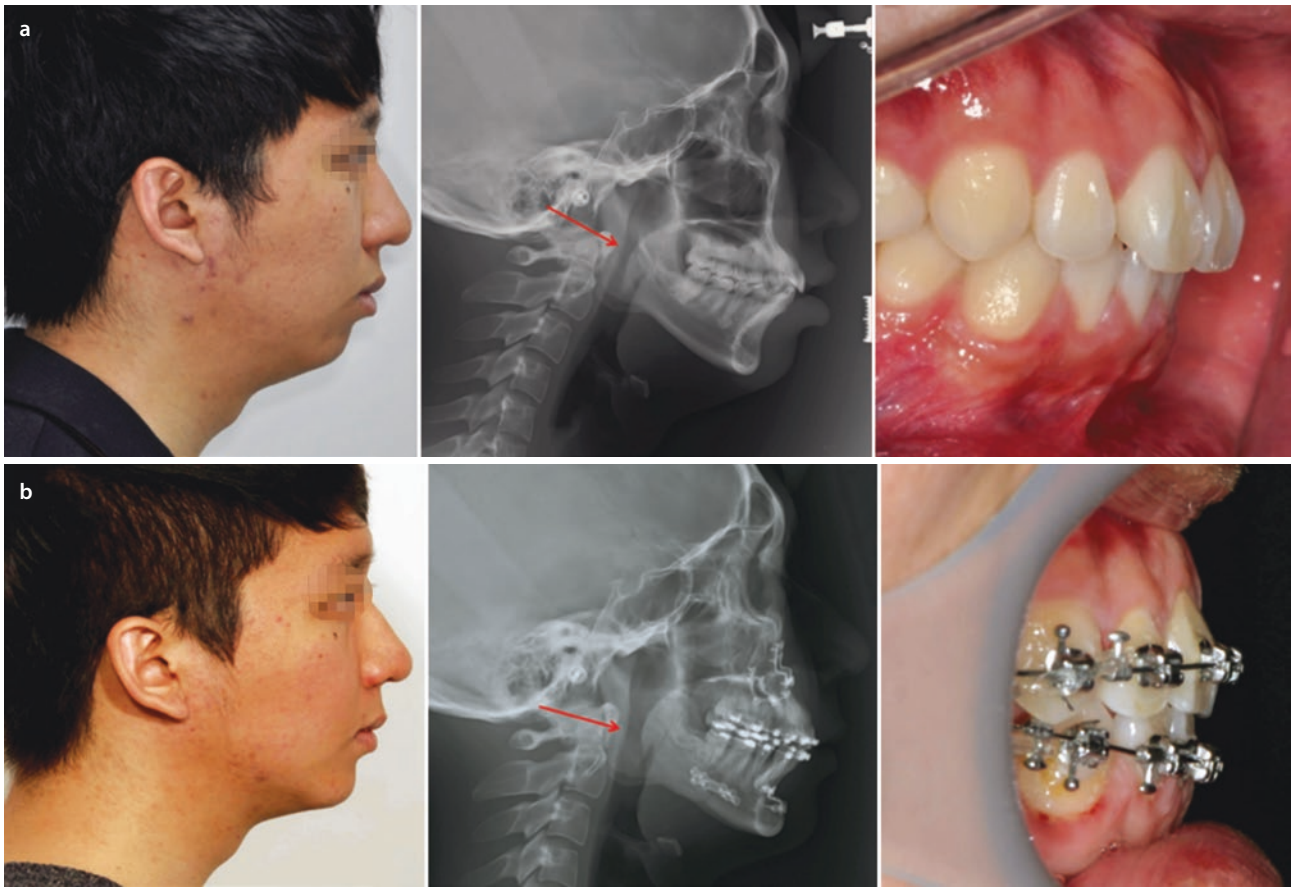


Fig. 22.5 Case 3 (patient number 7) treated with seg-MMA, which consisted of extraction of #14, #24, #34, #44, Le Fort I osteotomy with advancement of the posterior segment and counterclockwise rotation of the maxilla, and anterior segmental osteotomy and total advancement of the mandible. **a** Initial status, **b** after con-MMA. Note the change in the upper airway space (arrow)

length, gummy smile, 2 mm maxillary incisor showing, and 3.5 mm overjet (Fig. 22.5 and Table 22.2). Cephalometric measurement showed a slight vertical pattern, retrusive maxilla and mandible, and normal nasolabial angle (FMA, 32.8°; SNA, 78.5°; SNB, 73.6°; NLA, 94.0°; Fig. 22.5 and Table 22.2).

Although he had a retrusive maxilla, he refused to undergo advancement of the maxilla. Therefore, seg-MMA procedure with counterclockwise rotation was planned to minimally advance the anterior part of the maxilla and increase the amount of mandibular advancement. After the dental arches were minimally aligned with presurgical orthodontic treatment (half months), the posterior segment of the maxilla was advanced 9 mm to close the extraction space of the maxillary first premolars and the maxilla was impacted (anterior, 2 mm; post, 1.5 mm; Table 22.1). The anterior segment of the mandible was moved backward to close the extraction space of the mandibular first premolars and the total mandible was advanced 10.5 mm (Table 22.1). The CAD-CAM made condylar jigs were used to position the condylar segments in a stable position when fixating the plates and screws. The osteotomized segments were fixed by ligating stainless steel wires onto the brackets of the adjacent teeth.

The OSAS parameters showed significant improvement (AHI, 14.2 to 7.9 events/hour; RDI, 29 to 8.3 events/hour, Table 22.2). Although cephalometric measurement did not show significant change in SNA (78.5 to 79.2°), SNB (82.2 to 82.9°), and nasolabial angle (98.8 to 99°) (Fig. 22.5 and Table 22.2), there was a significant enlargement of the posterior airway space (Fig. 22.5).

22.5 Discussion

Caucasians usually have a convex profile, obtuse nasolabial angle, and large nose with high dorsums. On the contrary, East Asians (i.e., Korean, Chinese, and Japanese) have a tendency of protrusive lip, acute nasolabial angle, and small nose with low dorsums. Con-MMA procedure can improve facial esthetics in middle-aged obese OSAS patients with convex profile and obtuse nasolabial angle,

because large amount of advancement can somewhat rejuvenate their face and give a younger looking impression [26]. However, 5–10 mm advancement in young non-obese OSAS patients with protrusive maxilla and acute nasolabial angle should be compromised for conserving facial esthetics. Therefore, it is needed to set up proper consensus on the surgical method and amount of advancement for simultaneous improvement of the OSAS parameters as well as facial esthetics.

When the amounts of change in the cephalometric parameters were compared with the con-MMA group, the seg-MMA group revealed marginally significant changes in SNA and significant change in SNB (ΔSNA, 0.2° vs. 4°, $P = 0.057$ and ΔSNB, 0.9° vs. 4.5°, $P < 0.05$; Table 22.3). In addition, patients who underwent the seg-MMA procedure exhibited a tendency of close-to-normal values and a tendency of relatively forward positioned maxilla and mandible (SNA; 81.0° vs. 78.5°, Korean norm, 81.4°; and SNB; 76.7° vs. 71.9°, Korean norm, 79.5°), less hyperdivergency (FMA, 30.1° vs. 38.6°, Korean norm, 22.5°), greater maxillary incisor inclination (110.5° vs. 102.9°, Korean norm, 116.5°), and smaller nasolabial angle (90.2° vs. 102.6°, Korean norm, 91.1°) (Table 22.3). These cephalometric findings can be used a reference for differential diagnosis between con-MMA and seg-MMA.

Since the seg-MMA procedure did not produce significant differences in the amounts of change in the cephalometric parameters (Table 22.3), it can be considered to produce minimal alteration of facial esthetics by minimization of protrusion of the upper lip or maxilla as well as effective enlargement of the upper airway space and improvement of the OSAS parameters.

Surgical treatment plan for adult OSAS patients must be individualized to provide favorable changes in the respiratory function and facial esthetics [2, 4]. An individualized flow chart to plan the con-MMA or seg-MMA procedure, which is based on cephalometric analysis of the vertical and horizontal skeletal pattern, denture pattern, and soft tissue profile, can be set up using a step-by-step approach (Fig. 22.6).

- **Step 1.** Evaluation of the TMJ status

If OSAS patients have skeletal Class II facial pattern and show centric relation-centric occlusion (CR-CO) discrepancy or degenerative TMJs, too much advancement of the mandible may cause TMJ overload and lead to condylar resorption. In that case, since relapse is inevitable, the status of TMJ should be checked as the first step.

- **Step 2.** Evaluation of the anteroposterior position of the maxilla, upper lip, and nose

When evaluating the cephalometric parameters such as SNA, A-N perpendicular, nasolabial angle, nose height (projection), upper lip to Ricketts' esthetic line, and U1-FH, we have to look at the horizontal position of the maxilla, acuteness of the nasolabial angle, and inclination of the maxillary incisors. If the con-MMA procedure has a possibility of compromising the facial esthetics, the seg-MMA procedure should be considered.

- **Step 3.** Evaluation of the vertical position of the maxillary incisor and steepness of the maxillary occlusal plane

If the amount of the maxillary incisor showing at the resting position is excessive, impaction of the maxilla can be considered. In skeletal Class II patients with steep maxillary occlusal planes, allowing more impaction of the anterior part than the posterior part of the maxilla can be an effective way to produce counterclockwise rotation of the maxilla and to increase the amount of mandibular advancement.

- **Step 4.** Evaluation of the anteroposterior position of the mandible, lower lip, and chin

Once the maxillary position is set up, the mandible can be positioned using SNB, Pog-N perpendicular, ANB, and overbite/overjet. If the con-MMA procedure has a possibility of compromising the facial esthetics, the seg-MMA procedure should be considered. Arpornmaeklong et al. [27] stated that patients with steep mandibular plane angle and more than 10 mm advancement of the mandible experienced significant relapse due to remodeling and/or resorption of the condyle. Therefore, reduction of the amount of mandibular advancement and/or adjunctive treatment such as genioglossus advancement can be performed to minimize TMJ overloading.

- **Step 5.** Evaluation of the vertical height of the lower 1/3 of face

The lower 1/3 facial height should be in proportion with the upper and mid 1/3 facial height. If the lower 1/3 is vertically excessive, adjunctive surgical methods such as reduction genioplasty with or without GA can be applied. However, we have to consider that impaction of the maxilla and counterclockwise rotation of the mandible can also affect the amount of decrease in the vertical height of the lower 1/3.

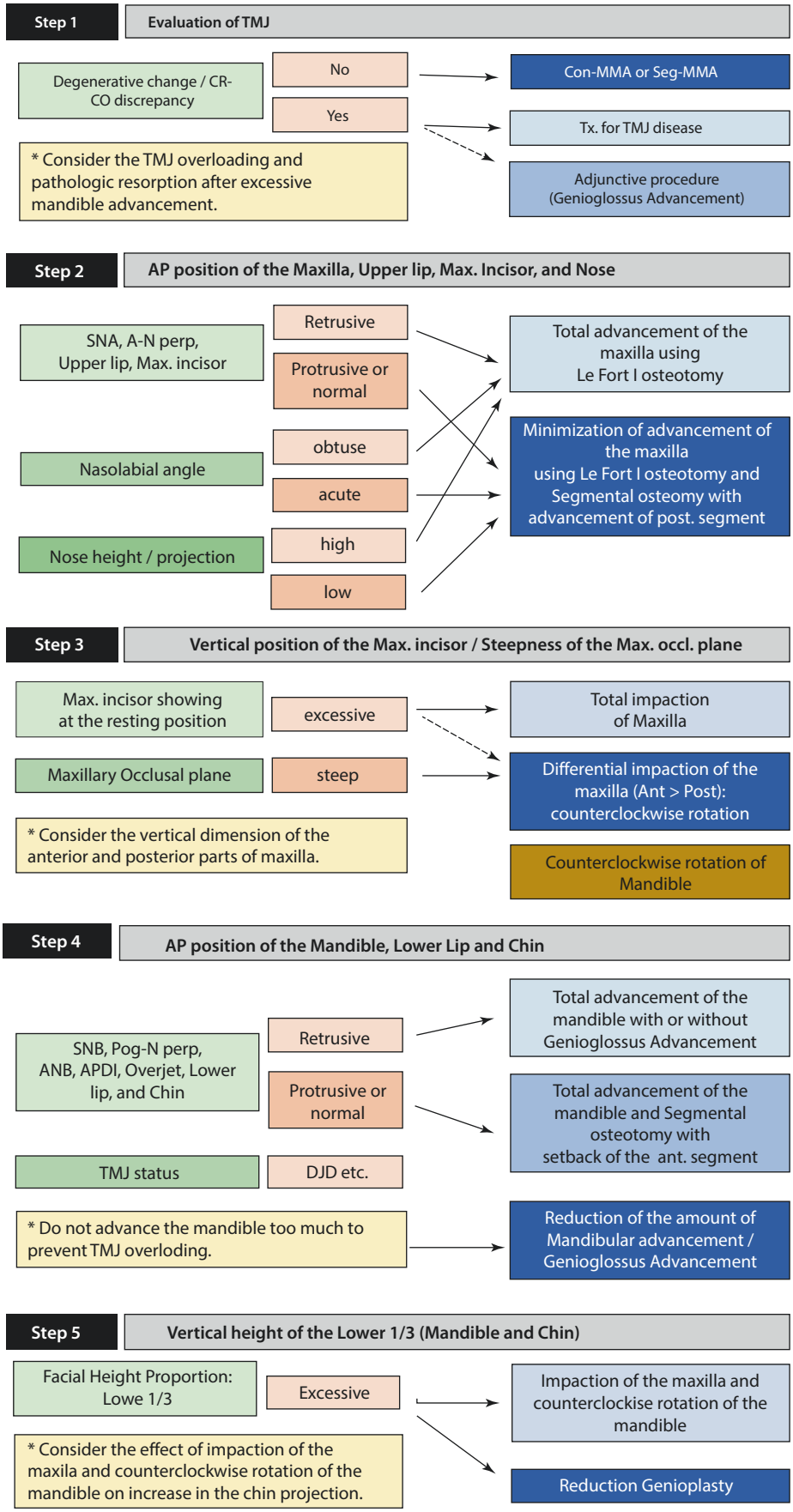


Fig. 22.6 Individualized flowchart for surgical treatment planning of con-MMA or seg-MMA for Asian adult OSAS patients

22.5.1 Worsening of the Symptoms of OSAS During Preoperative Orthodontic Treatment

If clinicians try to close the extraction spaces of the maxillary and mandibular first premolars with orthodontic treatment only, the tongue space can be decreased due to constriction of the dental arch during pre surgical orthodontic treatment [28]. For example, patient # 6 demonstrated worsening of the OSAS symptoms during 10 months of presurgical orthodontic treatment due to partial closure of the extraction space and narrowing of the tongue space. Therefore, in most seg-MMA cases of this study, surgery-first approach with minimal presurgical orthodontic treatment was performed (Table 22.1). Also, notice about worsening of OSAS symptoms during presurgical orthodontic treatment was given to the patients. The use of nCPAP can be recommended to the patients, who complained of worsening of OSAS symptoms during presurgical orthodontic treatment.

22.5.2 Proposition for Increasing the Success Rate

Holty and Guilleminault [18] suggested that younger age, lower preoperative AHI and BMI, and larger amount of maxillary advancement were positive predictors for the successful outcome of MMA. Liu et al. [21] reported 83.3% of success rate in 5–10 mm advancement cases, which was similar with the result of this study (87.5%; con-MMA group, $n = 4/4$ and seg-MMA group, $n = 3/4$). Although the surgical outcome was successful in the seg-MMA approach, several patients refused to take a postoperative PSG. Therefore, further investigation of the success rates of seg-MMA procedure with a larger sample size is needed in the future. In addition, it is necessary to accurately estimate the amount of airflow change per certain amounts of MMA using computational fluid dynamics, thus deciding an optimum amount of advancement satisfying both functional and esthetic aspects [3].

22.6 Conclusion

The seg-MMA procedure with or without counterclockwise rotation can effectively expand the upper airway by advancement of the posterior segment of the maxilla and the total mandible and obtain the facial esthetics by minimizing the forward movement of the maxilla and upper lip. Therefore, in adult OSAS cases with relatively forward positioned maxilla, acute nasolabial angle, and labioversed maxillary incisors, the seg-MMA procedure can be regarded as an effective alternative to the con-MMA procedure for simultaneous improvement of the respiratory function and facial esthetics.

Conflicts of Interest The authors declare no conflicts of interest and source of funding.

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Maxillomandibular Advancement for OSA

Contents

Clinical, Occlusal, and Cephalometric Analyses of the OSA Patient

Larry Wolford

Contents

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Patients with obstructive sleep apnea (OSA) may have upper airway obstruction and an associated dentofacial deformity contributory to OSA, commonly involving anteroposterior (A-P) and posterior vertical hypoplasia of the maxilla and mandible. There are three basic areas where upper airway obstruction can occur, which include the following: nasal cavity, oral cavity and jaws, as well as the oropharyngeal area. In addition, patients with OSA may have temporomandibular joint (TMJ) conditions and pathology that are co-existent with the dentofacial deformity, are the cause of the dentofacial deformity, or are a result of the preexisting dentofacial deformity. Patients with OSA require a comprehensive evaluation including clinical, radiology imaging, polysomnography, as well as any additional evaluations deemed necessary to finalize the diagnoses and establish an inclusive treatment plan to correct the OSA contributory factors. Many OSA patients with associated dentofacial deformities can benefit from corrective orthognathic surgical treatment. For patients to receive the state-of-the-art care in correction, the orthognathic team must be able to do the following: (1) correctly diagnose existing dentofacial deformities, areas of upper airway obstruction, and coexisting TMJ conditions if present, (2) establish an appropriate treatment plan, and (3) execute the recommended treatment. This chapter focuses primarily on clinical, radiology imaging, and dental model analyses, so important in diagnosing and treatment planning for the correction of OSA patients with dentofacial deformities.

The specific therapeutic goals for the OSA patient with coexisting dentofacial deformity requiring orthognathic surgery vary from patient to patient. These goals are directed toward the correction of specific musculoskeletal, dentoosseous, soft tissue deformities, and areas of airway obstruction, and associated TMJ pathologies. The specific therapeutic goals for the OSA patient may include the following:

1. Correct the dentofacial deformity and create optimal facial balance.
2. Establish a functional occlusion through normalization of the occlusal relationship, overbite, overjet, occlusal plane angulation, and transverse dimension.
3. Correct associated temporomandibular joint (TMJ) pathosis, dysfunction, and pain.
4. Correct nasal airway obstruction that may be related to narrow nostrils, constricted nasal valves, hyperplastic turbinates, nasal septal deviation, polyps, etc.
5. Correct oropharyngeal obstruction such as hyperplastic soft palate/uvula, enlarged tonsils, hyperplastic adenoid tissues, decreased oropharyngeal airway, etc.
6. Decrease or eliminate myofascial pain, TMJ pain, and headaches.

23.1 Patient Evaluation

Thorough evaluation and diagnosis is one of the most important aspects of overall patient management. Failure to recognize major functional and aesthetic problems may lead to compromise, complications, and unfavorable outcomes. OSA patient evaluation for corrective surgery may be divided into five main areas:

1. Patient concerns/chief complaints
2. Medical history
3. Clinical examination
4. Radiographic and imaging analysis
5. Dental model analysis

This diagnostic sequence may identify OSA patients who are candidates for orthognathic surgery and determine whether ancillary dental, medical, or other surgical procedures may be beneficial. Such patients may require further specialist evaluations for speech, audiometric, periodontal, general dental, psychological, neurological, ophthalmological, medical, otolaryngological, pulmonary, etc. or other concerns.

23.1.1 Patient Concerns

Not only do OSA patients have concerns about their sleep apnea and breathing difficulties, but they may also express concerns relative to their aesthetic appearance, difficulty eating, impaired jaw function, pain, headaches, etc. A patient's ultimate satisfaction with treatment outcome often depends on attention to the patient's concerns [1, 2]. An understanding of the patient's concerns, motivations, and expectations helps define treatment parameters and provides insight to the psychological health of the patient. Specific questions that may help identify the patient's chief concerns include the following:

1. What are your concerns or problems?
2. Have you had previous treatment for this condition, what was the treatment, and what was the outcome?
3. Why do you want treatment?
4. What do you expect from treatment?

This assessment of patient concerns helps develop a preliminary problem list and helps identify patients with unrealistic expectations. Patients need to understand treatment options, the anticipated outcomes, and the potential risks and complications. Accordingly, the surgeon and orthodontist must be careful not to mislead the patient into perceiving greater expectations than can be provided [3–5].

23.1.2 System-Oriented Physical Examination

Usually, orthognathic surgery is performed on reasonably healthy patients. However, OSA patients are often older and may have significant health issues that increase potential risks and complications. Presurgical evaluation, including medical and dental histories, physical examination, and appropriate laboratory studies are very important [6]. Obtaining an appropriate and current medical history may affect treatment planning and may help the surgeon avoid potentially life-threatening complications. Patient examination should rule out or identify patients with difficult airways, connective tissue or autoimmune diseases, bleeding disorders, or other pathological conditions that may preclude or modify surgery. OSA patients may have a tendency for obesity, high blood pressure, cardiac issues, pulmonary conditions, pain, allergies, nasal and sinus problems, endocrine issues, diabetes, etc.

23.1.3 Patient Preparation for Dentofacial Examination

The patient is evaluated best while sitting upright in a straight-backed chair with the examiner seated directly opposite at eye level. Generally, examine the patient with his or her pupillary plane parallel to the floor (Fig. 23.1a). Compensatory positioning may be appropriate for patients exhibiting orbital dystopia. The ear lobes can be used to establish a plane parallel to the floor. Orient the patient's head so that the clinical Frankfort horizontal plane (a line from the tragus of the ear to the bony infraorbital rim) is parallel to the floor (Fig. 23.1b). This is a reproducible position that mimics the natural head posture of most individuals with normal facial balance. This position may be used to obtain standardized measurements throughout the treatment sequence [7]. Patients with dentofacial deformities often develop alternative head postures for functional reasons particularly for OSA patients where they often tip the mandible and chin upward and forward as well as posture the head in a head postured forward neck position, which opens the oropharyngeal airway, improving the patients breathing abilities. Adjustment for such compensatory head posture is important during clinical, radiographic, and photographic evaluation by orienting the clinical Frankfort horizontal plane parallel to the floor [8]. Following surgical orthodontic correction, the OSA patient's "natural" head posture often reverts to a more normal position because functional and aesthetic compensations are no longer necessary to maintain the airway. Selecting a standardized and reproducible head position aids in proper diagnosis and evaluation of posttreatment results.

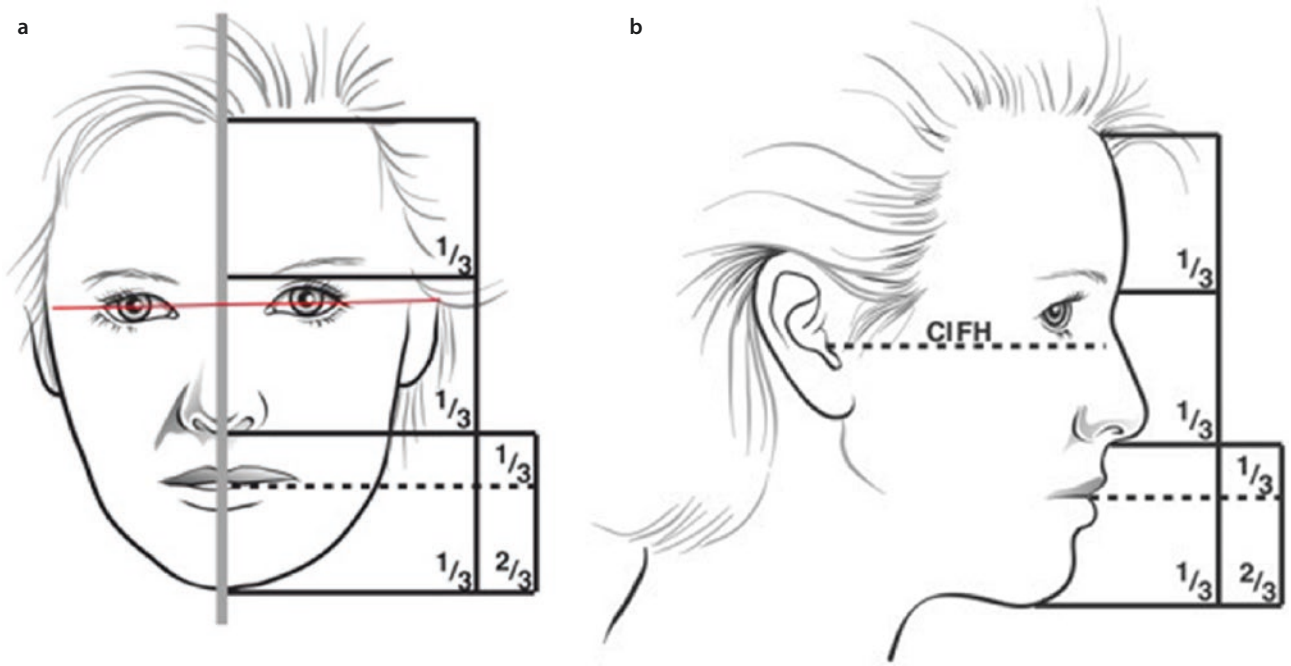


Fig. 23.1 **a** Vertically, the face can be divided into equal thirds for assessment. The lower third of the face can be divided into thirds with the distance from subnasale to upper lip stomion equaling one-third, and lower lip stomion to soft tissue menton equaling two-thirds. This ratio provides optimal vertical facial balance in the lower third of the face. **b** In profile, the face is divided in the same manner. Head orientation is important, with the clinical Frankfort horizontal plane oriented parallel to the floor. Clinical Frankfort horizontal plane is a line from the tragus of the ear to the bony infraorbital

Once the head is oriented properly, seat the mandibular condyles in the glenoid fossae with the teeth lightly touching together (centric relation). Although it is important to evaluate centric occlusion, perform the definitive clinical examination relative to surgical orthodontic diagnosis and treatment planning with the patient in centric relation. Failure to evaluate in centric relation may result in a misdiagnosis or incomplete diagnosis, inappropriate or compromised treatment plan, and unacceptable or compromised treatment outcome.

For proper evaluation, the patient's lips should be relaxed and not forced together. This relaxed lip posture allows evaluation of vertical facial height and the morphology and drape of the soft tissues. Relaxation of the lips allows evaluation of upper lip length, tooth-to-lip measurements, possible lip incompetence, and coincidence of the facial, dental, and chin midlines. Combined with mentalis muscle relaxation, lip relaxation also allows evaluation of the chin position and the presence or absence of skeletal abnormalities such as vertical maxillary excess or vertical maxillary deficiency. The lip posture frequently is overclosed in patients with vertical maxillary deficiency.

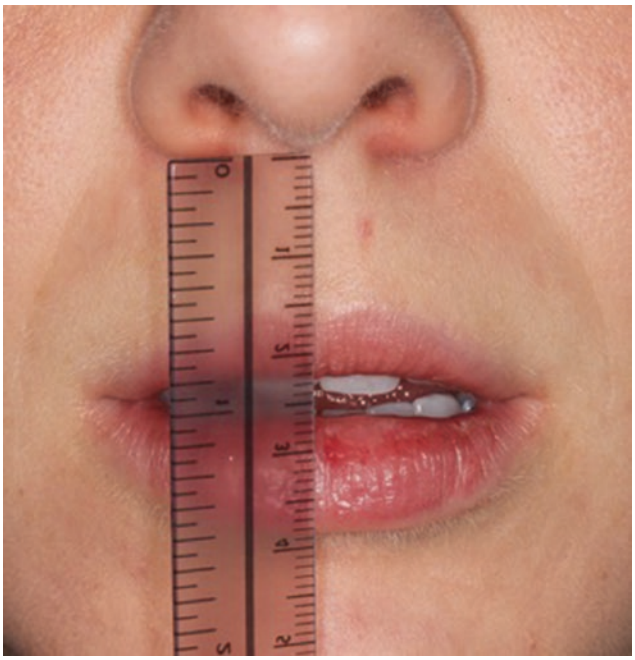


Fig. 23.2 Upper lip length is measured from subnasale to upper lip stomion. For males, the normal value is 22 ± 2 mm, and for females, 20 ± 2 mm

23.1.4 Facial Evaluation

We have previously described in detail, the comprehensive method for facial evaluation [9]. In this section, we will describe those factors more commonly associated with OSA patients, focusing on the deformities associated with the lower third of the face that are the primary factors that can contribute to OSA. For vertical facial analysis, the face is divided into equal thirds (Fig. 23.1a). The upper facial third extends from the hairline to glabella. The middle third extends from glabella to subnasale. The lower third extends from subnasale to soft tissue menton. Orthognathic surgery most commonly alters the lower third of the face, with some influence on the middle third. In addition to this vertical analysis, pretreatment facial evaluation also should address the frontal and lateral facial planes. Evaluation from the frontal view of the lower third face should include the following seven important anatomical relationships:

1. The upper lip length: The distance from subnasale to upper lip stomion in a relaxed state. The normal upper lip length is 22 ± 2 mm for males and 20 ± 2 mm for females (Fig. 23.2).
2. Tooth to upper lip relationship: The distance form the incisor edge to the upper lip with the lip in repose. The normal distance is 2.5 ± 1.5 mm (Fig. 23.3).
3. Midlines: The midlines of the face, nose, lips, dentition, and chin should be congruent, and the face should be reasonably symmetrical, vertically and transversely (Figs. 23.4 and 23.5).
4. Lips overclosed: If the patient's lips are overclosed, rotate the mandible open until the lips just begin to separate. The condyles should remain seated in centric relation. Then evaluate the true lip length and the tooth-to-lip relationship.
5. Smile: The smile is frequently one of the patient's chief concerns. When smiling, the vermilion of the

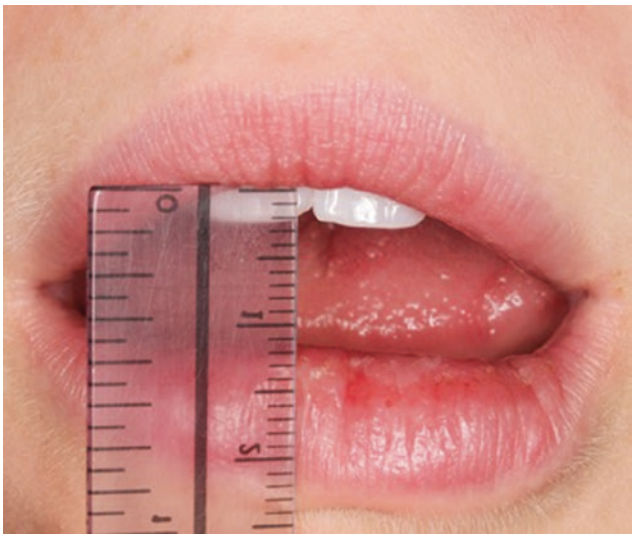


Fig. 23.3 The normal upper tooth-to-lip relationship is 2.5 ± 1.5 mm

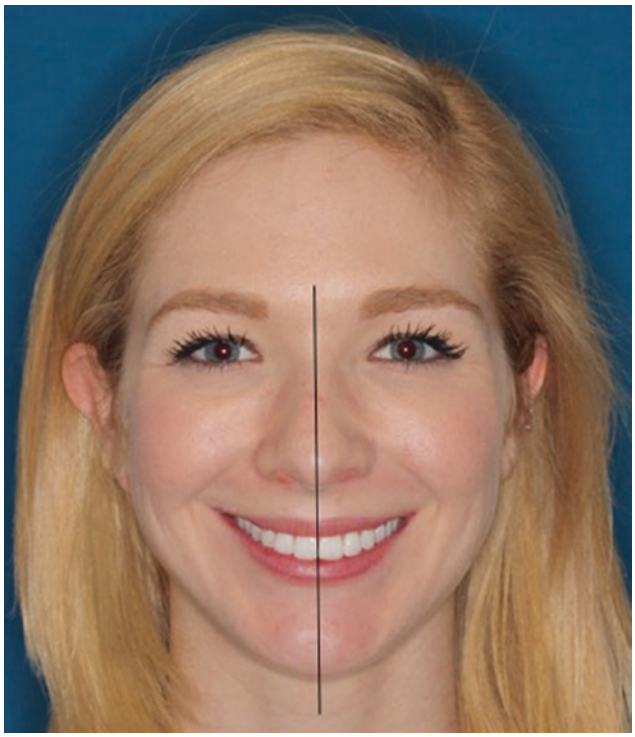


Fig. 23.4 The facial midlines are assessed, including the nasal, maxillary and mandibular dental midlines, and the chin midline, relative to the facial midline. Left to right facial symmetry also is evaluated

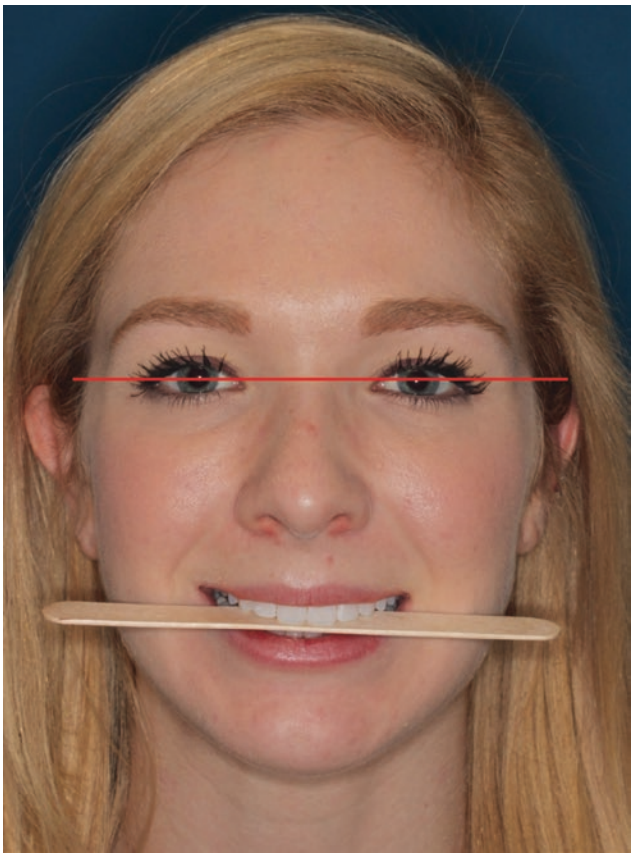


Fig. 23.5 Transversely, the occlusal plane should parallel the pupillary plane, providing there is no orbital dystopia

upper lip should fall at the cervicogingival margin with no more than 1–2 mm of exposed gingiva (Fig. 23.4). In addition to this relationship, surgical decisions also must consider the tooth-to-lip relationship with the lips in repose, because many factors may influence lip posture during animation. The amount of upper lip elevation during smiling may be affected by the following:

(a) Anteroposterior position of the maxilla and mandible in relation to the cranial base as well as to each other.
(b) Amount of overjet and overbite.
(c) Angulation of the anterior teeth and dentoalveolus.
(d) Occlusal plane angulation.
(e) Clinical crown length.
(f) Neuromuscular function of the lips.
(g) Dental coverage of periodontium.

Each of these factors may contribute to inaccuracies in the determination of the proper maxillary vertical position if this position is determined only by evaluation of the tooth-to- lip position during smiling.

6. Facial height balance: The distance from glabella to subnasale and subnasale to menton should be a 1:1 ratio, providing that the upper lip length is normal (Fig. 23.1a).
7. Lower third facial balance: The length of the upper lip should be one-third of the length of the lower facial third; that is, lower lip stomion to soft tissue menton should be twice the vertical dimension of the upper lip, providing that the upper lip is normal in length (Fig. 23.1a).

23.1.5 Lateral View

Evaluation of the lateral facial view is usually the most valuable assessment in determining vertical and anteroposterior problems of the jaws:

1. Facial height balance: The distance from glabella to subnasale and from subnasale to soft tissue menton should be in a 1:1 ratio if the upper lip length is normal (see Fig. 23.1b).
2. Facial morphology: Evaluate the morphology and relationships of the nose, lips, cheeks, and chin to each other.
3. Evaluate the cervicomandibular angle in reference to the chin position.
4. The length of the upper lip should be one-third of the length of the lower facial third; that is, lower lip stomion to soft tissue menton should be twice the

vertical dimension of the upper lip if the upper lip is normal in length (see Fig. 23.1b).
5. Upper lip projection: The upper lip labrale superius should be 1–3 mm anterior to the subnasale perpendicular plane; a line through subnasale perpendicular to the clinical Frankfort horizontal plane (Fig. 23.6).
6. Chin-neck line and angle: The distance from soft tissue pogonion to the chin-neck angle. Although there is a wide variation in this dimension, the normal measurement is around 65 ± 5 mm for males and 55 ± 5 mm for females (Fig. 23.6, green reference lines).

23.1.6 Oral Examination

Oral examination helps identify functional and aesthetic deformities of the dentoosseous and soft tissue structures. Occlusal and dental factors to be evaluated in the oral examination are discussed under Dental Model Analysis. The factors oral examination should evaluate the following issues:
1. Occlusal relationship (Class I, II, or III)
2. Anterior overbite or open bite, overjet, crossbites
3. Tooth size discrepancies, dental crowding or spacing
4. Curve of Wilson, curve of Spee
5. Missing, decayed, retained primary, nonsalvageable teeth
6. Discrepancies between centric occlusion and centric relation
7. Periodontal evaluation
8. Transverse, anteroposterior, or vertical asymmetries
9. Anatomical or functional tongue abnormalities
10. Any masticatory difficulties and dysfunctions
11. Any other pathological processes

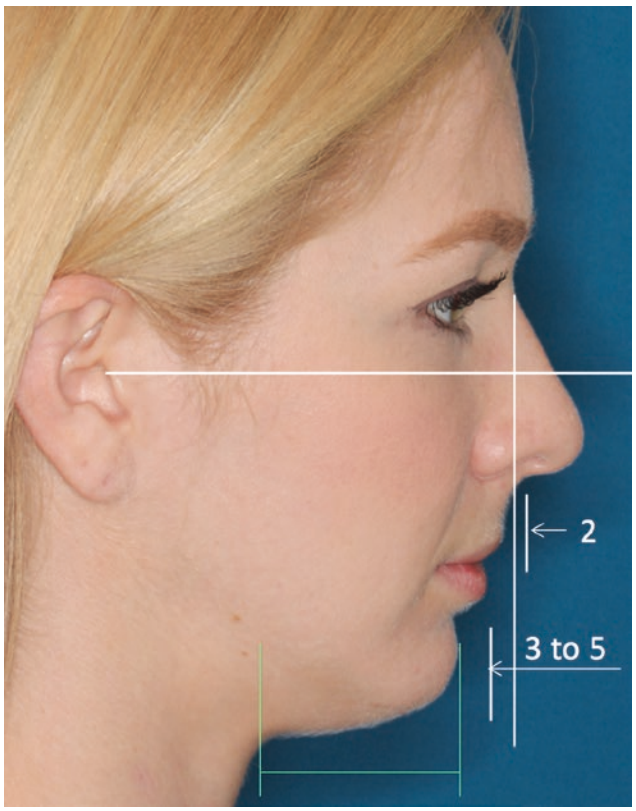


Fig. 23.6 A line perpendicular to the clinical Frankfort horizontal plane (ClFH) through subnasale (A) should be 3 ± 3 mm anterior to the chin in males and 5 ± 3 mm in females. Upper lip stomion should be 2 ± 2 mm anterior to the subnasale perpendicular plane

23.1.7 Periodontal Evaluation

There are several periodontal factors that should be evaluated before orthodontic treatment and orthognathic surgery. Patients with preexisting periodontal disease or gingivitis have an increased risk of disease exacerbation during orthodontic treatment and posttreatment, particularly in areas where interdental osteotomies may be required [10–16]. Factors that can affect periodontal health adversely in relation to orthognathic surgery include smoking, excessive alcohol or caffeine consumption, bruxism and clenching, connective tissue/autoimmune diseases, diabetes, malnutrition, and so on [11]. Address all periodontal diseases before orthodontics and orthognathic surgery.

Inadequate attached gingiva, most frequently associated with the mandibular anterior teeth, may contribute to the development of periodontal problems such as gingival retraction, tooth sensitivity, and bone loss (Fig. 23.7). In areas of inadequate attached gingiva, consider gingival grafting. When indicated, perform gingival grafting before the initiation of orthodontics. Providing adequate attached gingival tissue before orthodontic and orthognathic surgical intervention protects this tissue and minimizes gingival tissue retraction.

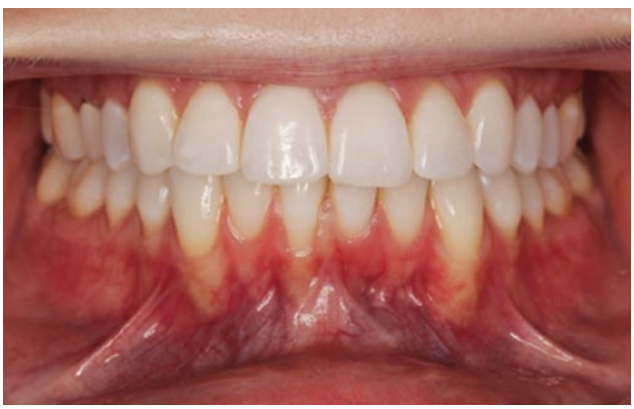


Fig. 23.7 Healthy attached gingiva should be present around the teeth. This patient shows lack of attached gingiva around the anterior teeth. If orthodontics or surgery performed in this area, periodontal problems can develop if this issue is not appropriately addressed prior to treatment

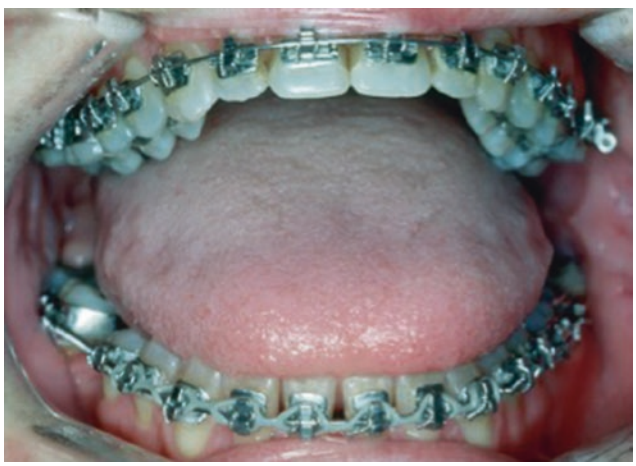
23.1.8 Tongue Assessment

An enlarged tongue can cause dentoskeletal deformities, instability of orthodontics and orthognathic surgical treatment, and can create masticatory, speech, and airway management problems. The condition can be defined as true macroglossia or pseudomacroglossia. True macroglossia indicates an increased disproportionate size of the tongue commonly caused by (1) muscular hypertrophy (◘ Fig. 23.8), (2) glandular hyperplasia, (3) hemangioma, or (4) lymphangioma. A number of congenital and acquired causes of true macroglossia exist, including conditions such as Down syndrome and Beckwith-Wiedemann syndrome. Acquired factors may include acromegalia, myxedema, amyloidosis, tertiary syphilis, cysts or tumors, and neurological injury [17]. Pseudomacroglossia is a condition in which the tongue may be normal in size, but it appears large in relation to its anatomical interrelationships, such as maxillary and mandibular hypoplasia, where the oral cavity volume is diminished. In either true or pseudomacroglossia, the relative excessive size of the tongue can contribute significantly to OSA and surgical correction may need to be considered. Patients with true macroglossia may be candidates for reduction glossectomy. Specific clinical and cephalometric features may help the clinician identify the presence or absence of macroglossia [17].

Pseudomacroglossia is commonly associated with maxillary and mandibular A-P hypoplasia and can usually be corrected by counterclockwise rotation-advancement of the maxillomandibular complex, increasing the oral cavity volume providing increased intraoral space to accommodate the tongue.

Most open bites are not related to macroglossia. In fact, it has been established that closing open bites with orthognathic surgery allows a normal tongue, which is an adaptable organ, to readjust to the altered volume of the oral cavity, with little tendency toward relapse [18, 19]. If true macroglossia is present with an anterior open bite, then instability of the orthodontics and orthognathic surgery are likely to occur, with a tendency for the open bite to return.

Although the indications for reduction glossectomy are few, when the procedures are indicated for true macroglossia, the following conclusions can be drawn: (1) reduction glossectomy can improve functional and aesthetic outcomes significantly, (2) the anterior resection combined with the midline keyhole type procedure is the best technique, (3) improved function relative to airway, speech, and mastication can be anticipated, and (4) if the excessively large tongue is causing significantly unfavorable mandibular growth, reduction of the tongue may help control the problem [17–19].



◘ **Fig. 23.8** Macroglossia indicates an enlarged tongue relative to the size of the oral cavity, and can contribute to malocclusion, speech and swallowing difficulties, and oropharyngeal airway obstruction contributing to sleep apnea

23.1.9 Temporomandibular Joint

The TMJs provide the foundation for orthognathic surgery. OSA patients commonly have TMJ conditions that may require surgical intervention to provide outcome stability for those patients requiring orthognathic surgery to correct the OSA (◘ Fig. 23.9). Presurgical TMJ dysfunction or undiagnosed TMJ pathosis can result in orthognathic surgery unfavorable outcomes such as postoperative pain, condylar resorption, condylar over-growth, malocclusion, jaw dysfunction, and relapse resulting in recurrence of the facial deformity and sleep apnea issues [20, 21]. Common TMJ conditions and treatment are addressed in ▶ Chaps. 23 and 27. Assess the TMJs before treatment and periodically throughout the treatment. Basic TMJ factors to consider include the following:

1. The patient history may reveal: TMJ pain, dysfunction, clicking and popping, crepitation, limited opening, difficulty chewing, headaches, ear problems, myofascial pain, progressive development of recession of the mandible and open bite, shifting of the mandible and bite, neck and shoulder pain problems, and so on. Document etiological factors, time of onset, signs and symptoms, previous treatments and outcomes, symptom frequency and duration parafunctional habits, and other modifying factors.
2. Identify or rule out polyarthritides or other systemic conditions. These conditions may include connective tissue or autoimmune diseases such as rheumatoid arthritis, juvenile idiopathic arthritis, systemic lupus erythematosus, scleroderma, sarcoidosis, reactive arthritis, psoriasis, psoriatic arthritis, Sjögren's disease, ankylosing spondylitis, Reiter syndrome, etc. (◘ Fig. 23.9c) [20, 21].

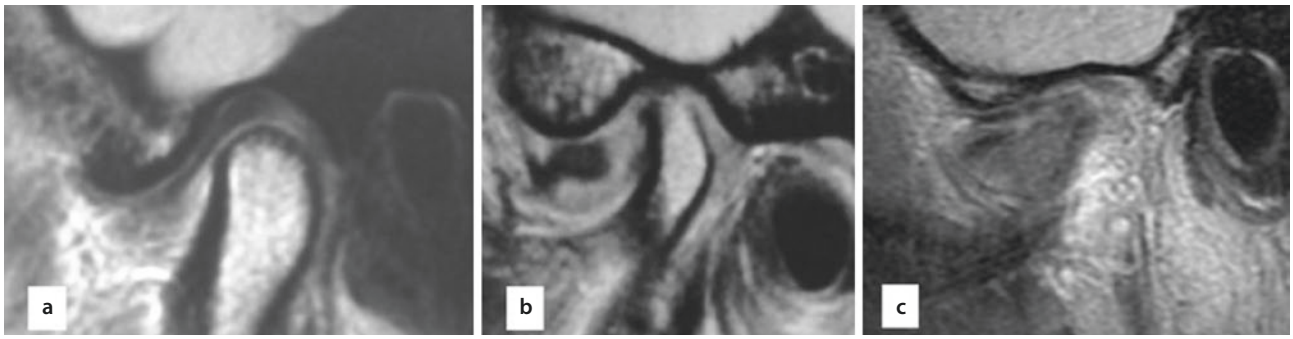


Fig. 23.9 MRI imaging of TMJs. **a** Normal condyle and disc in fossa. **b** TMJ arthritis and anteriorly displaced articular disc that is significantly deformed. **c** TMJ with juvenile idiopathic arthritis (JIA), with significant loss of condylar vertical height, although the disc is in normal position, but surrounded by a reactive pannus that is causing bone and disc destruction

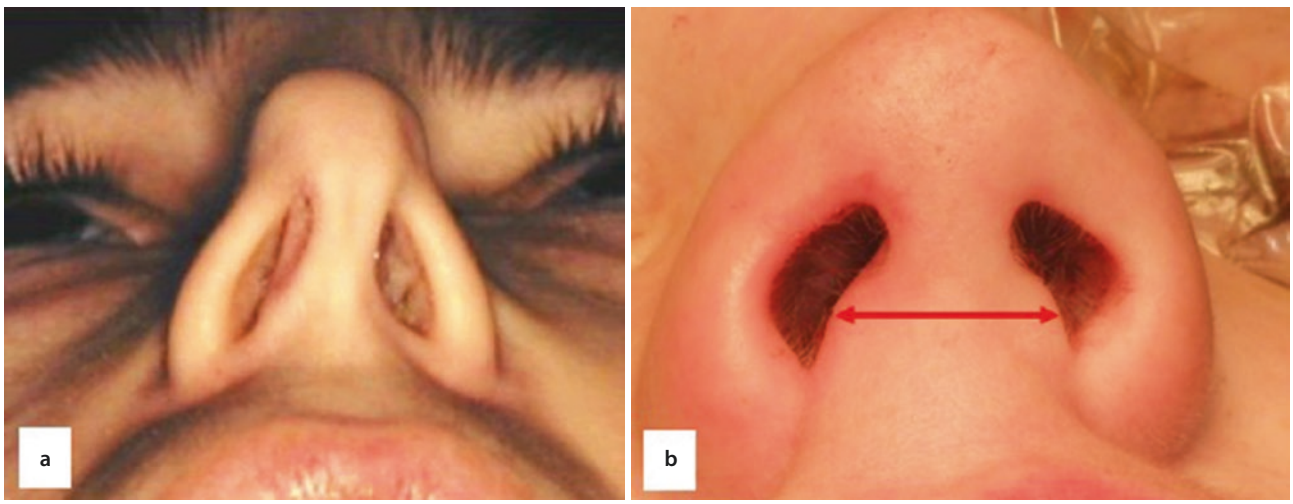


Fig. 23.10 **a** Severe constricture of the nostrils and nasal valves. **b** Wide columella creating narrow nostrils. Narrow nostrils will restrict air from passing into the nose and is the first component of nasal airway obstruction

3. Clinical examination should assess pain, function, and joint noise. Deviation of the mandible during opening, for example, may indicate a unilateral closed lock or fibrous ankylosis. Joint noises such as clicking and popping may suggest articular disk displacement. Crepitation within the joint may indicate osteoarthritis or perforation of the retrodiscal tissues.
4. Obtain imaging to assess the TMJs such as cone beam imaging, panoramic radiographs, transcranial radiographs, transpharyngeal radiographs, tomograms, computed tomography (CT) scans, magnetic resonance imaging (MRI) (Fig. 23.9), (see ▶ Chaps. 23), and other imaging modalities as indicated.
5. Properly diagnose existing TMJ conditions and discuss them with the patient. The surgeon should properly sequence and plan treatment for conditions requiring correction. Inform the patient of any abnormal TMJ findings and how such conditions may influence the orthodontic and orthognathic surgery outcome, even if these conditions do not require intervention.
6. If preexisting TMJ pathologies or conditions are present in patients requiring orthognathic surgery for correction of OSA, consideration must be made for surgical correction of the TMJ pathologies in a preliminary surgery or concomitantly with the required orthognathic surgery.

23.1.10 The Nose

There are a number of anatomical factors that can contribute to nasal airway obstruction including the following: (1) narrow nostrils, (2) wide columella (Fig. 23.10), (3) constriction of luminal (nasal) valves, (4) transverse collapse of nose, (5) deviated nasal septum, (6) hypertrophied turbinates (Fig. 23.11), (7) nasopharyngeal adenoid tissue (Fig. 23.12), and (8) other anatomical

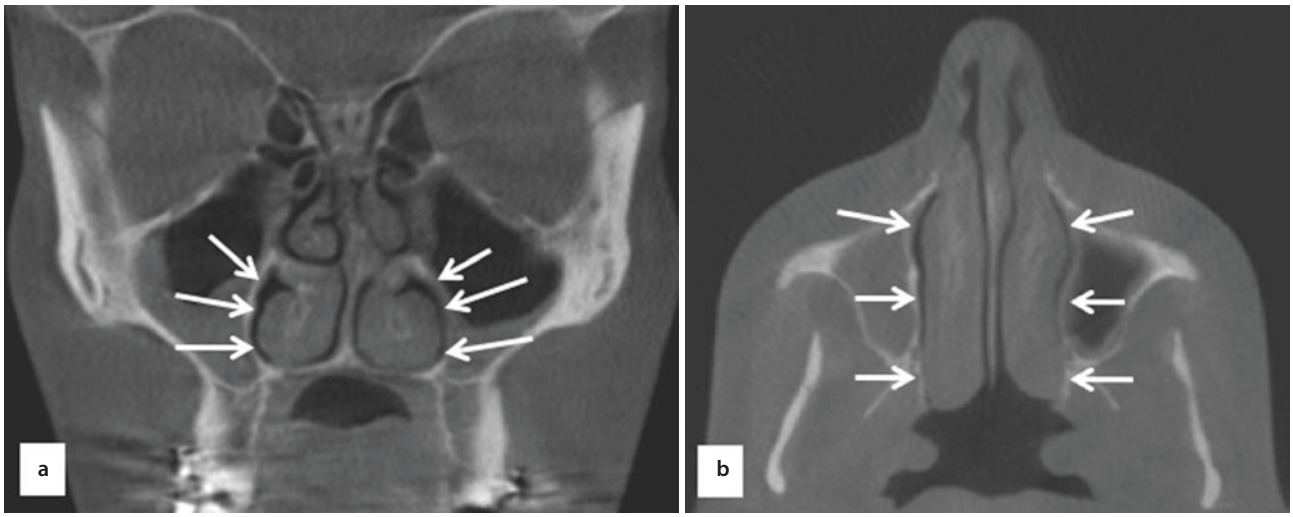


Fig. 23.11 **a** Coronal tomographic view of hyperplastic nasal turbinates with almost total obliteration of the nasal airway. **b** Axial view of bilateral hyperplastic turbinates, creating nasal airway obstruction

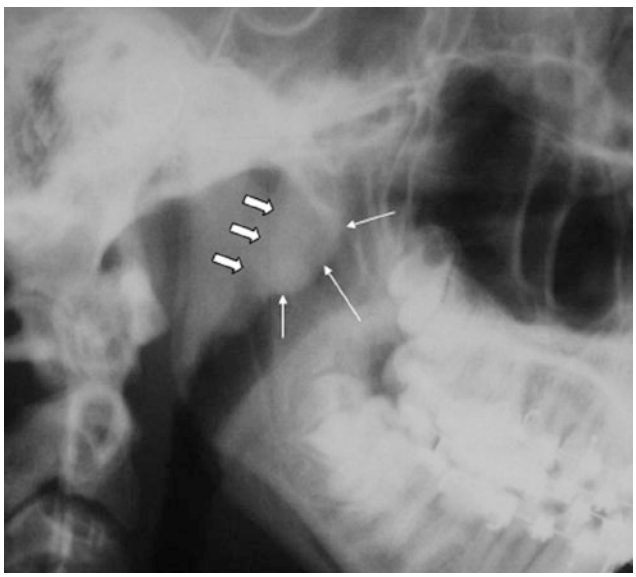


Fig. 23.12 Hyperplastic nasopharyngeal adenoid tissue creating oropharyngeal airway obstruction

variances and pathologies [22]. Clinical, radiographic, fiberoptic scoping, etc. are methods for evaluating nasal airway obstruction. Evaluation begins by assessing the nasal width of the nostrils and columella (Fig. 23.10). A nasal speculum can be used to evaluate the luminal nasal valves inside the nose and to evaluate the anterior aspect of the turbinates and nasal septum. The most common forms of nasal obstruction are hyperplastic nasal turbinates (Fig. 23.11). The turbinates are a honeycombed bony structure covered with glandular and erectile tissues covered with ciliated mucosa. When these turbinates are enlarged, and/or septum is deviated or spurring is present, this can cause significant nasal airway obstruction. Usually, laying down at night will cause increased swelling of the turbinates, further blocking off the functional airway. Allergic rhinitis is a common contribution to nasal obstruction. Hypertrophied turbinates are the most common factor causing nasal airway obstruction [23]. A deviated nasal septum also can cause significant obstruction as well as bending the nose off to one side or the other or with septal spurs that may be present. Nasopharyngeal adenoid tissue can cause a major blockage of the nasopharynx and posterior nasal choanae (Fig. 23.12). This can be a problem in young kids, although adenoid tissues are usually resorbed by the age of 14, but may remain present in some patients for many years longer.

Upper airway obstruction involving the nose and oropharyngeal areas, can have significant adverse effects on facial growth and development when occurring in growing children creating an increased vertical facial growth pattern with downward and backward growth vectors for the maxilla and mandible. Upper airway obstruction can affect the health and well-being of children and adults afflicted with this anatomical variance of obstruction and can create functional and aesthetic facial, skeletal, muscular, and dental imbalances.

23.1.11 Oropharyngeal Airway Assessment

Common factors that contribute to airway obstruction of the oropharyngeal area include the following: (1) mandibular and maxillary hypoplasia displacing the tongue posteriorly, (2) elongated (hyperplastic) soft palate, (3) hyperplastic uvula (Fig. 23.13), (4) constriction of the fascial pillars, (5) hypertrophied tonsils and adenoids (Fig. 23.12), (6) decreased oropharyngeal airway, (7) tumors or other pathologies decreasing the oropharyngeal airway, and (8) pharyngeal flaps (in

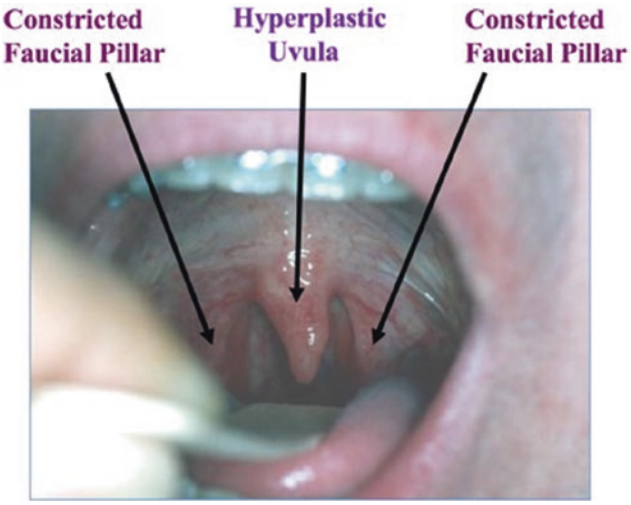


Fig. 23.13 Hyperplastic soft palate and uvula are observed, and can contribute to oropharyngeal airway obstruction. There can also be a transverse constricture of the faucial pillars, further contributing to OSA

cleft palate patients) [22]. Clinical assessment of the oropharyngeal airway included evaluation of the Mallampatti score, length of the soft palate and uvula as well as function, transverse width and function of the fascial pillars, presence and size of the tonsils, indirect evaluation of the adenoid tissues, etc. A high Mallampatti score (Class III and IV) can indicate retrusion of the mandible and a high occlusal plane facial morphology with associated OSA. When the oropharyngeal airway is significantly reduced in the presence of a hypoplastic mandible and maxilla, this may indicate the requirement for orthognathic surgery for maxillomandibular complex advancement with or without counterclockwise rotation. A hyperplastic soft palate and uvula (Fig. 23.13) can contribute significantly to oropharyngeal airway obstruction acting as a valve blocking the nasal airway. An enlarged uvula can act as a vibrating structure contributing to snoring, as can an elongated flaccid soft palate. Evaluation of the soft palate/uvula length in conjunction with the lateral cephalogram and 3-D soft tissue imaging of the oropharyngeal area may indicate the need for an uvulopalatopharyngoplasty (UPPP) procedure if the structures are hyperplastic.

Hypertrophied tonsils and adenoid tissue (Fig. 23.12) also can contribute significantly to oropharyngeal airway obstruction. Particularly those that suffer from recurrent infections can cause further enlargement making it difficult to breathe through the nose or mouth. Hypertrophied tonsils and hypertrophied adenoid tissues often go together, providing a significant mechanical obstruction. Evaluation of the tonsils and adenoid tissues as contributory factors to OSA could indicate the need for a tonsillectomy and adenoidectomy [22].

23.2 Radiographic Evaluation

23.2.1 Types of Imaging Techniques

Cone beam technology provides a 1:1 ratio of imaging with panographic, cephalometric and tomographic imaging, including 3-D imaging, and is currently the gold standard for orthognathic surgery imaging. Other commonly used radiographs for diagnosis of dentofacial deformities are (1) lateral cephalometric radiograph, (2) panoramic radiograph, and when indicated, (3) periapical radiograph. Panoramic and periapical radiographs can be helpful to determine tooth alignment, root angulation, and existing pathoses. Other imaging modalities such as posteroanterior cephalograms, TMJ tomograms, transcranial radiographs, Water's view images, CT scans, and MRI (Fig. 23.9) may be required as determined by individualized patient diagnostic needs.

23.2.2 Lateral Cephalometric Radiograph

The lateral cephalometric radiograph is one of the most important tools in the diagnosis of jaw deformities [9]. The lateral cephalometric radiograph is used to analyze skeletal, dentoalveolar, and soft tissue relationships in the anteroposterior and vertical dimensions. For proper head positioning for lateral cephalometric acquisition, pose the patient's head so that the jaws are in centric relation with the teeth lightly touching and the lips relaxed. Position the head so that the clinical Frankfort horizontal plane (line from tragus of the ear through the bony infraorbital rim) is parallel to the floor. Both hard and soft tissue structures should be visible on the radiograph. If the patient's bite is overclosed (such as in vertical maxillary deficiency), then take a second lateral cephalometric radiograph with the condyles still seated in centric relation but the mouth opened until the lips just begin to separate. This posture allows assessment of soft tissue and bony structures without distortion of the lips. Anteroposterior cephalometric radiographs may be helpful, particularly in diagnosing and treatment planning for patients with significant transverse asymmetries.

23.2.3 Cephalometric Analysis Versus Clinical Diagnosis

Numerous cephalometric analyses are available to evaluate lateral cephalometric radiographs. Regardless of the specific analysis the clinician uses, it is important to understand that there may be significant differences between the clinical evaluation and the values obtained from cephalometric analysis. When a significant differ-

ence occurs, the clinical evaluation is far more important for treatment planning [9]. Cephalometric analysis is only an aid to clinical assessment and should not be used as the sole diagnostic tool.

23.2.4 Corrected Frankfort Horizontal Plane

In cases in which the cephalometric values do not correlate with the clinical impression, make adjustments in the reference cranial base structures (i.e., corrected Frankfort horizontal line) [9, 24]. Adjust values to correlate with the clinical impression for use in diagnosis and treatment planning (Fig. 23.14). The Frankfort horizontal plane may be positioned aberrantly because of vertical malposition of porion or orbitale and/or anteroposterior malposition of nasion. The anatomical landmarks for Frankfort horizontal plane also may be difficult to locate because of difficulty in the radiographic identification of porion and orbitale. A corrected Frankfort horizontal plane to correlate the cephalometric values for maxillary and mandibular AP positions with the clinical impression provides a cephalometric analysis that assists in diagnosis and treatment planning (Fig. 23.14). Cephalometric analysis tempered with good clinical judgment can be a valuable tool in establishing the most appropriate orthodontic and surgical treatment plan.

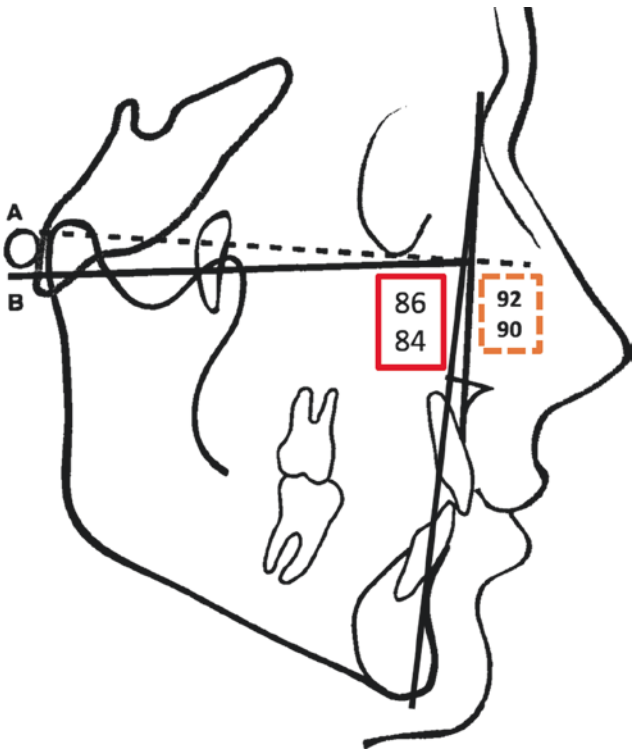


Fig. 23.14 (A) Cephalometric numerical values based on standard anatomical landmarks may not correlate to the clinical impression or the patient's deformity. Using the anatomically defined Frankfort horizontal plane (dotted line A), the cephalometric values for maxillary depth and mandibular depth (orange rectangle) do not correlate with the clinical assessment of this patient. In such instances, a corrected Frankfort horizontal plane (CFH) can be constructed (solid line B) so that the numerical cephalometric values (red solid rectangle) correlate with the clinical diagnosis of the patient. Subsequently, normal cephalometric values based on the CFH can be used in the diagnosis, treatment planning, and development of a surgical prediction tracing

23.2.5 Cephalometric Analysis

Many reasonable cephalometric analyses are available for clinical decision-making [25]. The author uses an analysis that evaluates 14 cephalometric relationships. This analysis permits a rapid diagnostic assessment as follows:

1. Maxillary depth: The angle formed by the Frankfort horizontal plane and a line from nasion through point A (NA line). The normal value is 90 ± 3 degrees (Fig. 23.15, angle A).
2. Mandibular depth: The angle formed by the Frankfort horizontal plane and a line from nasion through point B of the mandible (NB line). The normal value is 88 ± 3 degrees (Fig. 23.15, angle B).
3. Mandibular plane angle: The angle formed by the Frankfort horizontal plane and a line from the menton through the gonion. The normal value is 25 ± 5 degrees (Fig. 23.15, angle C).
4. Occlusal plane angle: The angle formed by the Frankfort horizontal plane and a line drawn tangent to the buccal groove of the mandibular second molars through the cusp tips of the premolars. The normal value is 8 ± 4 degrees. The occlusal plane has significant influence on function and aesthetics, particularly when double jaw surgery is performed (Fig. 23.15 angle D).
5. Aesthetic line (Fig. 23.15, red E and line): A line tangent to the labial surface of the maxillary central incisors extended vertically to cross the Frankfort horizontal plane and should form a 90 degree angle when ideally aligned. This places the central incisor crown in the best aesthetic position.
6. Upper incisor angle: The angle formed by the long axis of the maxillary incisor to the NA line. The normal value is 22 ± 2 degrees. The labial surface of the incisor tip should be 4 ± 2 mm anterior to the NA line. Upper incisor angulation is important in establishing the presurgical orthodontic goals (Fig. 23.16, angle A and linear line B).
7. The lower incisor angle: The angle formed by the long axis of the mandibular incisor to the NB line. The normal value is 20 ± 2 degrees. The labial sur-

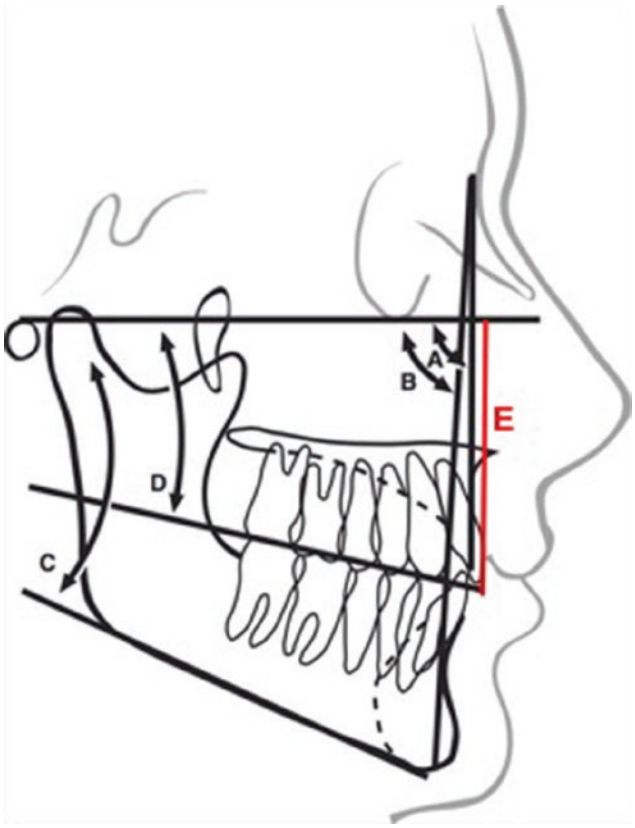


Fig. 23.15 A normal maxillary depth (A) is 90 ± 3 degrees. The normal mandibular depth (B) is 88 ± 3 degrees. The normal mandibular plane angle to Frankfort horizontal plane (C) is 25 ± 5 degrees. The normal occlusal plane angle (D) is 8 ± 4 degrees. The normal aesthetic line (red E and line) is constructed tangent to the labial surface of the maxillary central incisors and should create a 90 ± 2 degrees angle with Frankfort horizontal plane for best aesthetic positioning of the maxillary central incisors

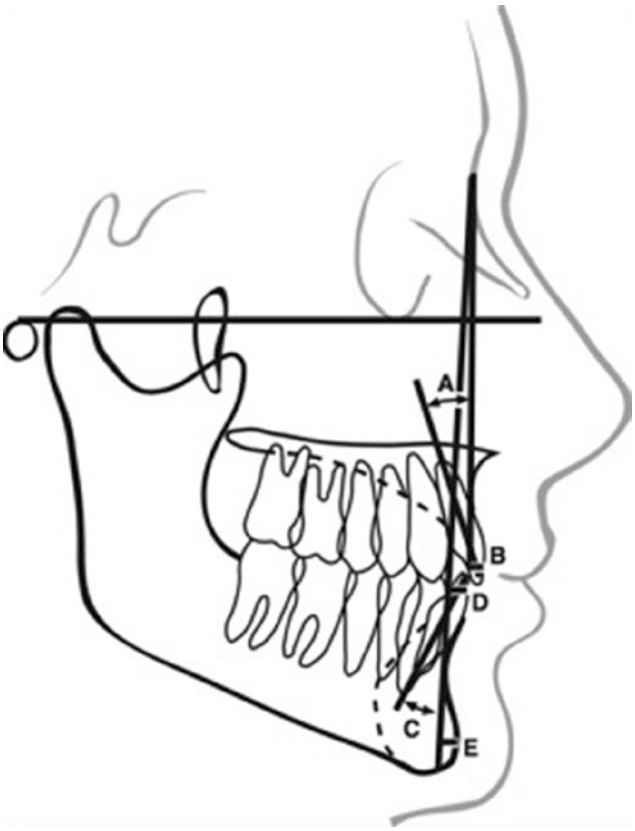


Fig. 23.16 The long axis from the upper incisor to the NA line (A) has a normal value of 22 ± 2 degrees. The labial surface of the upper incisor (B) should be 4 ± 2 mm anterior to the NA line. The long axis of the lower incisor to the NB line (C) has a normal value of 20 ± 2 degrees. The labial surface of the mandibular central incisors (D) should be 4 ± 2 mm anterior to the NB line. Hard tissue pogonion (E) should be 4 ± 2 mm anterior to the NB line with a 1:1 ratio, with the position of the labial surface of the mandibular central incisors anterior to the NB line

face of the incisor tip should be 4 ± 2 mm anterior to the NB line. Assessment of the lower incisor angulation is important in determining the presurgical orthodontic goals (Fig. 23.16, angle C and linear line D).

8. Pogonion projection: The distance from the most protrusive point of bony pogonion to the NB line. The normal value is 4 ± 2 mm. Optimal mandibular dentoskeletal balance is achieved when the labial surface of the lower incisors and pogonion are in a 1:1 ratio anterior to the NB line (Fig. 23.16 linear line E).
9. Upper lip length: The distance from the base of the nose (subnasale) to the inferior part of the upper lip (upper lip stomion). The normal length of an adult male lip is 22 ± 2 mm. For a female, it is 20 ± 2 mm. Upper lip length is the basis for establishing vertical facial dimensions in the lower third of the face because the upper lip length usually is not altered easily. This measurement is the basis for establishing the vertical length of the lower two-thirds of the lower third of the face (Fig. 23.17, distance A).
10. Upper tooth-to-lip relationship: The distance from the relaxed upper lip stomion to the incisal edge of the upper incisor. The normal value is 2.5 ± 1.5 mm. This evaluation is important in establishing the vertical dimensions of the face, particularly when there are vertical dysplasias present in the maxilla (Fig. 23.17, distance B).
11. Lower anterior dental height: The distance from the lower incisor tip to hard tissue menton. The lower anterior dental height for a male is 44 ± 2 mm, and for a female is 40 ± 2 mm. For optimal balance in the lower third of the face, the lower anterior dental height should be approximately twice the upper lip length. If the upper lip is longer than normal, then the lower anterior dental height should be longer than normal so that the facial dimensions will be balanced in the lower facial third (Fig. 23.17, distance C).

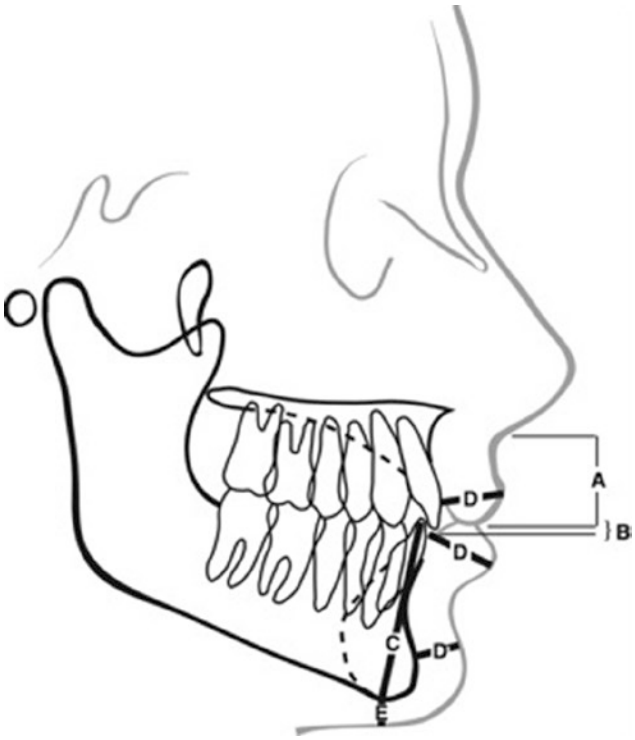


Fig. 23.17 Normal upper lip length (A) for a male is 22 ± 2 mm and for females is 20 ± 2 mm. Normal tooth-to-lip relationship (B) is 2.5 ± 1.5 mm. The lower anterior dental height (C) is measured from the mandibular central incisor tips to hard tissue menton. It has a normal value of 44 ± 2 mm in males and 40 ± 2 mm in females. An important interrelationship is two times the upper lip length should equal the lower anterior dental height. The soft tissue thickness of the upper lip, lower lip, and chin area (D) usually ranges from 11 to 14 mm, but more importantly should be a 1:1:1 ratio. The soft tissue thickness in the menton area (E) is normally 7 ± 2 mm

12. Soft tissue thickness: The thickness of the upper lip, lower lip, and chin area normally ranges from 11 to 14 mm. More importantly, there should be a 1:1:1 ratio. Variations in this ratio may influence treatment planning decisions regarding the lips and chin (Fig. 23.17, distance D).
13. Soft tissue thickness of menton: The distance measured perpendicular to Frankfort horizontal plane from hard tissue menton to soft tissue menton. The normal dimension is 7 ± 2 mm. Excessive thickness or thinness of this area may influence alterations in the height of the anterior mandible (Fig. 23.17, distance E).
14. Oropharyngeal airway: The oropharyngeal airway is measured from the posterior pharyngeal wall to the posterior aspect of the soft palate and from the posterior pharyngeal wall to the base of the tongue. The normal dimension for both of these areas is 11 ± 2 mm (Fig. 23.18).

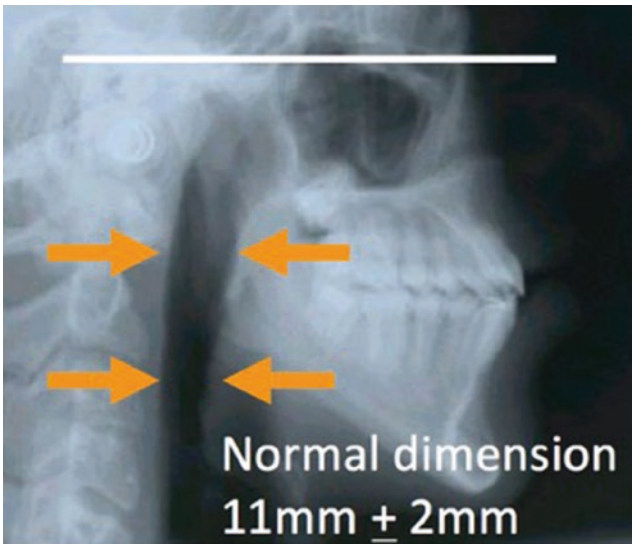


Fig. 23.18 The oropharyngeal airway is measured from the posterior pharyngeal wall to the soft palate, and from the posterior pharyngeal wall to the posterior base of the tongue. The normal value for both areas is 11 ± 2 mm

23.3 Dental Model Analysis

Dental model analysis is important in establishing proper diagnoses and treatment goals, particularly in reference to orthodontics. Proper dental model analysis improves the understanding and development of the presurgical orthodontic goals. Nine basic dental model evaluations to make are as follows:

1. Arch length measurements
2. Tooth size analysis
3. Crowding, spaces
4. Tooth position
5. Arch width analysis
6. Curve of occlusion (curve of Spee)
7. Cuspid-molar position
8. Tooth arch symmetry
9. Buccal tooth tipping (curve of Wilson)
10. Missing, broken down, or crowned teeth

23.3.1 Arch Length Measurements

Arch length measurements should correlate the widths of the teeth relative to the amount of alveolar bone available. The evaluation of arch length and cumulative dental width helps to identify the presence or absence of crowding or spacing. This evaluation helps to determine whether teeth need to be extracted, spaces need to be created, or spaces need to be closed (Fig. 23.19).

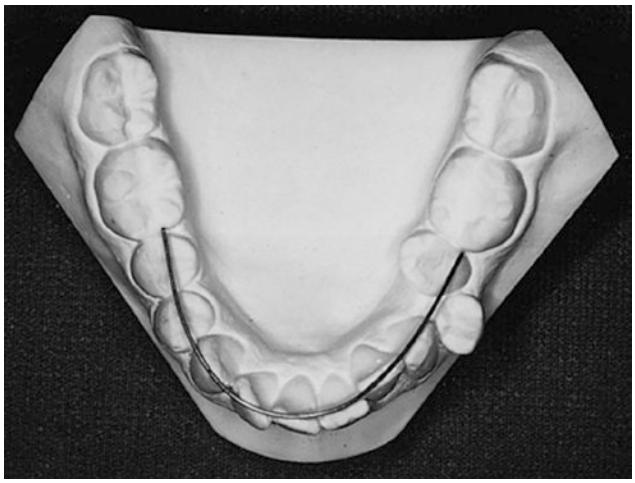


Fig. 23.19 The arch length evaluation correlates the widths of the teeth in relation to the amount of alveolar bone available. It also helps in determining whether extractions are indicated and what specific orthodontic mechanics may be necessary to align the teeth properly

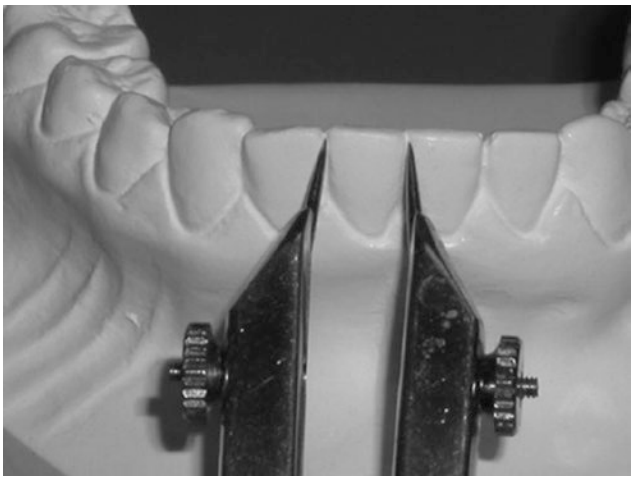


Fig. 23.20 A tooth size analysis evaluates the combined widths of the six mandibular anterior teeth in relation to the widths of the six maxillary anterior teeth. Measurements are made at the widest mesial-distal dimension of the crown. Evaluation of the tooth size compatibility is necessary so that appropriate orthodontic treatment can be used to correct the problem before surgery. Needlepoint calipers are helpful in this assessment

Orthodontic treatment can contribute to sleep apnea, particularly if bicuspid teeth are extracted to facilitate alignment of the teeth. To this day, some orthodontists continue to routinely extract bicuspid teeth, use headgear, or other mechanical methods of retraction to retrude the maxilla and maxillary teeth to fit with a retruded mandible, decreasing the oral cavity volume, displacing the tongue posteriorly, and contributing to the development of sleep apnea. Unfortunately, the sleep apnea symptoms do not surface until many years later. Sometimes the orthodontics needs to be totally reversed so that the bicuspid spaces are reopened to improve the size of the oral cavity to better accommodate the tongue and to provide better functional and aesthetic alignment of the teeth, with the best results coupled with orthognathic surgery when indicated.

23.3.2 Tooth Size Analysis

Tooth size analysis relates the relationship of the mesiodistal width of the upper teeth compared with that of the lower teeth. Although tooth size discrepancies can occur in the premolar and molar areas, this analysis is used primarily in relation to the anterior six maxillary and mandibular teeth. Many patients with dentofacial deformities have anterior tooth size discrepancies, often with a decreased maxillary tooth width (most commonly attributable to small lateral incisors) in relation to the mandibular teeth. In such cases, proper tooth alignment with all spaces closed often precludes the establishment of a good Class I cuspid relationship. Instead, an end-on or slight Class II cuspid-molar occlusal relationship often results. Bolton's analysis is a method of correlating the widths of the upper and lower anterior six teeth. Tooth size discrepancy between the anterior maxillary and mandibular teeth is determined by direct measurements of the anterior teeth. Needlepoint calipers (Fig. 23.20) and a tablet make for an easy method of calculation. (1) Measure anterior six teeth in each arch at the widest dimension of the crowns and punch holes in a tablet for each tooth for each arch (Fig. 23.21). (2) Measure the length of each arch to determine the actual arch lengths. (3) Multiply the lower arch length × 1.3. This provides the calculated upper arch length, or the length the upper anterior arch should be to fit the lower arch with a normal overbite, overjet, and a Class I cuspid relationship. (4) Subtract the actual upper arch length from the calculated arch length to determine the tooth-size discrepancy. Usually, the lower teeth are relatively larger than the upper teeth, commonly related to small upper lateral incisors. Tooth size discrepancies also can occur in the premolar and molar areas, where the maxillary and mandibular teeth should be approximately the same mesiodistal width. The management of tooth size discrepancies is important to achieve the best occlusal relationship. Tooth size discrepancies can be managed by alterations on the lower anterior teeth by changing position or slenderizing the teeth. Alternatively or in combination, the upper dental arch can be adjusted, commonly by creating space in the arch around the lateral incisors, requiring subsequent buildup of the lateral incisors with bonding, veneers, or crowns.

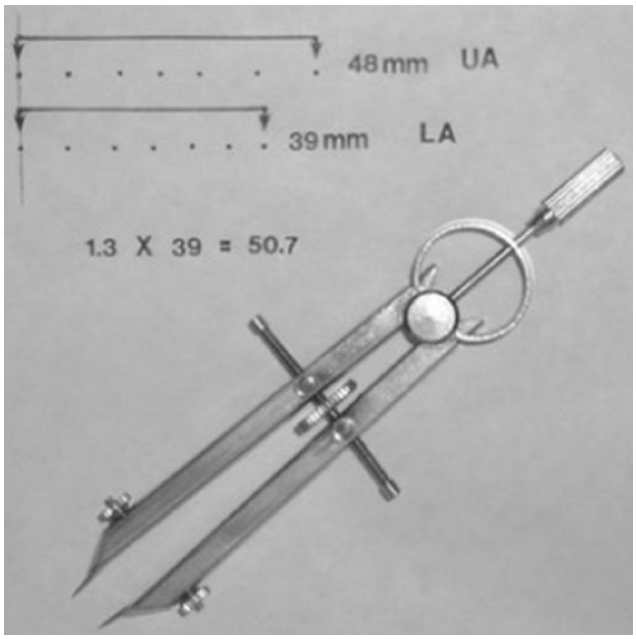


Fig. 23.21 Tooth size discrepancy between the anterior maxillary and mandibular teeth is determined by direct measurements of the anterior teeth. Needle point calipers and a tablet make for an easy method of calculation. (1) Measure anterior six teeth in each arch and punch holes in a tablet for each tooth for each arch. (2) Measure length of each arch to determine the actual arch lengths. (3) Multiply the lower arch length × 1.3. This provides the calculated upper arch length, or the length the upper anterior arch should be to fit the lower arch with a normal overbite, overjet, and a Class I cuspid relationship. (4) Subtract the actual upper arch length from the calculated arch width to determine the tooth-size discrepancy

23.3.3 Tooth Position

Tooth position in the context of orthognathic analysis refers primarily to the angulation of the maxillary and mandibular incisors in relation to the basal bone. The dental models are correlated with the cephalometric evaluation (Fig. 23.16), and the ideal axial inclination of the incisors is determined. The tooth position analysis determines whether extractions are necessary, spaces need to be created or eliminated, and what mechanics are needed to align and level the arches or segments of the arches.

23.3.4 Arch Width Analysis

Arch width analysis refers to the evaluation of the intraarch widths between the maxilla and the mandible. Arch width is best analyzed by holding the dental models in the occlusal position that is to be achieved with the orthodontic and surgical correction and then assessing the transverse relationship. For example, if a patient has a true skeletal Class III occlusion with a Class III cuspid-molar relationship, then position the models in a Class I cuspid-molar relation and evaluate the transverse relationship. Likewise, evaluate a skeletal Class II patient in a Class II cuspid-molar relationship by positioning the models into a Class I cuspid-molar relationship. Consider evaluating the transverse relationship by placing the models into a Class II molar position to determine whether a Class I cuspid and a Class II molar relationship would be best for that particular patient. Arch width analysis is helpful in determining presurgical orthodontic mechanics and contributes to the selection of the appropriate surgical procedures.

23.3.5 Curve of Occlusion (Curve of Spee)

The curve of occlusion has significant influence on whether the curve of occlusion in the arches is corrected orthodontically, whether extractions are necessary, or whether surgical intervention is indicated to level the occlusal plane. If an accentuated curve of occlusion in the lower arch is leveled orthodontically, the lower incisors will move anteriorly approximately 1 mm for every vertical millimeter of leveling required (Fig. 23.22a). After about 2 mm of leveling the lower arch by intrusion of the lower incisors, the orthodontics become less stable. Correcting a reverse curve of occlusion, particularly in the lower arch, by extruding the incisors may not provide a stable result. To correct a reverse curve, surgical leveling of the arches may be preferred. Surgical leveling may be achieved by subapical osteotomies or bilateral body osteotomies in the mandible or a segmental procedure in the maxilla.

In the maxillary arch, an accentuated curve of occlusion (Fig. 23.22b) when relatively minor, can be corrected orthodontically, but with significant accentuation, may be best correct surgically. Orthodontic extrusion of teeth may not be stable with a tendency for postsurgery orthodontic relapse. Segmental alignment of the maxillary arch with a major curve of Spee and surgical correction will provide a more predictable outcome. Severe reverse curves of Spee likewise may have limitations as to orthodontic correction and subsequent stability. Assessment of the curve of Spee and understanding limitations orthodontically and surgically, will help formulate a stable treatment plan.

23.3.6 Cuspid-Molar Position

The cuspid-molar position dictates the occlusal functions. A Class I cuspid-molar relationship usually is preferable; however, a Class II molar relationship is acceptable. A Class III molar relationship is less desirable, but it may be indicated in some cases.

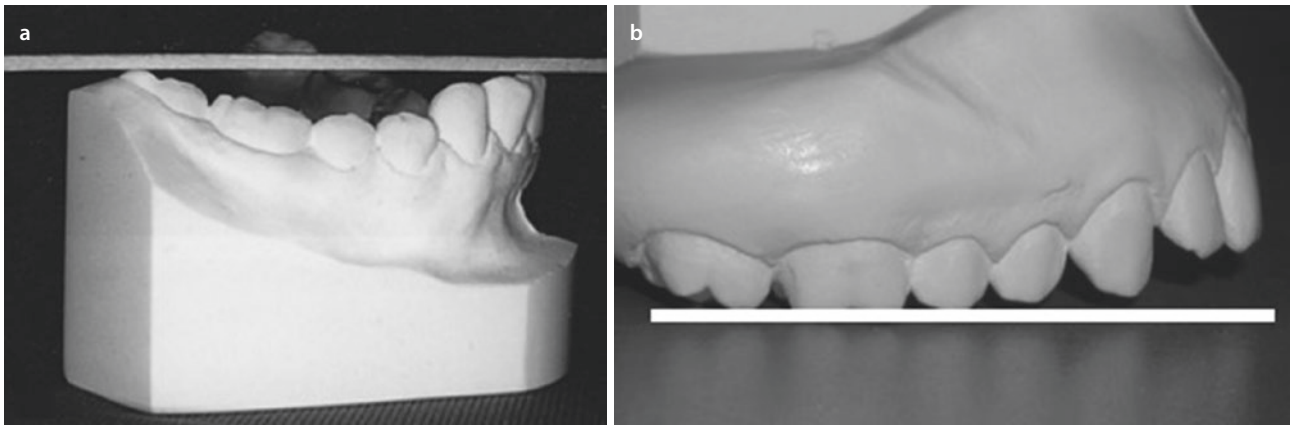


Fig. 23.22 **a** An accentuated curve of occlusion is seen in the mandibular arch, with midbuccal teeth being several millimeters below a line tangent to the incisors and second molars. For every millimeter of vertical leveling, the lower anterior teeth will come forward approximately 1 mm. **b** In the maxillary arch, the incisors should be about 1 mm above a flat plane with the posterior teeth on that flat plane. The degree of accentuated or reverse curve of occlusion will help dictate the orthodontic and surgical procedures necessary to achieve predictable treatment outcomes

23.3.7 Tooth Arch Symmetry

Tooth arch symmetry compares the left to right symmetry within each arch. A significant asymmetry may be present within the arch, such as a cuspid on one side being more anteriorly positioned than the cuspid on the opposite side. This problem often occurs when one side of the arch is missing a tooth. Correction may require special orthodontic mechanics, unilateral extraction, or additional surgical procedures.

23.3.8 Buccal Tooth Tipping (Curve of Wilson)

Buccal tooth tipping evaluates the position of the occlusal surfaces of the maxillary posterior teeth in a medial-lateral direction (Fig. 23.23). If the occlusal surfaces of the maxillary posterior teeth are tipped buccally, it may be difficult to achieve a proper occlusal relationship. In the presence of a transverse maxillary deficiency with preexisting buccal tipping, such tipping is even more difficult to correct orthodontically, orthopedically, or even with surgically assisted orthopedic expansion. The buccal tipping usually worsens with these mechanics. Even with surgically assisted rapid palatal expansion, the palate only expands approximately one-third of the amount of the expansion that occurs at the occlusal level, thus increasing the curve of Wilson. Surgical expansion is usually advantageous because the palate can be expanded by a greater amount than the occlusal level if indicated, thus decreasing the curve of Wilson, and segments of the maxilla can be repositioned in all three planes of space.

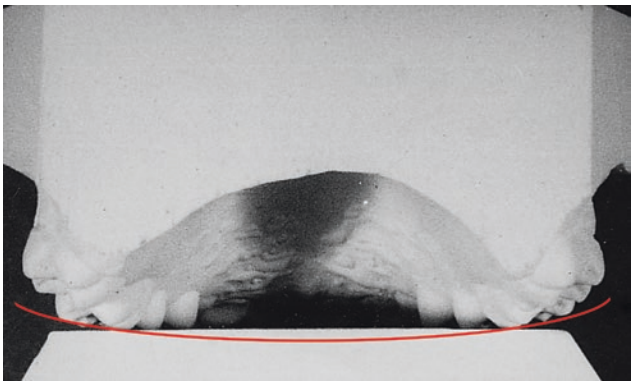


Fig. 23.23 The maxillary dental model is being evaluated from a posterior view, showing significant buccal tipping (increased curve of Wilson), with the palatal cusp tips being significantly lower than the buccal cusps. With an increased curve of Wilson in the presence of a transverse maxillary hypoplasia, orthodontic, orthopedic, and surgically assisted maxillary expansion will result in further increase of the curve of Wilson. Surgical expansion may be more predictable, as the arch can be expanded and curve of Wilson decreased by expanding the palate a greater amount than at the occlusion

23.3.9 Missing, Broken Down, or Crowned Teeth

Missing, broken down, or crowned teeth may influence treatment design. If a tooth is not restorable and requires extraction in a potential osteotomy location, the extraction space may need to be closed orthodontically or the space maintained. In some cases, it may be helpful to maintain the tooth to improve stability during surgical alignment of the jaws or segments thereof, with removal after surgery.

23.3.10 Ankylosed Teeth

Ankylosis of teeth is the abnormal adherence of alveolar bone to dentin or Cementum. The periodontal ligament and cementum on the root surface are resorbed by macrophages and osteoclastic cells, and new bone is produced by osteoblasts on the root surface without formation of a normal periodontal ligament, rendering the tooth non-movable with orthodontic mechanics. If an ankylosed tooth does not respond to orthodontic forces, surgical procedures may be indicated to facilitate movement of the tooth to the correct position. This could include subluxation of the tooth, segmental osteotomy, or extraction [26, 27].

23.4 Summary

OSA patients commonly have associated dentofacial deformities affecting the functional airway. This chapter was designed to illustrate a systematic method to evaluate dentofacial deformities, develop a comprehensive diagnosis, and establish an encompassing treatment plan. Primary factors contributing to sleep apnea are the following: (1) decreased oropharyngeal airway, (2) nasal airway obstruction, and (3) mandibular and maxillary hypoplasia. The normal A-P dimension from the posterior pharyngeal wall to the soft palate and posterior pharyngeal wall to the posterior base of the tongue is 11 ± 2 mm. In patients who have a retruded maxilla and mandible (very common in OSA patients), this airway may be significantly decreased. Accompanying these skeletal deficiencies is usually a high occlusal plane angle facial morphology. A normal occlusal plane to the Frankfort horizontal plane is 8 ± 4 degrees, but in the OSA patient with a retruded maxilla and mandible, the occlusal plane can be significantly increased making it more challenging for many surgeons to correct and open the oropharyngeal airway. There is a triad of factors that commonly go together in OSA patients, and they include the following: (1) a high occlusal plane angle facial morphology with associated retruded maxilla and mandible, (2) nasal airway obstruction related to hypertrophied turbinates and/or nasal septal deviation or spurring, and (3) TMJ pathology. TMJ pathology, particularly involving condylar resorption, is a common etiology for mandibular and maxillary retrusion, contributing to OSA. When TMJ issues are involved, the TMJ pathology must be addressed in order to provide stable treatment outcomes, decrease, or eliminate TMJ and myofascial pain as well as TMJ-related headaches and other associated symptoms. Patients with the high occlusal plane angle facial morphology with a retruded maxilla and mandible should always be assessed for nasal airway obstruction, decreased oropharyngeal airway, and TMJ pathology. Proper diagnosis and treatment planning can result in highly predictable and stable, functional, and aesthetic outcomes. This chapter reviewed the basic protocols for assessment of the OSA patient for diagnosis and treatment planning.

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MRI Evaluation for Patients with TMJ Disorders and Obstructive Sleep Apnea

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Contents

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Magnetic resonance imaging (MRI) is one of the most important tools available to the oral and maxillofacial surgeon for diagnosis and treatment planning of patients with temporomandibular joint (TMJ) disorders. However, it is estimated that 40–60% of TMJ MRIs are misread by radiologists. Therefore, it is very important for the oral and maxillofacial surgeon to be able to interpret TMJ MRIs. This chapter incorporates magnetic resonance imaging (MRI) into the diagnostic evaluation of obstructive sleep apnea (OSA) patients that also have temporomandibular joint (TMJ) pathology. TMJ pathology may coexist with the dentofacial deformity or may be the etiology of the jaw deformity that is responsible for creating the OSA. This chapter will present the MRI findings of the common TMJ pathologies associated with OSA. TMJ pathologies associated with mandibular condylar hyperplasia, benign, or malignant tumors will not be discussed, as they do not contribute to OSA.

One of the primary factors contributing to sleep apnea is a decreased oropharyngeal airway. Using lateral cephalometric analysis, the normal A-P dimension of the oropharyngeal airway from the posterior pharyngeal wall to the soft palate and posterior pharyngeal wall to the base of the tongue should be 11 mm, plus or minus 2 mm. OSA patients commonly have a high occlusal plane angle facial morphology that includes a retruded mandible and maxilla as well as a decreased oropharyngeal airway. A normal occlusal plane angle to the Frankfort horizontal plane is 8 degrees, plus or minus 4 degrees, but OSA patients commonly have a significantly increased occlusal plane angle. There is a triad of factors that are commonly observed in OSA patients, and they include: (1) high occlusal plane angle facial morphology associated with retruded maxilla and mandible, (2) nasal airway obstruction related to hypertrophied turbinates and/or nasal septal deviation or spurring, and (3) TMJ pathology. Patients with the high occlusal plane angle facial morphology should routinely be assessed for nasal airway obstruction, decreased oropharyngeal airway, and TMJ pathology.

For many OSA patients, the most highly predictable surgical treatment is to advance the maxilla and mandible in a counterclockwise direction [1–4], which opens up the oropharyngeal airway significantly [5–10]. The counterclockwise rotation of the maxillomandibular complex usually provides the best facial aesthetic balance while maximizing the increase of the oropharyngeal airway dimensions. The traditional method of straightforward or clockwise advancement of the maxilla and mandible that most surgeons perform may compromise the esthetic outcome and decrease the potential increase of the oropharyngeal airway.

Many OSA patients have TMJ issues that need to be surgically addressed in order for the orthognathic surgery to be successful and provide a stable, predictable outcome. Preexisting TMJ pathology, if ignored, can result in postsurgery condylar resorption and mandibular relapse with skeletal instability, malocclusion, pain, and decrease of the oropharyngeal airway that was achieved from the orthognathic surgery [11–21]. As the maxillary and mandibular complex is advanced forward in a counterclockwise direction in the presence of healthy TMJs or surgically corrected TMJs, the overall facial balance is predictably improved, skeletal and occlusal stability is established, jaw function is enhanced, the oropharyngeal airway is opened, and pain is eliminated.

To evaluate the presence or absence of TMJ pathology, radiographic evaluation is very helpful in the diagnostic process and cone-beam CT (CBCT) technology makes accessibility to low-cost, low-radiation CT scans, but are primarily methods to evaluate hard-tissue structures. MRI allows evaluation of hard and soft tissues of the TMJ, such as condyle, fossa, and disc position, morphology, mobility, extent of joint bone and soft-tissue degenerative changes, inflammation, condylar resorption, tumors, and connective tissue/autoimmune diseases [22, 23]. Additional imaging such as panograms, cephalometric radiographs, CT scans, CBCT scans, bone scans, 3D imaging, and 3D modeling may also be indicated for some OSA patients.

MRIs can help in the diagnosis of TMJ pathology in the silent joint where disc displacement and degenerative changes can be present but may not make noise and may not be particularly uncomfortable or painful, and therefore, clinicians often ignore the TMJ issues. If untreated in a patient requiring orthognathic surgery for maxillary/mandibular advancement (MMA) with or without counterclockwise rotation (CCWR) to correct OSA, surgery could result in a poor outcome relative to function, skeletal and occlusal stability, airway, and pain. The MRI provides a method to identify these patients and the associated TMJ pathologies.

Dr. Raymond Damadian is credited with the development of the MRI imaging technology, and on July 3, 1977, the first human MRI exam was performed. Superconducting magnets from 1.5 to 3.0 tesla (15,000–30,000 gauss) are required to achieve the imaging. Interestingly, the earth's magnetic field is equal to 0.5 gauss. There are radiofrequency coils that transmit waves into the body. Superconducting magnets align the protons head to feet. The radiofrequency magnets change the rotation of the protons causing resonance at Larmor frequency. There are three gradient magnets within the machine that create the image slices. The signals generated are picked up by special TMJ coils and sent to a computer, and Fourier transform formula maps the tissues and then integrates into 2D or 3D images. Contrast materials can be injected as normal and abnormal tissues will react differently. There are no known

biological hazards; however, it is not recommended during pregnancy. Dangers include magnetic coding will be erased from such devices as credit cards, etc. Pacemakers may malfunction. Aneurysm clips in the brain could move, and magnetic materials around or in the patient can cause serious or life-threatening damage.

MRI requirements for TMJ imaging include the following:

1. A closed 1.5–3.0 tesla MRI machine.
2. TMJ coils are highly recommended to enhance the imaging. The coils, particularly in the lower grade machines (1.5 tesla), will significantly enhance the imaging. Without the coils, the imaging may be unreadable and nondiagnostic.
3. MRI is best done prior to the application of orthodontic appliances as the metal devices can create distortion and interference of the TMJ anatomy, although in the presence of orthodontic appliances, an adequate MRI of the TMJ can usually be acquired. However, the more metal that may be associated with the orthodontic appliances and additional nonremovable metal devices, the greater the risk of interference of the MRI imaging. Any metal orthodontic appliances or devices must be nonmagnetic.

The recommended TMJ views for adequate MRI interpretation include the following:

1. *Coronal Closed Views* – in centric relation, maximum closure, without splints.
2. *Sagittal Closed Views* – in centric relation, maximum closure, without splints.
3. *Sagittal Open Views* – with maximum jaw opening.
4. *Sagittal Dynamic Views* – from a slight open position (to accommodate the ratchet device) to maximum open position. The dynamic views are often helpful in determining the point at which the displaced discs may or may not reduce mobility of the condyle and disc, presence of adhesions, etc. However, it is important to understand that the "dynamic" views are not acquisitioned with the patient's normal voluntary jaw opening. On the contrary, the opening is achieved with the patient in a supine position using an opening ratchet device placed between the maxillary and mandibular incisors. This opens the bite to accommodate the device so that the dynamic imaging begins with the jaws slightly open, unless the patient has an anterior open bite. The device opens the jaw in increments with MRI data recorded at each increment of opening until the maximum opening is achieved with the device still in place. The MRI data gathered at the various increments are integrated to appear as a continuous motion from the slight open position to maximum opening. This may or may not duplicate the patients normal jaw function when the patient is in an upright position.

In the MRI, different tissues are contrasted dependent of the tissue properties (proton density), and the pulse sequence parameters are usually defined as a T1- or T2-weighted image. In general, T1 images are helpful in identifying disc position, the presence of alteration in bone and soft-tissue structures, and interrelationships of the bony and soft-tissue anatomy. T2 MRI images are more helpful in identifying inflammatory responses in the TMJ. The importance of disc position cannot be overemphasized, and the MRI is the best diagnostic tool to determine disc position, TMJ pathology, quality, and salvageability of the disc and condyle, as well as will dictate the treatment protocol, particularly if surgery is indicated.

With a normal healthy TMJ (Fig. 24.1), the condyle should have a uniform shape and consistent thickness of cortical bone. The condyle should be positioned in the fossa with equal joint space between the condyle and fossa posteriorly, superiorly, and anteriorly. The articular disc should sit on top of the condyle with the posterior band at about the 12 o'clock position. The disc should have a bowtie shape with increased thickness of the posterior band and anterior band and a thinner area for the intermediate zone. The articular eminence should have a moderate inclination, although the articular eminence may be quite variable in steepness. There should be no joint effusion, inflammation, or synovitis evident. On opening, the condyle and disc should translate down and forward as a unit beneath the articular eminence. The MRI imaging can be correlated to CBCT and CT scan imaging of the TMJs for joint space and greater interpretation of bony pathology.

24.1 TMJ Articular Disc Displacement

The most common type of disc displacement is anterior as seen in Fig. 24.2a. The posterior band of the disc is anterior to the condylar head. When opening, the disc may (Fig. 24.2b) or may not reduce. Upon reduction, there is usually a palpable and sometimes audible pop in the joint as the head of the condyle comes downward and forward over the back end of the posterior band as the condyle reduces onto the disc (Fig. 24.2b). The mandible may then open the rest of the way with a normal condyle–disc relationship. Upon closing, the condyle may slide back off the posterior band of the disc, making a reciprocal closing click. As TMJ disease progresses, on opening the disc may not reduce back in position creating a silent joint, as the disc remains anterior throughout jaw function. Displacement of the articular disc can initiate a cascade of events leading to arthritis.

The anteriorly displaced disc may eventually become deformed and nonreducing but still be mobile, or

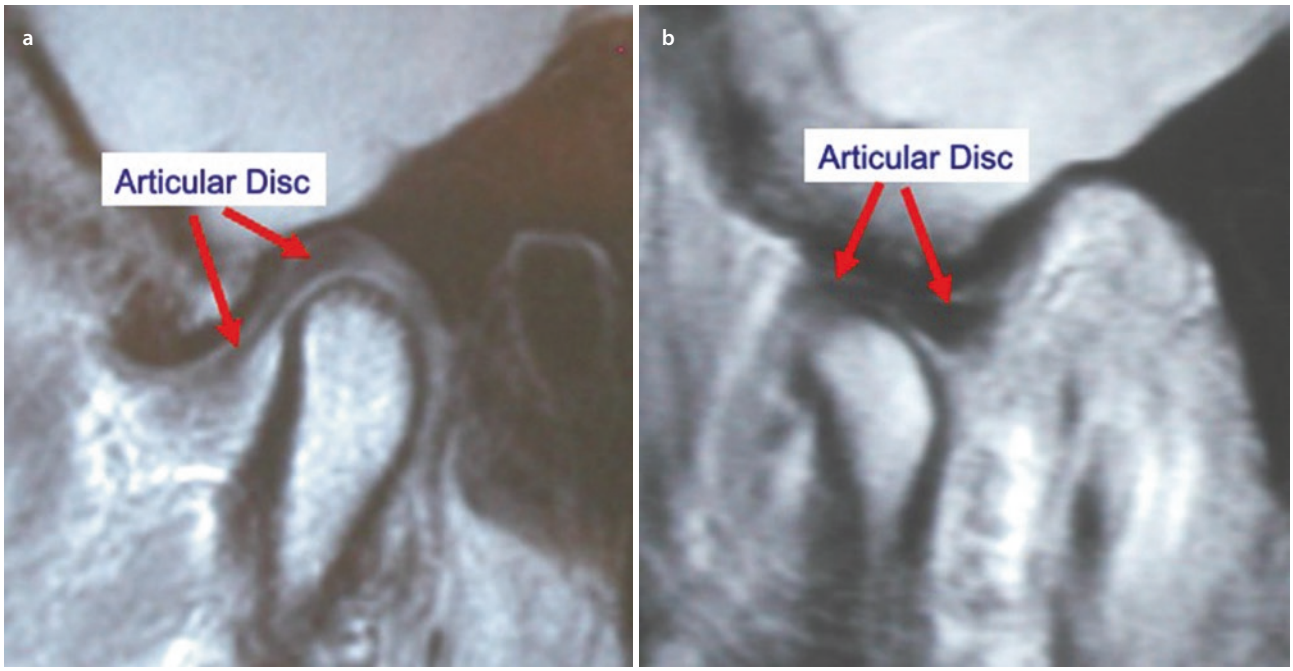


Fig. 24.1 MRI sagittal view of a normal healthy TMJ. **a** The joint space is equal posterior, superior, and anterior with the posterior band of the disc at 12 o'clock position relative to the condyle. The condyle has a smooth regular contour. **b** On opening, the condyle and disc translate down and forward as a unit beneath the articular eminence

become adherent to the articular eminence and/or fossa limiting the translation abilities of the condyle and disc creating the "closed lock" phenomenon (Fig. 24.2c). It is not uncommon to see a lateral rotational disc displacement where the disc may be anteriorly displaced at the lateral aspect of the joint, but toward the medial, the disc is in a more normal position, thus the importance of the evaluation of sagittal MRI views from lateral to medial in a sequential fashion. Since the lateral attachment of the disc to the condyle is weaker compared to the medial attachment, the disc more commonly can displace at the lateral aspect of the joint initially and then progress toward the medial side. This also can cause the disc to displace medially, so the disc becomes anteromedially displaced.

Medial displacement of the disc can occur where the disc is dislocated to the medial aspect of the joint and lacks coverage of the lateral portion of the condyle. There is typically a decreased lateral joint space, more evident on the coronal view (Fig. 24.3a). This can result in a condition called lateral capsular impingement, where the disc displaces medially and the capsule is pulled over between the lateral pole and the fossa, which can create pain issues. Discs can also be displaced laterally (Fig. 24.3b), but this is less common than anterior and/or medial displacement. There is a breakdown of the attachment at the medial pole, and the disc is displaced lateral to the condyle and can cause pain and dysfunction.

24.1.1 Silent TMJ with Disc Displacement

There are a number of TMJ pathological processes where the disc is displaced, but yet, the disc is silent with function. An MRI can determine the following silent joint disorders: (1) anterior displaced disc that does not reduce on opening (Fig. 24.4a, b); (2) steep articular eminence where the articular disc is anteriorly displaced, but in a vertical orientation so that upon opening, there is an immediate reduction of the disc as it is in a "preclick" position (Fig. 24.5a); (3) medial or lateral disc displacements (Fig. 24.3a, b); (4) certain pathological conditions such as adolescent internal condylar resorption (AICR) where there may be thickening of the bilaminar tissues so that there is a smooth transition from the thickened bilaminar tissue onto the displaced disc (Fig. 24.5b); (5) long-term splint therapy with downward and forward posturing of the mandibular condyle with thickening of the bilaminar tissues so that there is a smooth transition onto the disc (similar to Fig. 24.5b); and (6) Class II mechanics that may artificially pull the condyle down and forward onto the disc, but in an unstable position relative to the condylar centric relation.

When discs are anteriorly displaced for an extended time period, the discs may become deformed with loss of the intermediate zone and thickening of the posterior and anterior bands with progressive arthritis, rendering the discs and possibly the condyles nonsalvageable

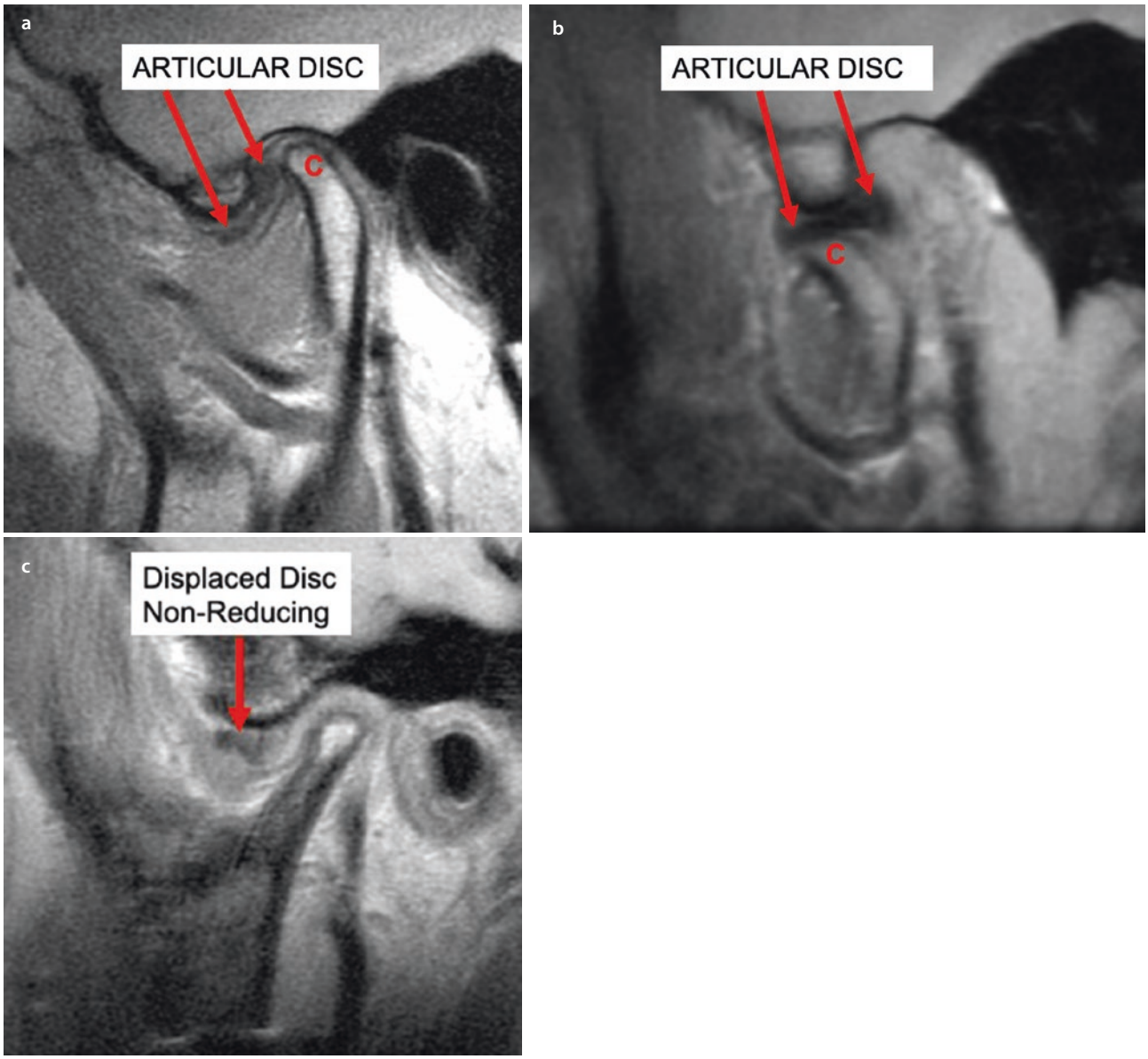


Fig. 24.2 **a** In the closed position, the condyle is positioned posterior in the fossa and the disc is anteriorly displaced with the posterior band at about the 10 o'clock position. **b** On opening, the disc reduces into a normal position. **c** The disc is severely deformed and anteriorly displaced. On opening, the disc will not reduce. If the disc becomes adhered to the articular eminence, it can cause a "closed lock" situation

(Fig. 24.2c). Also, there may be a degenerative process developing in the discs where there is a breakdown of the cartilaginous substance with vascular invasion and degeneration. When discs advance to a certain level of deformation and degeneration, they become nonsalvageable. When discs are displaced and nonreducing, the deformation and degenerative processes progress more rapidly as compared to displaced discs that reduce. Bony degenerative changes occur as well. An MRI will help determine the degree and progression of the degenerative and deformation changes to the joint structures and indicate the corrective surgical procedures that will provide the most predictable outcome for each patient's specific presentation. When discs are salvageable, a surgical option is to reposition and stabilize the disc into a normal position with a Mitek bone anchor and artificial ligaments (Mitek anchor technique) (Fig. 24.6) [24–33]. For success of this technique, specific criteria must be met. The MRI is strategic in determining if this technique will be beneficial.

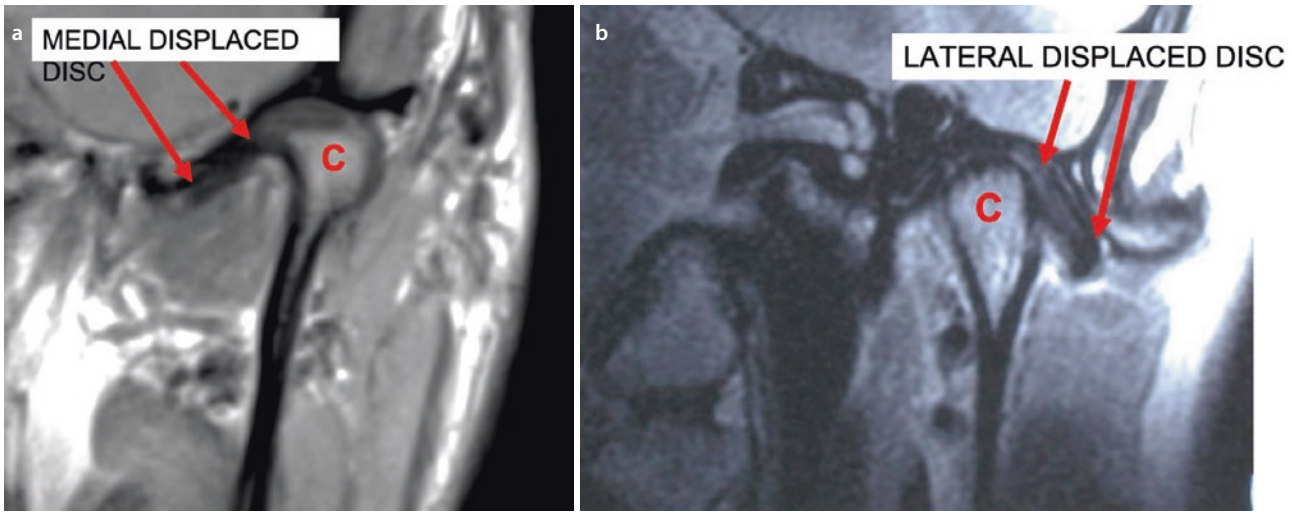


Fig. 24.3 **a** MRI coronal view demonstrates a medially displaced articular disc. Notice the decreased vertical joint space toward the lateral aspect of the fossa. **b** Coronal view demonstrates lateral displacement of the articular disc. Medial joint space may be narrowed

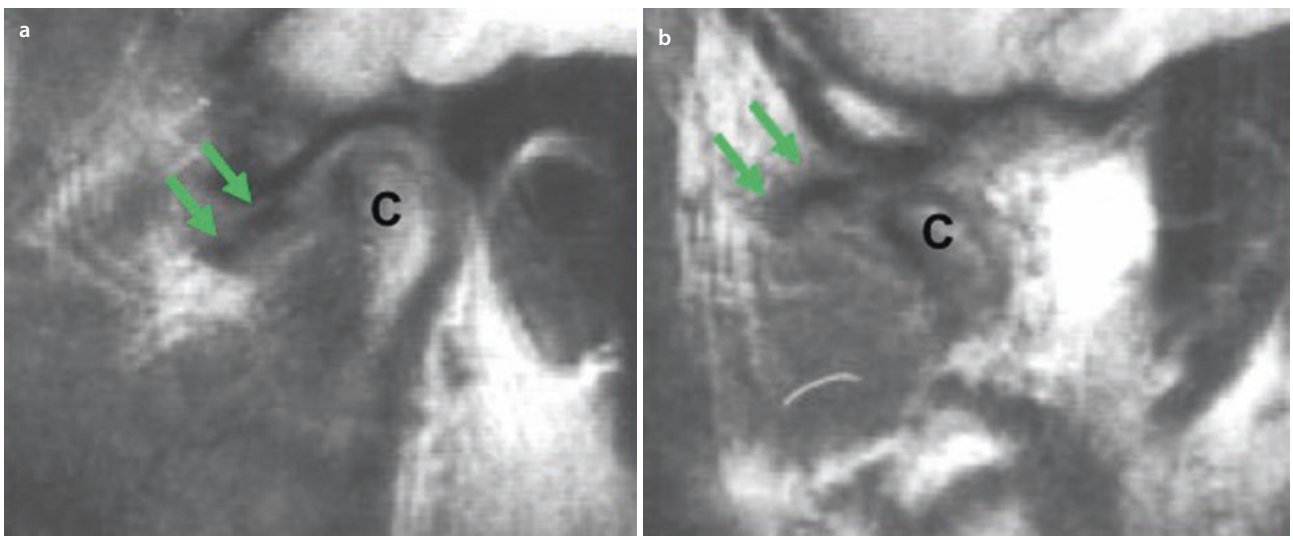


Fig. 24.4 **a** Sagittal view of an anteriorly displaced disc. Green arrows identify the disc and "C" indicates the condyle. **b** In the open view, the condyle translates forward beneath the articular eminence, but the disc remains anteriorly displaced without reduction. Green arrows identify the disc and "C" indicates the condyle

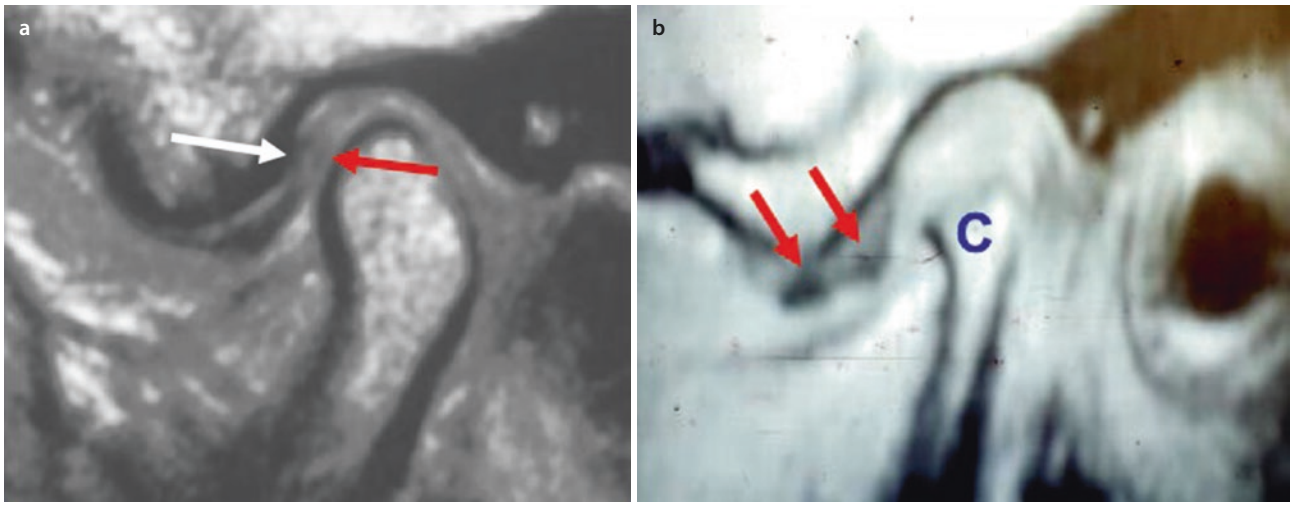
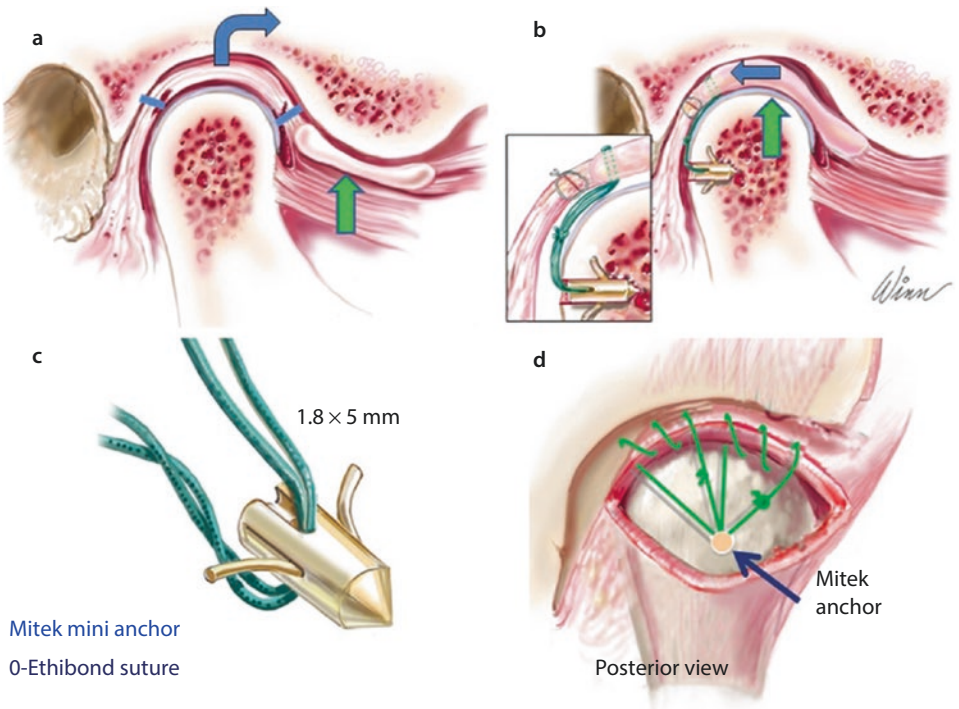


Fig. 24.5 **a** Sagittal view with the white arrow pointing to the posterior band of the disc that is anteriorly displaced but in a vertical orientation. The red arrow points to the condyle–disc interface. As the mandible is opened, there is a smooth transition between the condyle and disc rendering an opening without a click or pop. This is a silent joint with a displaced disc, but can cause pain and dysfunction. **b** There is significant thickening of the bilaminar tissues in this TMJ with AICR. The thickening of the bilaminar tissues can occur with certain pathologies as well as long-term splint therapy. A thickened bilaminar tissue can result in a smooth transition of the condyle onto the disc rendering a silent joint. The red arrows identify the position of the disc and "C" indicates the condyle. The distance between the head of the condyle and the fossa identifies the hyperplastic bilaminar and synovial tissues

Fig. 24.6 Mitek anchor technique. **a** The disc is anteriorly displaced (green arrow). The bilaminar tissue on top of the condyle is excised and the disc is mobilized. **b** The disc is repositioned over top of the condyle. The Mitek anchor is inserted into the posterior head, and artificial ligaments (0-Ethibond suture) are used to secure the disc in position. **c** Illustrates the Mitek anchor and the placement of two 0-Ethibond sutures through the eyelet that will act as artificial ligaments. **d** Posterior view of the condyle showing the insertion of the Mitek anchor lateral to the mid sagittal plane and the placement of the sutures (artificial ligaments) through the posterior aspect of the posterior band of the disc to secure it in position



24.1.2 Criteria for Articular Disc Repositioning with the Mitek Anchor Technique

1. Anterior, medial, or lateral disc displacement.
2. Four years or less since initial disc displacement.
3. Salvageable disc and condyle.
4. No other joints involved (no polyarthritis).
5. No reactive arthritis.
6. No connective tissue/autoimmune disease.
7. No intracapsular adhesions.
8. No history of recurrent infections, such as sexually transmitted diseases; upper respiratory or pulmonary infections; urinary tract infections; genital infections or history of endometriosis or other gynecological pathologies; gastrointestinal problems such as irritable bowel syndrome, GERD, and Crohn's disease; and eye infections. These conditions can cause a reactive arthritis, where patients may not do well, even with an ideal surgical disc repositioning as the pathological process may continue to progress postsurgery.

24.1.3 Implications for the OSA Patient

In OSA patients that have TMJ disc displacement, but meet the criteria for disc repositioning, and if the procedure is properly performed prior to or concomitant with MMA with or without CCWR, surgical treatment should provide skeletal and occlusal stability, improved jaw function, significant decrease or elimination of pain, and improved dimension of the oropharyngeal airway. Postsurgical MRI sagittal view of a repositioned disc with a Mitek anchor is seen in Fig. 24.7. There is some distortion of the MRI imaging because of the metal anchor in the head of the condyle, but the reduced position of the disc is noted.

OSA patients that fall out of the criteria for disc repositioning will benefit from custom-fitted total joint prostheses [8, 34–55] where the prostheses are used to reconstruct the TMJ as well as advance the mandible for MMA (Fig. 24.8). Virtual surgical planning (VSP)

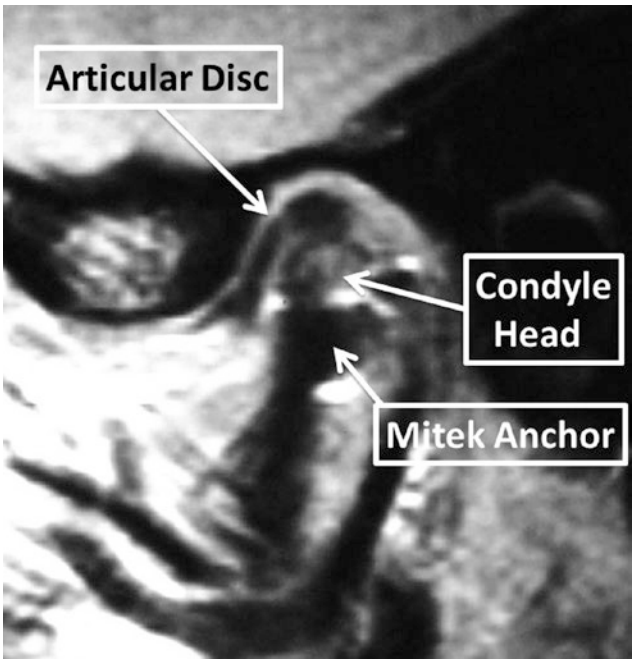


Fig. 24.7 MRI demonstrates the position of the articular disc over top of the condyle, secured by the Mitek anchor and artificial ligaments. The Mitek anchor creates some image distortion because of the metal content. The articular disc is the ideal position relative to the condyle

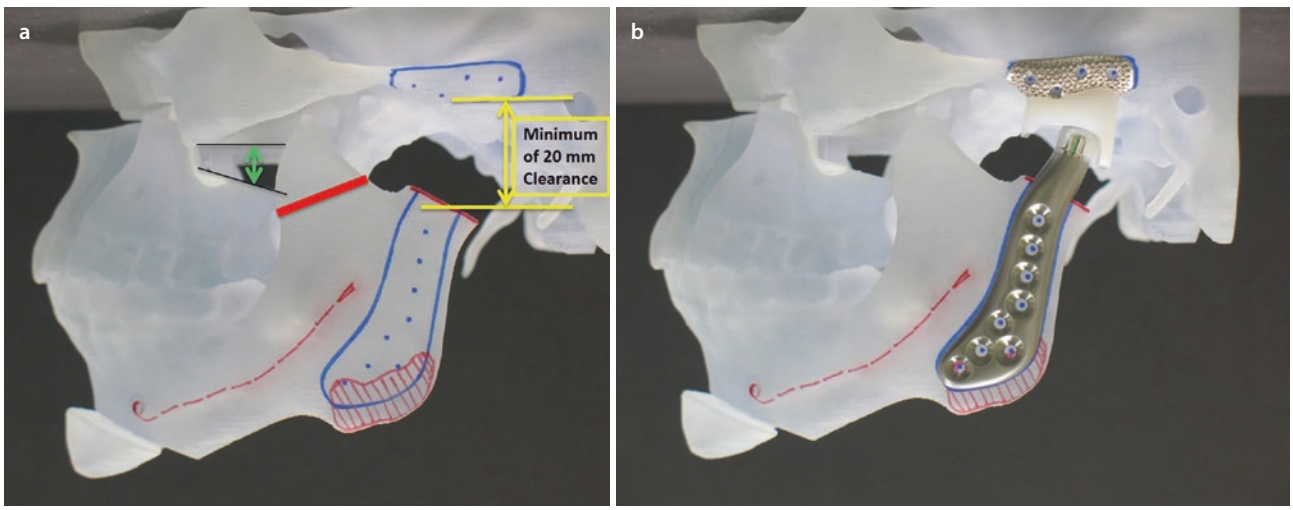


Fig. 24.8 **a** Preparation of the stereolithic model for a patient undergoing bilateral TMJ total joint prostheses reconstruction and maxillary osteotomies for counterclockwise rotation and advancement of the maxillomandibular complex. The condyle has been removed, and a 20 mm gap has been created to accommodate the prosthesis. Coronoidectomy must also be performed in order to achieve the counterclockwise rotation. **b** TMJ concepts custom-fitted total joint prostheses have been manufactured to fit this patient's specific anatomical requirements with the jaws repositioned to achieve the final desired result

has improved the accuracy and the projected surgical outcome and the custom adaptation of the prostheses to each patient's specific anatomical requirements [49, 56–59]. With orthognathic surgery only, without corrective TMJ surgery, the negative effects that can occur include development or worsening of TMJ pain, myofascial pain, headaches, ear symptoms, etc. There is an 84% chance of developing pain postsurgery and a significant increase of the postsurgical pain level (84% increase) compared to presurgery. There is a risk (30%) that condylar resorption can occur postsurgery [11].

24.2 Adolescent Internal Condylar Resorption (AICR)

Adolescent internal condylar resorption (AICR) has a relatively classic MRI presentation. This hormonally mediated condition is initiated usually between the ages of 11 and 15 years and predominantly in females (ratio 8:1 females to males); there is no genetic predisposition; only the TMJ joints are involved with no other joints affected; discs are anteriorly displaced; condyles progressively decrease in size; and the mandible is retruded. Following the onset of the process, the rate of condylar resorption is about 1.5 mm per year. The mandible will slowly retrude into a Class II occlusal and skeletal relationship with a tendency toward anterior open bite. These patients all have high occlusal plane angle facial morphological profiles [60–63].

MRIs of these cases present with a condyle that is slowly becoming smaller in size in all three planes of space. In some cases, there is a significant thinning of the cortical bone on top of the condyle contributing to the inward collapse of the condylar head in this pathological process (Fig. 24.9). Interestingly, the fibrocartilage on the condylar head and in the fossa remains intact. This is the only form of condylar resorption where the fibrocartilage remains intact. The articular discs are anteriorly displaced and may or may not reduce on opening. Nonreducing discs will degenerate and deform at a more rapid rate as compared to discs that reduce.

24.2.1 Implications for the OSA Patient

Treatment considerations for OSA patients with AICR usually require MMA with or without CCWR including disc repositioning with Mitek anchor versus total joint prostheses. Consideration for disc repositioning with Mitek anchors follows the guidelines previously described for TMJ disc displacement. Our studies [60–63] demonstrate that AICR can be arrested if the articular discs are put back into position on top of the condyle and stabilized with the Mitek anchor technique (Fig. 24.6) [24–33]. Results are best for AICR if the TMJ surgery is performed within 4 years of the onset of the TMJ pathology. After 4 years, the discs may not be salvageable. Patients that fall outside of these criteria will be candidates for total joint prostheses [8, 34–59], resulting in a significantly greater outcome predictability relative to stability, improved function, and airway, as well as decrease in pain.

OSA patients that have AICR undergoing MMA without appropriate TMJ surgically management will have predictably unstable skeletal and occlusal out-

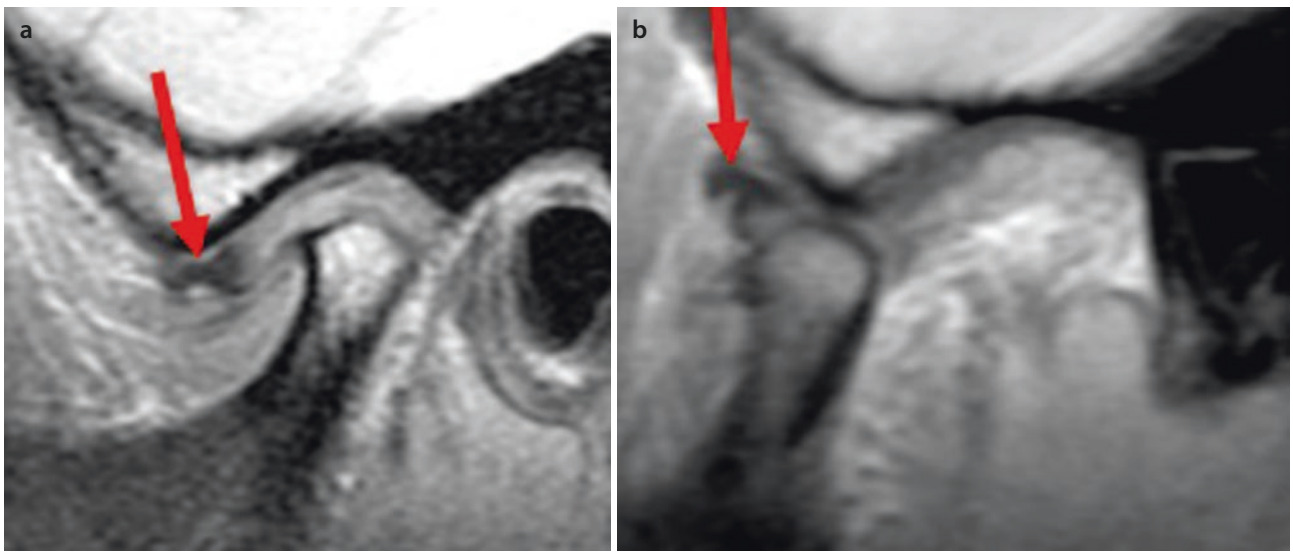


Fig. 24.9 **a** MRI of TMJ AICR. The disc is anteriorly displaced. Notice the thinning of the cortical bone on top of the condyle and the loss of condylar vertical dimension. The disc is anteriorly displaced (red arrow). **b** On opening, the articular disc commonly remains anteriorly displaced without reduction (red arrow) in AICR

comes because of the condylar resorption. A common approach of many surgeons is to follow the patient until the "condylar resorption burns-out" and then perform the MMA with clockwise rotation of the MMA by increasing the occlusal plane even greater than the already high occlusal plane angulation. However, with MMA, the TMJs will have increased loading and could reinitiate the condylar resorption process. Additional negative effects of MMA on this patient population may result in development or worsening TMJ pain, myofascial pain, headaches, ear symptoms, etc. Also, the amount of advancement required to correct the OSA would dictate that the maxilla would need to be advanced a greater amount in order to advance the mandible enough to open the oropharyngeal airway creating major and unaesthetic compromises in the facial appearance. Additional surgery may be required that would include total joint prostheses (Fig. 24.8) and repeat orthognathic surgery to correct the original failed surgery, as well as MMA and CCWR to correct the TMJ pathology, restore facial balance, increase the oropharyngeal airway, and eliminate residual pain.

24.3 Reactive Arthritis (ReA)

Reactive arthritis (ReA), or seronegative spondyloarthropathy, is an inflammatory disease in joints usually caused by venereal and respiratory bacteria. ReA is one of the most common forms of arthritis, but least understood. In the TMJ, ReA commonly develops in the late-teens through the fourth decade, predominately in females, and can cause TMJ pain, arthritis, and condylar resorption. Systemic symptoms of ReA may include joint pain, fever, fatigue, back pain, degenerative joint disease, polyarthritis, and dysfunction of the immune system. The most common bacteria that cause ReA are from two genera: chlamydia and mycoplasma. The specific species identified contributory to knee and TMJ ReA include *C. trachomatis, C. pneumoniae, C. psittaci, M. genitalium, M. pneumoniae,* and *M. fermentans* [64–75].

The plausible theory for chlamydia- and mycoplasma-induced TMJ ReA begins with a triggering infectious site established elsewhere in the body that is often asymptomatic. Host cells such as macrophages and monocytes become infected. When an injury or inflammatory reaction occurs in the TMJ, the host cells respond, transport the bacteria through the hemopoietic system, infiltrate the synovial and bilaminar tissues, and colonize the bacteria; the TMJ infection is initiated; and bone and cartilage degeneration begins, as well as the production of pain. Chlamydia and mycoplasma bacteria stimulate pro-inflammatory and pain mediators, such as tissue necrosis factor alpha, nitric oxide, cytokines, chemokines, and interleukins (IL-1, IL-6, IL-8), and may be the primary source of pain experienced by many TMJ patients [1, 6–8]. Currently, there are no predictable nonsurgical treatments to eliminate this TMJ pathology although some promising techniques may be developing.

MRI of ReA may initially show a localized area of fluid effusion, inflammation, and synovitis, with or without disc displacement and with or without erosion of the condyle and/or fossa. As the disease progresses, it can present as a more profuse inflammatory process surrounding the disc and through the bilaminar tissues and capsule (Fig. 24.10). The ReA can cause significant destruction of the TMJ structures. The MRI may show the presence of disc displacement as well as joint effusion, synovitis, and inflammation with the soft tissues in association with condylar degeneration that can include resorption. However, in low-grade inflammatory conditions, bone deposition may occur on the condyle and fossa that could lead to osteophytes, heterotopic bone deposition, and ankylosis (Fig. 24.11).

24.3.1 Implications for the OSA Patient

Surgical options to treat ReA include arthroscopy and arthrocentesis that may reduce symptoms temporarily but will not eliminate the bacteria. Open joint debridement and disc repositioning may be effective in the very early stages, but will be ineffective with more advanced disease. The most predictable TMJ treatment option for OSA patients with ReA requiring MMA is custom-fitted total joint prostheses to reconstruct the TMJs and advance the mandible in conjunction with the MMA with CCWR (Fig. 24.8) [8, 34–59]. This approach will usually provide the best outcome predictability relative to skeletal and occlusal stability, improved jaw function, increased the airway dimension, decreased pain, and maximized facial balance.

OSA patients that have ReA undergoing MMA with or without CCWR, and without appropriate TMJ surgically management, may have predictably unstable skeletal and occlusal outcomes if the ReA has caused presurgery condylar resorption or the surgery initiates the resorption. A common approach of many surgeons is to follow the patient until "condylar resorption burns-out" and then perform the MMA. However, the MMA will load the TMJs and could reinitiate the condylar resorption process. Additional negative effects of MMA on this patient population without TMJ surgical management may result in the development of, or worsening TMJ pain, myofascial pain, headaches, ear symptoms, etc.

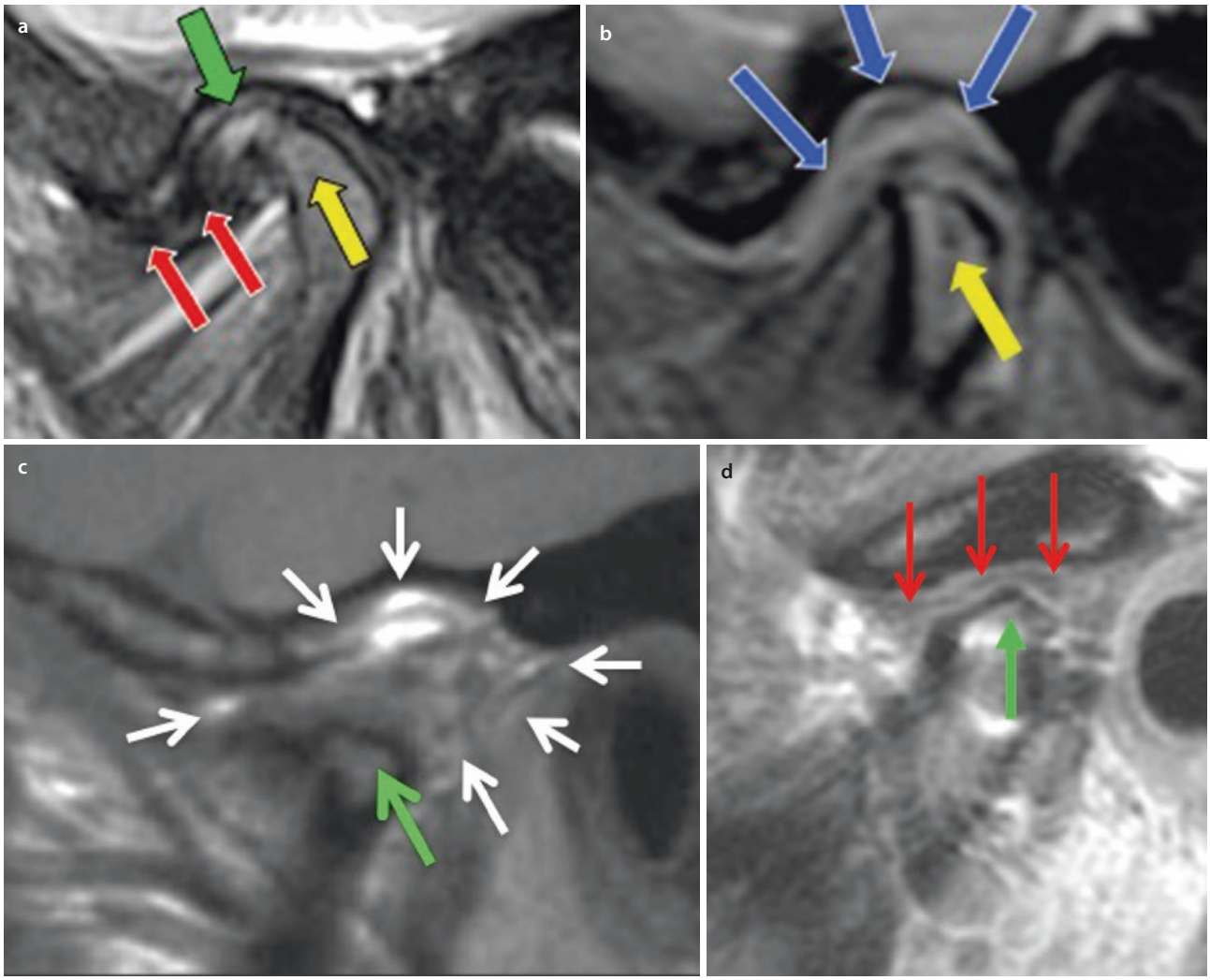


Fig. 24.10 **a** The disc is anteriorly displaced (red arrows) with an inflammatory process (reactive arthritis – ReA) within the joint as illustrated by the whitish tissue (green arrow) surrounding the disc and in the bilaminar tissues. The condyle (yellow arrow) is undergoing degenerative changes at the anterosuperior aspect. **b** ReA in a more advanced form. The blue arrows point out the inflammatory tissue within the joint. The yellow arrow indicates the condyle with evidence of erosion and loss of vertical height. There may be some remnants of the disc, but for the most part, it has been destroyed by the inflammatory process. **c** Severe ReA that has caused significant destruction of the condyle (green arrow) with large mass of reactive tissue within the joint space as outlined by the white arrows. Even the articular eminence has been resorbed. **d** This MRI demonstrates a post-Mitek anchor repositioning of the articular disc in a patient with ReA. The disc (red arrow) is slowly being resorbed by the reactive tissue surrounding it (the grayish tissue) as well as causing arthritic changes to the condylar head

24.4 Connective Tissue and Autoimmune Diseases (CT/AI)

The common CT/AI diseases that can affect the TMJs include juvenile idiopathic arthritis (JIA), rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, Sjogren's syndrome, systemic lupus erythema, scleroderma, mixed connective tissue disease, etc. Multiple systems are commonly involved with these diseases. Peripheral joints are usually affected bilaterally and symmetrically inflamed, resulting in progressive destruction of articular structures. Facial deformity can occur with TMJ involvement with associated condylar resorption. Clinical and radiographic features include the following: (1) retruded mandible, (2) posterior maxillary vertical hypoplasia, (3) progressive worsening facial and occlusal deformity, (4) high occlusal plane angle facial morphology, (5) Class II occlusion and anterior open bite, and (6) TMJ symptoms such as noises, pain, jaw dysfunction, headaches, and ear symptoms [45, 76].

MRI features include loss of condylar vertical dimension, significant mediolateral condylar narrowing but the residual condylar stumps may mushroom and become broad in the A-P direction; articular eminence

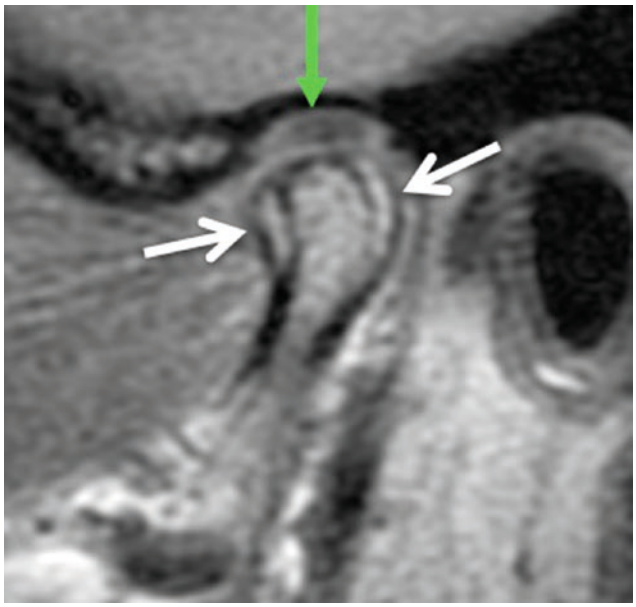


Fig. 24.11 A low-grade ReA may actually cause bone deposition around the condyle and fossa (white arrows), with slow destruction of the articular disc (green arrow). ReA that stimulates bone deposition can result in bony ankylosis

resorption; and articular disc may be in position but surrounded by a pannus (reactive tissue) that eventually destroys the disc but also is the cause of condylar and articular eminence resorption (Fig. 24.12). In more severe cases, particularly in JIA patients, the condylar stump may function forward beneath the remaining articular eminence.

24.4.1 Implications for the OSA Patient

The most predictable treatment for OSA patients with the TMJs affected by CT/AI diseases includes the following: (1) bilateral reconstruction of the TMJs and CCWR and advancement of the mandible with custom-fitted total joint prostheses (Fig. 24.8); (2) coronoidectomies if the ramus is significantly advanced or vertically lengthened with the prostheses; (3) autogenous fat graft harvested form the abdomen or buttock, packed around the prosthesis in the articulation area [76–79]; (4) maxillary osteotomies for advancement with CCWR; and (5) additional adjunctive procedures indicated (i.e., genioplasty, rhinoplasty, turbinectomies, and septoplasty) [8, 34–59]. Other techniques that have been advocated for TMJ reconstruction for this patient population include using autogenous tissues such as temporal fascia and muscle flaps, dermal grafts, rib grafts, sternoclavicular grafts, and vertical sliding ramus osteotomy. However, the disease process that created the original TMJ pathology can attack the autogenous tissues used in the TMJ reconstruction causing failure of the grafts. Performing orthognathic surgery only for MMA with or without CCWR will have a high failure rate relative to skeletal and occlusal stability, pain, and maintenance of the oropharyngeal airway.

24.5 Trauma

Traumatic injuries to the mandible may create facial deformities leading to OSA, particularly involving untreated displaced bilateral or unilateral subcondylar fractures. Patients may present with: (1) mandible retruded with deviation toward the affected side if unilateral; (2) pain and jaw dysfunction; (3) deficient growth on the affected side(s) in growing patients; (4) Class II skeletal and occlusal relationships with anterior open bite; and (5) unilateral cases, premature contact of the posterior occlusion on the affected side with anterior and contralateral open bite. Imaging features could include the following: (1) evidence of subcondylar fractures; (2) condyles malpositioned downward, forward, and medial to the fossa; and (3) decreased vertical ramus/condyle length.

MRI will also show the disc position and condition. The disc may be displaced with the condyle, or the disc can remain in the fossa with only the condyle displaced (Fig. 24.13).

24.5.1 Implications for the OSA Patient

At the initial presentation of the trauma, the options for treating subcondylar fractures are open reduction, closed reduction, or no treatment. The amount of displacement and the condition of the fracture(s) will dictate the necessary treatment to fix the problem. When identified early, fractures may be best treated by open reduction for significantly displaced segments or closed reduction for minimally displaced segments to achieve a symmetric face and stable occlusion. If the condyle is minimally to moderately displaced, still salvageable along with its articular disc but already healed, then it is possible that orthognathic surgery could realign the jaw structures properly, and if the disc is displaced, it can be repositioned with a Mitek anchor (Fig. 24.2) [24–33].

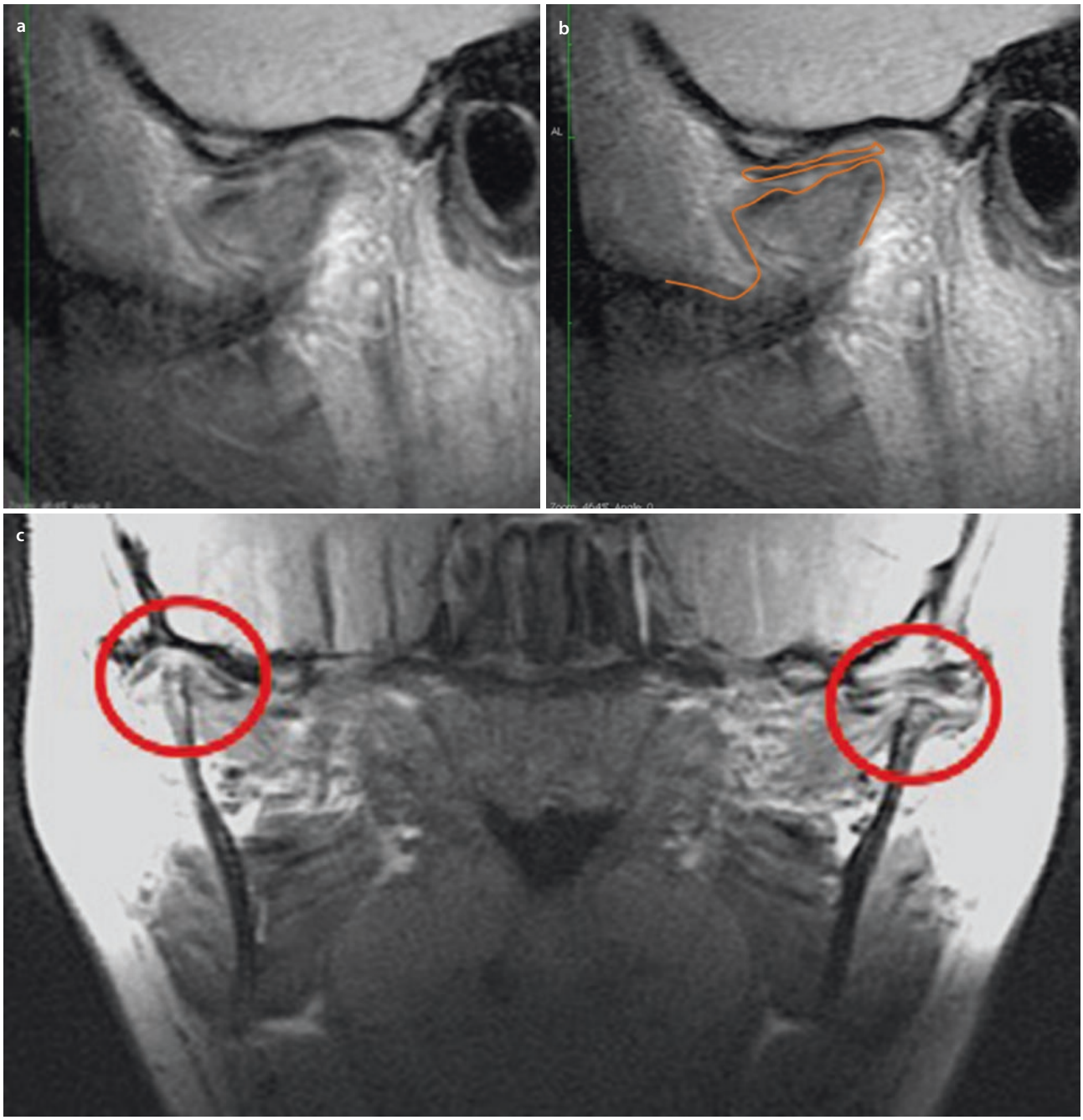


Fig. 24.12 **a** Sagittal view of the joint with JIA. There is significant loss of the vertical height of the condyle and commonly "mushrooming" (increased AP dimension of the residual condyle). The articular disc is commonly in position but surrounded by a reactive pannus (thin gray tissue surrounding the disc). **b** This is the same image but with the mushroomed condylar head and disc outlined. The gray tissue surrounding the disc is responsible for destruction of the joint. Notice also that the articular eminence has significantly resorbed as well. **c** Coronal views show the significant condylar resorption and transverse narrowness of the residual condylar elements classic with JIA

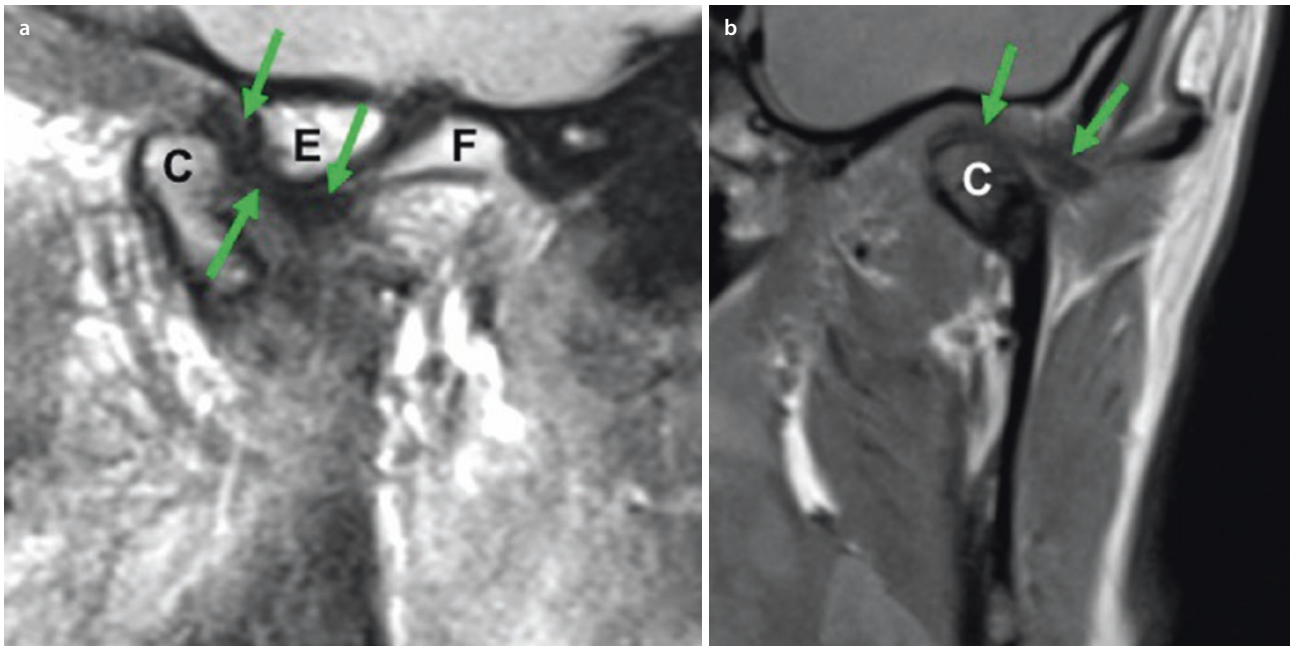


Fig. 24.13 **a** Sagittal view of a left mandibular subcondylar fracture with the condyle displaced anteromedial to the ramus. The articular disc (green arrows) is anteriorly displaced relative to the fossa but posteriorly displaced relative to the condylar head. C condyle, E articular eminence, F fossa. **b** Coronal view illustrates the medial displacement of the condylar head **c**. The disc (green arrows) is laterally displaced relative to the condylar head

If the condyle is severely deformed and nonsalvageable, then the most predictable method for reconstruction of the TMJ is using custom-fitted total joint prostheses (Fig. 24.8) [8, 34–59], TMJ fat grafts [76–79], and repositioning of the mandible, if there is an associated mandibular malalignment. Other treatment options for TMJ reconstruction following removal of the displaced condyles are rib grafts, sternoclavicular grafts, vertical ramus osteotomies, etc., but these outcomes are far less predictable.

OSA patients that have subcondylar fractures with malunion and malalignment and a retruded mandible will have the best outcome predictability with TMJ reconstruction and mandibular advancement with custom-fitted total joint prostheses and MMA with CCWR if also indicated.

24.6 TMJ Ankylosis

TMJ bony ankylosis can occur bilateral or unilateral, usually develops as a result of trauma, inflammation, sepsis, and/or systemic diseases, resulting in severely limited jaw function as well as oral hygiene and nutritional problems. When this condition occurs during the growing years, it can severely affect jaw growth and development as well as contribute to OSA. In unilateral ankylosis, the other condyle will continue to grow but may be retarded in its true growth potential. The common clinical and radiographic characteristics of TMJ ankylosis, particularly when occurring in children, include the following: (1) decreased jaw mobility and function; (2) decreased growth on the involved side(s); (3) retruded mandible; (4) facial asymmetry if unilateral involvement with the mandible shifted toward the ipsilateral side; (5) Class II occlusion; (6) radiographic evidence of heterotopic bone around the TMJ(s); (7) decreased vertical height of the ramus and posterior maxilla; and (8) decreased oropharyngeal airway [80].

MRI may demonstrate evidence of fibrous or bony ankylosis between the condyle and the fossa or heterotopic bone surrounding the joint (Fig. 24.14) appearing as a dense black mass. In the early stages of the process, the disc may be identifiable with or without displacement and there may be evidence of inflammation particularly when the etiology is related to an inflammatory or infectious process. Areas of calcification and osteophytes may be seen. As the disease progressed, the disc and joint space may not be visualized.

24.6.1 Implications for the OSA Patient

The most predictable treatment for the OSA patient with ankylosis includes the following: (1) release of the ankylosed joint, condylectomy, removal of the heterotopic and reactive bone with thorough debridement of the TMJ and adjacent areas; (2) coronoidectomies if the

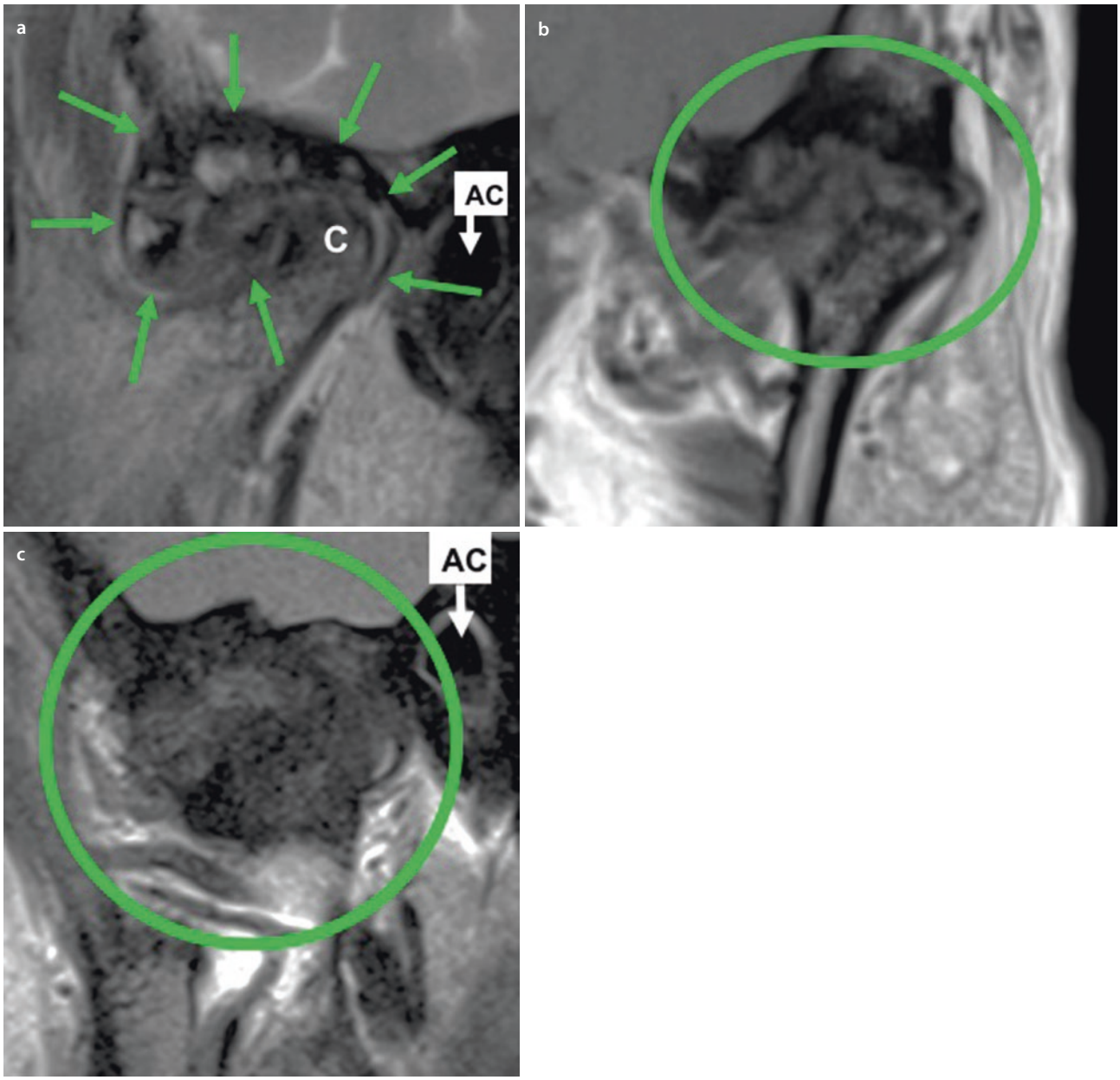


Fig. 24.14 **a** MRI sagittal view of TMJ ankylosis. "C" identifies the condylar head. The bony mass outlined by the green arrows surrounds the condyle. AC auditory canal. **b** Coronal view shows bony continuity of the condyle and fossa. **c** Sagittal view of another case with a large dense bony mass surrounding the condyle associated with ankylosing spondylitis. The condyle is not identifiable. AC auditory canal

ramus is significantly advanced or vertically lengthened with the prosthesis, or if coronoid hyperplasia developed which is a risk, particularly with ankylosis at an early age; (3) reconstruct the TMJs and advance the mandible with a custom-fitted total joint prosthesis (Fig. 24.8); (4) autogenous fat graft (harvested from the abdomen or buttock) packed around the prosthesis in the TMJ articulation area; (5) maxillary osteotomies for MMA with CCWR; and (6) adjunctive procedures indicated such as genioplasty, turbinectomies, nasoseptoplasty, and rhinoplasty [80–83].

Other techniques that have been advocated for reconstruction of TMJ ankylosis include using autogenous tissues such as temporal fascia and muscle flaps, dermis-fat grafts, rib grafts, sternoclavicular grafts, vertical sliding osteotomy, and gap arthroplasty. The total joint prosthesis with a fat graft packed around it is a superior technique.

24.7 Other End-Stage TMJ Conditions

Other TMJ end-stage conditions that can contribute to OSA include (1) congenital deformities (i.e., hemifacial microsomia, Treacher-Collins syndrome); (2) multiply operated joints; (3) failed TMJ alloplastic implants; and (4) failed autogenous tissue used for TMJ reconstruction.

MRI evaluation of these conditions may not be particularly helpful for diagnosis and treatment planning as significant distortion and interference may render the MRI unreadable and nondiagnostic. CBCT and CT scans would be the imaging of choice for initial evaluation of these conditions.

OSA patients with these TMJ pathologies may benefit from TMJ reconstruction and mandibular advancement with custom-fitted total joint prosthesis (Fig. 24.8), placement of fat grafts around the articulating part of the prostheses, as well as concomitant maxillary osteotomies for MMA with CCWR, and other indicated adjunctive procedures to achieve the best outcome results relative to function, stability, esthetics, and elimination of pain.

Studies show good outcomes with these treatment protocols. However, the quality of results decreases as the number of previous TMJ surgeries increases, particularly in reference to pain relief and jaw function. When the TMJ concepts total joint prostheses system is used as the first or second TMJ surgery, the success rate is very good relative to jaw function, stability, facial balance, and pain relief. After two or more previous TMJ surgeries, the decrease or elimination of pain and jaw function is far less predictable [8, 34–59, 76–87].

24.8 Summary

Healthy and stable TMJs are necessary for quality treatment outcomes in orthognathic surgery for the OSA patients. If TMJ pathology is preexisting, orthognathic surgery results may be unsatisfactory relative to function, esthetics, skeletal, and occlusal stability as well as pain. The oral and maxillofacial surgeon should be suspicious of possible TMJ problems in the OSA patient with the following conditions: (1) Class II high occlusal plane angle facial morphology with retruded mandible; (2) progressively worsening Class II occlusal and jaw relationship; (3) facial asymmetry, particularly with progressive worsening; (4) anterior open bite and/or lateral open bite; (5) patients reporting headaches, TMJ pain, myofascial pain, history of clicking and popping of the TMJs, and/or ear symptoms; and (6) history of CT/AI diseases, other joint problems, facial trauma, etc. The surgeon should not ignore these symptoms. With one or more of these symptoms, OSA patients should be evaluated for possible TMJ pathology. An MRI of the TMJs can aid in the identification of the specific TMJ pathology and progression of the disease process and indicate the surgical procedures necessary to maximize the treatment outcomes. Failure to recognize and treat these conditions can result in significant relapse, increased pain, decrease of the oropharyngeal airway, and a greater complexity of subsequent treatment.

During the past three decades, major advancements have been made in TMJ diagnostics and the development of surgical procedures to treat and rehabilitate the pathological, dysfunctional, and painful TMJ. Research has clearly demonstrated that TMJ and orthognathic surgery can be safely and predictably performed at the same operation, but it does necessitate the correct diagnosis and treatment plan, as well as requires the surgeon to have expertise in both TMJ and orthognathic surgery. The surgical procedures can be separated into two or more surgical stages, but the TMJ surgery should be done first. With the correct diagnosis and treatment plan, combined TMJ and orthognathic surgical approaches provide complete and comprehensive management of OSA patients with coexisting TMJ pathology and dentofacial deformities.

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Maxillary Surgical Procedures for Correction of Obstructive Sleep Apnea

Will R. Allen and Matt J. Madsen

Contents

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25.1 Background

Obstructive sleep apnea (OSA) is a sleep disorder that affects approximately 5–15% of the adult general population [1]. It is characterized by repetitive intermittent complete or partial breathing obstruction during sleep due to collapse of the airway. This interrupted respiration affects sleep pattern due to continuous arousals which often leads to daytime sleepiness and contribute to a host of medical comorbidities. These obstructions lead to decreased oxygen saturation and increased partial pressure of blood CO_2 levels. This affects patient well-being and has been documented to be associated with a host of medical comorbidities including hypertension [2–6], cardiovascular disease [4], heart failure [4–6] metabolic syndrome [3, 4, 6], and stroke [5, 6].

Obstructive sleep apnea is commonly observed in patients with obesity [6], large neck circumference, male sex, maxillary or mandibular deficiency [7, 8], long upper airway length [8–13], narrow nasal passage [14], maxillary constriction [14], and a narrow pharynx [15]. A more detailed discussion of the anatomy, physical findings, and diagnosis is described in detail elsewhere in this text.

First-line treatment for OSA is nonsurgical medical management with continuous positive airway pressure (CPAP). This treatment modality is poorly tolerated by some patients which necessitates other avenues including surgical intervention. Patients with a Respiratory Disturbance Index (RDI) of greater than 20 episodes per hour, oxygen saturation less than 90%, hypertension, arrhythmia, anatomical abnormalities of the upper airway, or failure of medical management are candidates for surgical intervention.

Surgical management has traditionally been divided into two phases. Phase 1 focuses on correction of aberrant nasal, palatal, tongue, and septum anatomy. Therapy includes nasal septoplasty, turbinectomy, tongue advancement, palate reduction including UPPP, and hyoid myotomy. Phase 2 therapy includes skeletal correction, most often in the form of maxillomandibular advancement.

For patients with failed surgical intervention via phase 1 therapies or nonsurgical therapy, maxillomandibular advancement (MMA) often with counterclockwise rotation of the mandible has become the surgery of choice. This surgery consists of advancing the maxilla with a LeFort I osteotomy and the mandible with a bilateral sagittal split osteotomy of the mandible (BSSO) as much as 10 millimeters. Often, surgical correction involves rotating the occlusal plane in a counterclockwise (CCW) fashion. MMA results in a decrease in lateral pharyngeal wall tension, which is a determinate factor in increasing the airway size which improves a patient's apnea hypopnea index (AHI) [16–18], O_2 saturation [16, 18], and ESS score [16–18] following CCW advancement. This CCW rotation has been shown to improve OSA in patients by reducing the apnea hypopnea index (AHI) [7, 12, 17–24], improve Epworth Sleepiness Scale [16, 17, 20, 21], improve oxygen saturation [7, 16, 19], increase airway diameter [11, 12, 15, 20, 25–28], decrease airway length [11, 12] which improves clinical symptoms and overall quality of life.

Traditional approaches have focused on bimaxillary surgery or single mandibular surgery, specifically mandibular advancement to improve retrolingual airway space. However, recent studies have sought to understand the role of improving OSA via maxillary orthognathic surgery procedures. Orthopedic procedures such as rapid maxillary expansion (RME) [29–35] in children and surgical assisted rapid palatal expansion (SARPE) [14, 36–39] or segmental Lefort surgery in adults demonstrate improvements in OSA. The mechanism by which AHI is improved is likely due to widening of the nasal floor which widens the nasal cavity [29, 30, 33]. This decreases the nasal resistance to airflow reducing obligate mouth breathing which has been shown to be a contributing factor to abnormal dentofacial development [40]. Additionally, a pathologically constricted maxillary arch does not allow for a normal tongue posture contributing to a posterior and inferior tongue position [41]. Normalizing maxillary arch width and increasing the horizontal dimension of the dentoalveolar framework, allows the tongue enough space within the dentoalveolar process. This new tongue posture improves pharyngeal airway [35] and even improves the airway dimension in the lower airway near the epiglottis [14]. In addition to tongue position, correction and widening of the skeletal landmarks, the surrounding soft tissue anatomy of the airway responds in kind. Maxillary advancement pulls the velum and velopharyngeal muscles forward which results in less tension [7, 19, 27].

The greatest perceived benefit to maxillomandibular advancement or advancement of the maxilla or mandible in single jaw surgery is to decrease airway resistance. As described in Pouiseuilles's law, $\Delta P = 8\mu LQ/\pi r4$ where P is pressure difference at ends of the airway, L = length airway, Q = volume of air which passes per given time, μ = dynamic viscosity, r = airway radius. In short, the resistance of a tube is proportional to the fourth power of its radius. As we surgically increase the radius of the airway, resistance greatly decreases. This chapter will discuss maxillary procedures for the OSA patient.

25.2 Treatment Planning Maxillary Surgery

Prior to surgery, an accurate and comprehensive examination should be performed to determine the patient's diagnosis. Clinical records for orthognathic surgery con-

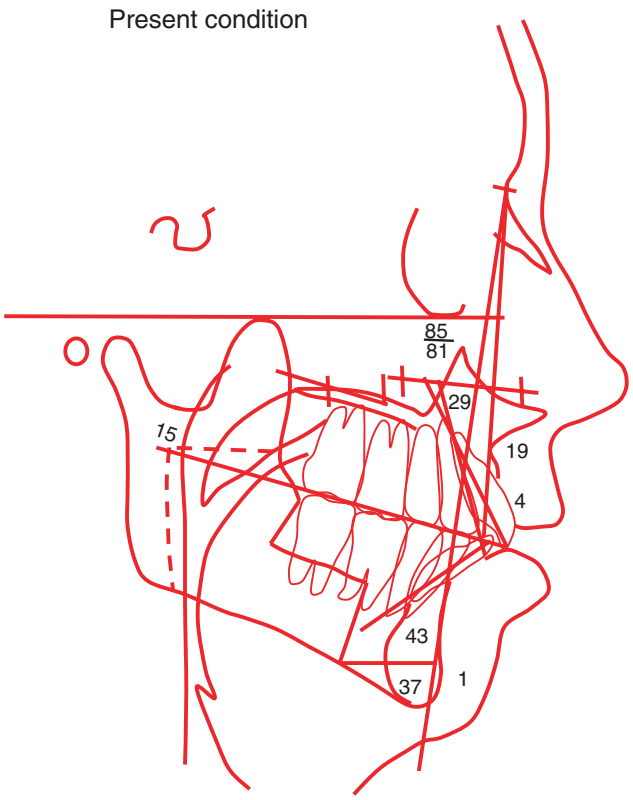


Fig. 25.1 Tracing of lateral cephalometric radiograph of the patient prior to surgery with Wolford Analysis [42]

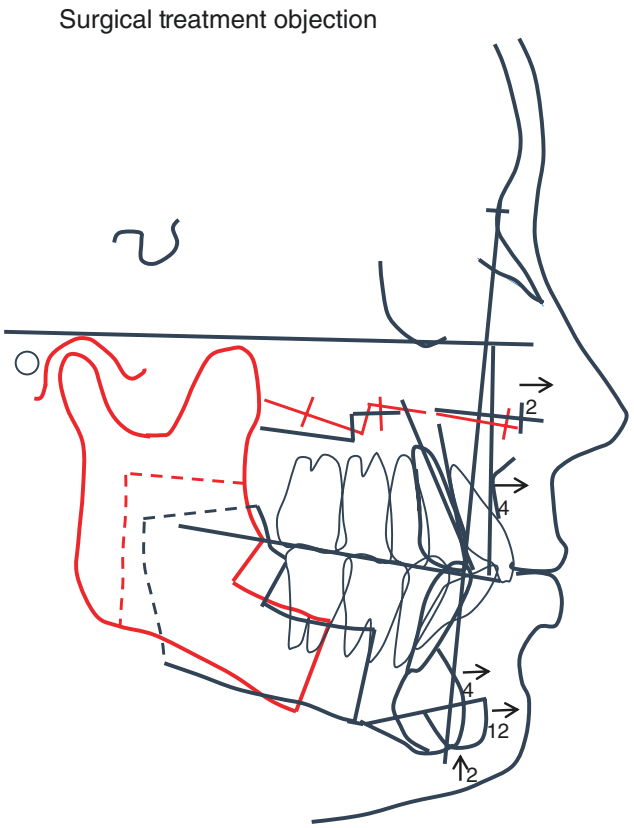


Fig. 25.2 Surgical treatment objective tracing demonstrates planned movement of maxilla and mandible during surgery using Wolford Analysis [42]

sist of a clinical exam, radiographs, models, and prediction tracing of lateral cephalogram. The clinical exam will consist of measuring facial and occlusal landmarks on a patient and comparing them against a standard which accounts for a patient's age, race, and sex. Radiographs should include a panoramic film, lateral cephalometric film, or a 3D cone beam CT. A prediction tracing should be constructed for the patient's current condition which when taken along with clinical impression help determine the diagnosis (Fig. 25.1). The surgical treatment objective (Fig. 25.2) tracing should demonstrate the planned movement during surgery. More recently, virtual surgical planning (VSP) based on advanced imaging, has in some cases, replaced traditional haptic methods (Fig. 25.3). Maxillary and mandibular models should be related to each other in centric relation and mounted in an articulator to allow spatial orientation of the planned advancement and surgical splint fabrication (Fig. 25.4).

25.2.1 Bone Anatomy

The body of the maxilla is composed of two halves which fuse midline and contain four projections called the frontal, zygomatic, dentoalveolar, and palatine processes. The frontal process of the maxilla extends superiorly to form the lateral aspect of the nose and the medial aspect of the orbit. The inferior-medial aspect of the orbital rim is formed from the maxilla. Approximately, 5–7 mm below the orbital rim is where the infraorbital nerve exits its canal. The zygomatic process extends laterally to meet the zygomatic bone. The palatine processes extend medially as a horizontal shelf of bone which fuses midline into the median palatine suture. This process extends posteriorly to join the palatine bone to form the hard palate. Distal to the maxillary tuberosity is the infratemporal surface of the maxilla which is the site of lateral pterygoid plate separation during LeFort I and SARPE surgery (Fig. 25.5).

25.2.2 Vascular Anatomy

The LeFort I osteotomy was performed before the vascular anatomy was fully understood. During surgery, the nasopalatine artery and descending palatine artery can be separated and direct perfusion from the maxillary soft tissue is disrupted. This led to the belief that collateral

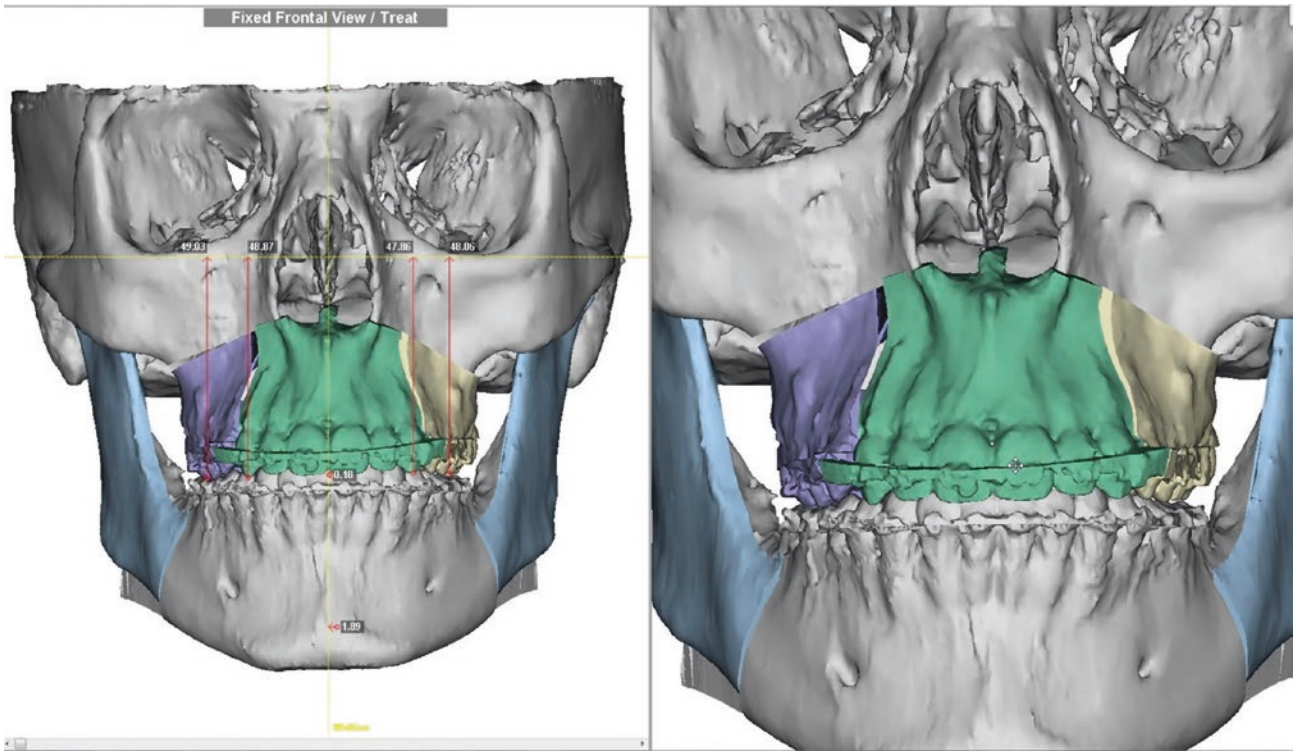


Fig. 25.3 Virtual surgical planning (VSP) is performed using 3D cone beam CT and digital scan of dentition. This planning can supplement or take the place of conventional lateral cephalometric tracing

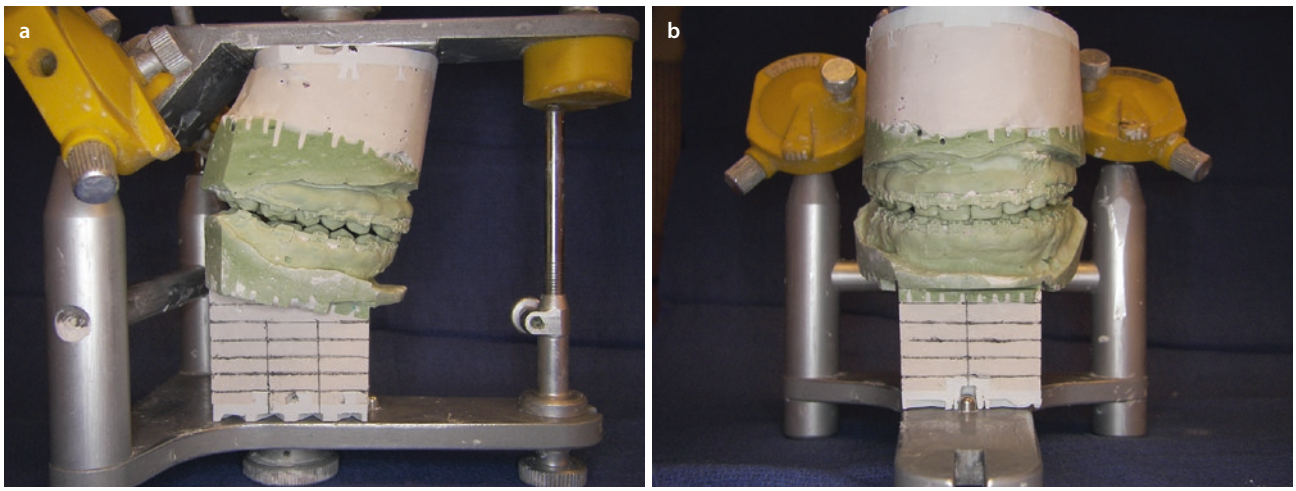


Fig. 25.4 Dental models of maxillary and mandibular arch are related to each other in centric relation on an articulator using mounted facebow transfer. Model surgery is performed on these models to fabricate acrylic splints used to control movements of each arch during surgery

circulation from the soft palate was sufficient to provide vascular support to the maxilla. Bell demonstrated in a classic study that blood supply to the maxilla came from the ascending pharyngeal artery and the ascending palatine artery [43]. The ascending palatine artery is a branch of the facial artery and the ascending pharyngeal artery is a branch of the external carotid artery (Fig. 25.6).

25.3 Lefort I Osteotomy Including Modifications

The OSA application of the Lefort I Osteotomy is similar to that described by Obwegeser in 1965 [44]. The goal is complete mobilization of the maxilla to allow for significant advancement. An external reference is recom-

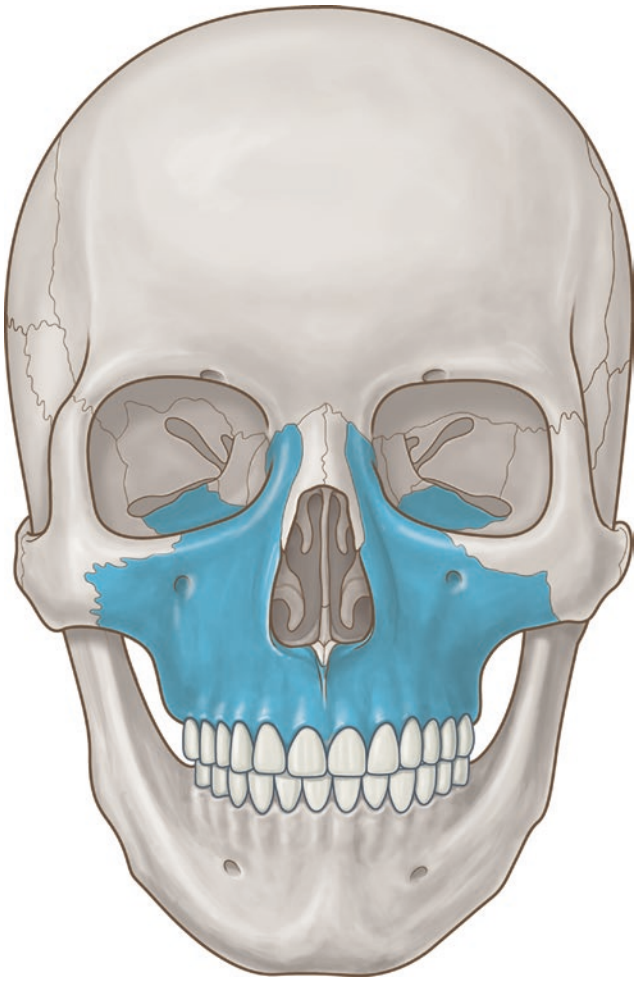


Fig. 25.5 The maxilla has four projections called the frontal, zygomatic, dentoalveolar, and palatine processes which articulate with nine bones of the midface and cranium

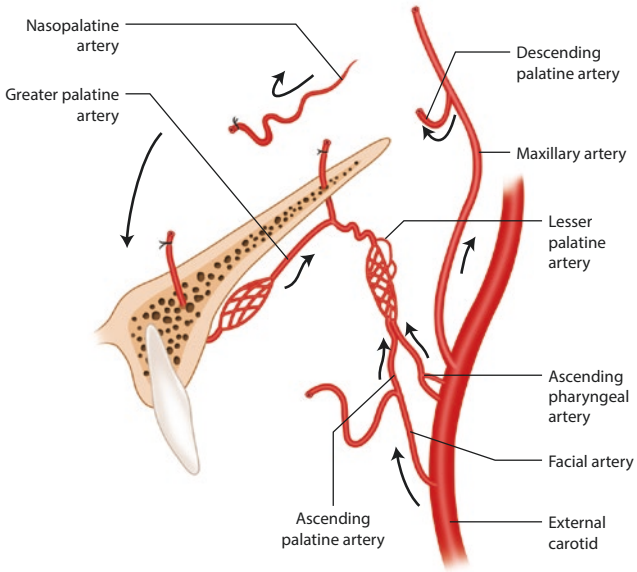


Fig. 25.6 Ascending palatine artery and ascending pharyngeal artery anastomose with the greater palatine artery to provide blood supply to the maxilla

mended to allow proper orientation. This is accomplished with Kirschner wire (0.035 inch) insertion into the nasion region (Fig. 25.7). A caliper is then used to measure from the central incisor brackets to the K-wire and recorded for reference (Fig. 25.8). Surgical exposure is accomplished using a maxillary vestibular incision that extends from first molar to first molar. It is imperative to leave an adequate margin of nonkeratinized mucosa for closure. The incision should be made 5 mm above the mucogingival junction (Fig. 25.9). A full thickness mucoperiosteal flap is elevated with dissection and elevation of periosteum around the piriform rim and anterior nasal spine. Dissection is then carried superiorly identifying the infraorbital nerve and infraorbital foramen. Next, the lateral maxilla is the dissected to the zycomaticomaxillary junction and dissection is carried to the pterygomaxillary junction. The nasal mucosa is elevated from the nasal floor and lateral nasal walls with a curved Freer elevator or Molt 9 elevator.

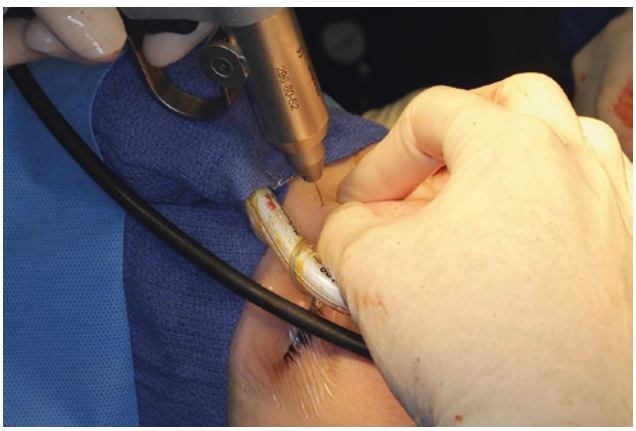


Fig. 25.7 External reference is performed by insertion of a K-wire into the intersection of frontal and nasal bones

Once surgical exposure is complete, a curved tip Obwegeser retractor is placed into the pterygomaxillary junction using care to make sure the instrument is subperiosteal and is resting on the lateral pterygoid plate. A Seldin retractor is placed submucosally along the lateral nasal wall. A horizontal osteotomy is then completed using a reciprocating saw starting from the pterygomaxillary junction medially to the piriform rim or lateral nasal wall depending on where the cut is desired. Care should be exercised to maintain at least 5 mm of distance from the tooth root apices and horizontal osteotomy. Prior to horizontal osteotomy, internal reference

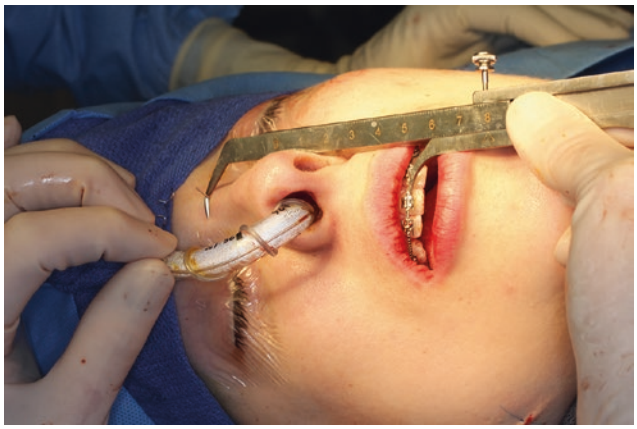


Fig. 25.8 The vertical height of the maxilla is confirmed using a caliper which measures from the K-wire to the orthodontic bracket on the central incisor

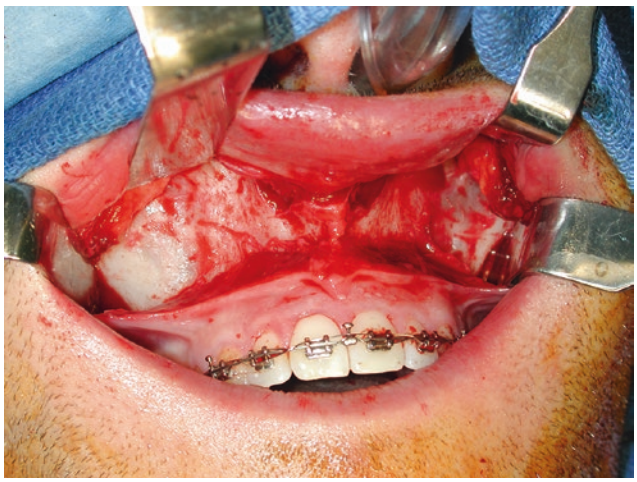


Fig. 25.9 Incision is made in alveolar mucosa 5 mm above the keratinized gingiva. This cuff of alveolar mucosa allows for proper closure following surgery

points may be scored on the bony surface but are not always necessary. Next, separation of the nasal septum, lateral nasal walls, and pterygoid plates is accomplished in that order. A spatula or straight edge osteotome is used to begin separation of the nasal septum from the anterior nasal spine. The septal osteotomy is completed by the use of a double-guarded septal osteotome. It is important to maintain contact with the bony nasal floor to prevent tearing of the nasal floor mucosa. Drive the osteotome inferiorly and posteriorly with a mallet while keeping one finger on the posterior aspect of the palatine bone for spatial reference. Lateral nasal wall osteotomies are completed using single-guarded osteotomes. A mallet is used to drive the osteotome posteriorly and parallel to the nasal floor until resistance is met at the pyramidal process of the palatine bone. The final osteotomy is of the pterygoid plates, performed with a curved osteotome placed at the bony junction of the

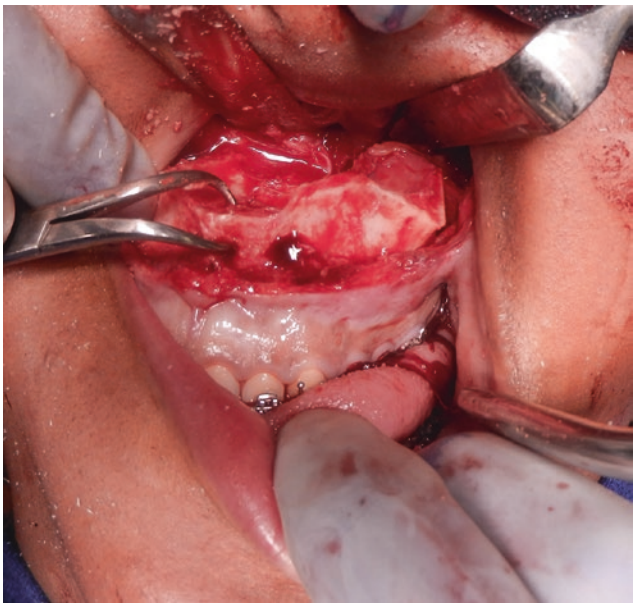


Fig. 25.10 Bony reduction of nasal septum, tuberosity, pyramidal process, and zygomatic buttress is performed to allow proper condylar positioning prior to fixation

pterygoid plate and posterior maxilla. A mallet is used to drive the osteotome in an inferior and medial direction. A finger can be placed intraorally near the tuberosity for spatial orientation. Hypotensive anesthesia can be utilized to minimize intraoperative blood loss. At this point, maxillary down fracture is initiated using manual pressure by distraction of the anterior maxilla inferiorly. If resistance is encountered, refine the osteotomies in the areas where resistance is met. Once the down fracture is complete, Rowe disimpaction forceps or Tessier mobilizers may be utilized to pull the maxilla down and forward. The maxilla must be mobilized freely from all of its bony attachments. The soft tissues of the nasal floor and posterior maxilla should be protected from trauma during this mobilization using Seldin retractors or Obwegeser retractors. Following down fracture and mobilization (Fig. 25.10), all bony interferences are removed that inhibit the forward positioning of the maxilla. This usually involves reduction ostectomy of the maxillary bony septum, posterior tuberosity regions, pyramidal process and lateral maxillary wall, or zygomatic buttress areas. Once mobilization of the maxilla and interferences have been removed, the segmental surgery of the maxilla is completed. Depending on the surgical plan, widening of the maxilla is accomplished using a 2 or 3 piece modification of the traditional Lefort I osteotomy. Interdental osteotomies are made with a fine-tapered fissure bur, oscillating saw or piezoelectric unit (Fig. 25.11). Spatula osteotomes are used for refinements (Fig. 25.12). Sagittal or paramedian cuts in the posterior maxilla connect to anteriorly based interdental vertical cuts in the premaxilla to allow mobilization of the segments (Fig. 25.13a–c). The inter-

dental osteotomies should be outlined prior to downfracture to allow a precise osteotomy in the maxilla that is stable rather than mobile. Palatal incisions may be implemented to allow more definitive mobilization of the maxillary segments. (Fig. 25.14a, b). This incision is paramedian and made over supported bone, not the osteotomy to prevent postoperative oronasal fistula. Final refinements are carried out after the surgical splint has been placed. Acrylic surgical splints which are

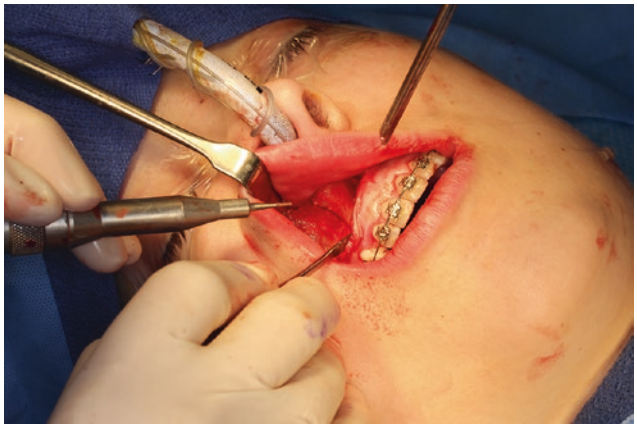


Fig. 25.11 Maxillary segmentation is performed using monocortical interdental osteotomies

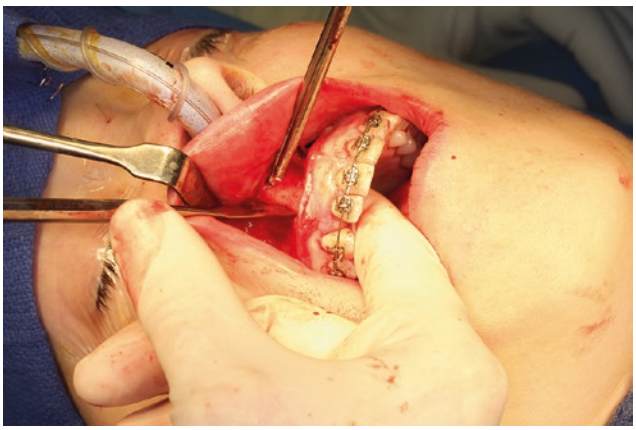


Fig. 25.12 The initial monocortical osteotomy is completed using spatula osteotome. The index finger is placed on the palate. Care should be taken to avoid perforation of palatal soft tissue

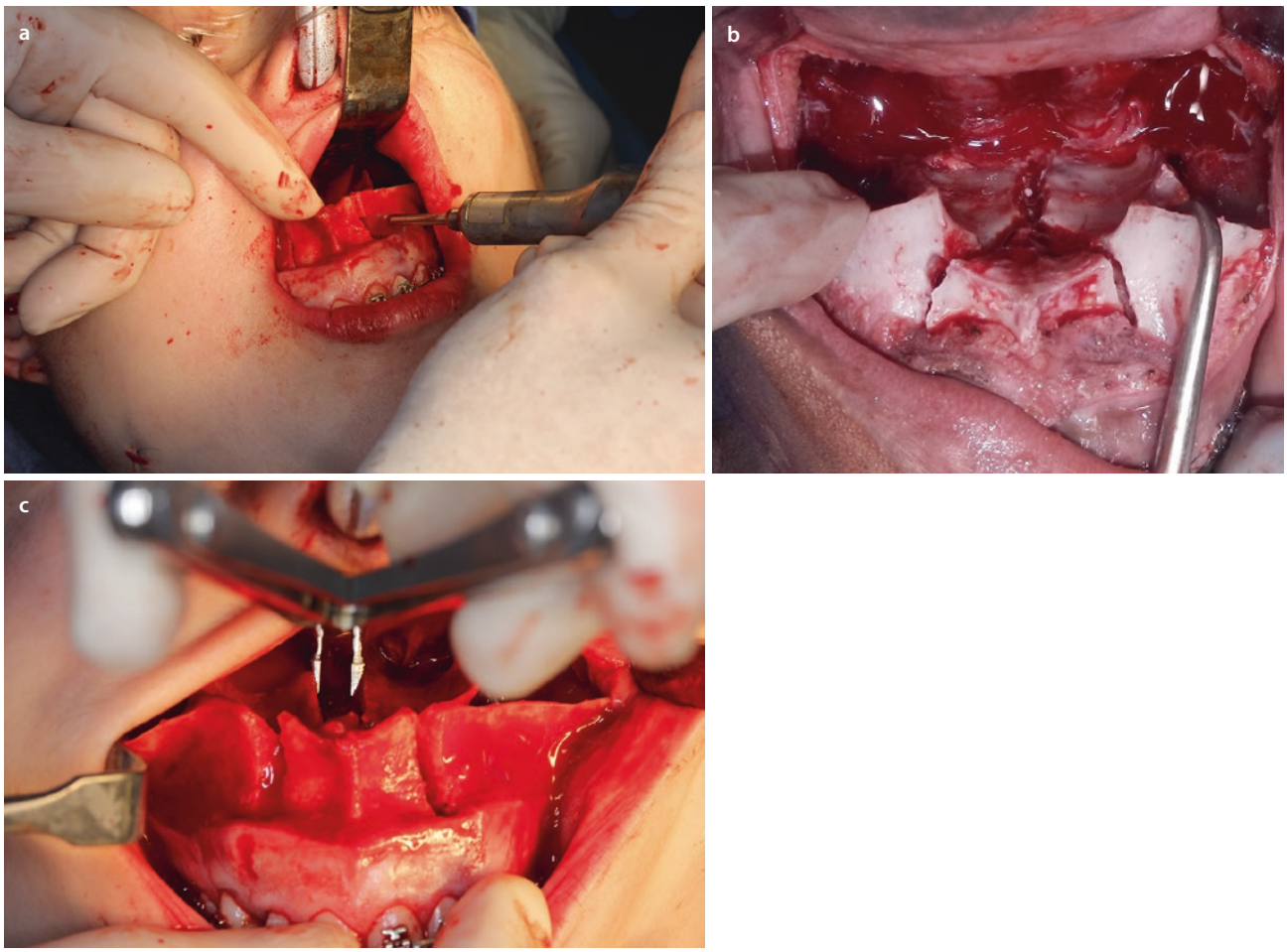
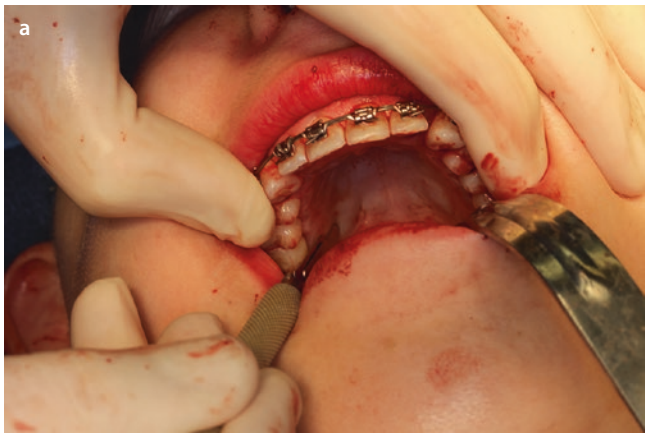


Fig. 25.13 Sagittal osteotomies are performed midline or paramedian where the bone is slightly thinner. These osteotomies are connected with the interdental osteotomies. The maxilla can be widened with Turvey maxillary expander



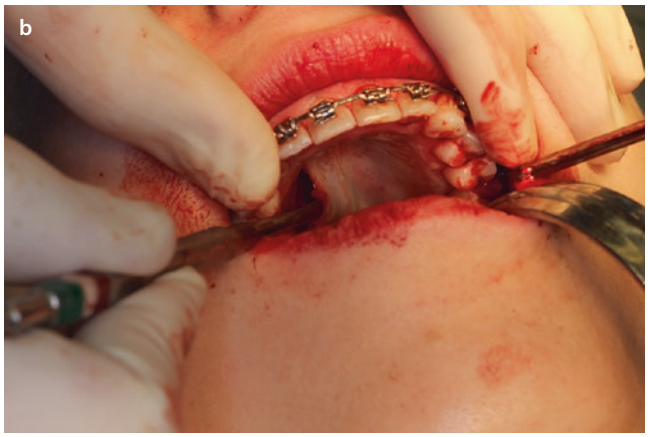


Fig. 25.14 Widening of the maxilla greater than 5 mm will require palatal releasing incisions to undermine the soft tissue prior to bony expansion. These incisions are made over supported bone. The soft tissue is undermined to midline. Passive maxillary expansion without stretching palatal soft tissue is important to prevent tearing which will result in oronasal perforation

fabricated preoperatively on the articulated models are ligated against the maxillary arch with 26-gauge wire. With segmental surgery, care must be taken to ensure that all pieces are securely ligated to the splint passively. The mandible is then wired into the splint. This allows the manipulation of the maxilla and mandible as a single unit. While attempting to seat the mandibular condyle upward, posterior pressure is applied in a posterior and superior vector and final interferences can be identified and removed. Vertical reference is verified using a caliper (Fig. 25.8). Rigid fixation is performed using mini-plates at the buttress regions of the zygoma and piriform (Fig. 25.15). Incision closure is performed using V-Y suturing to optimize aesthetic outcome (Fig. 25.16). A single skin hook is used to retract the superior margin of the incision upward. The vertical midportion of the incision is closed first, followed by standard vestibular closure of the remainder of the incision. This closure seeks to prevent foreshortening of the upper lip.

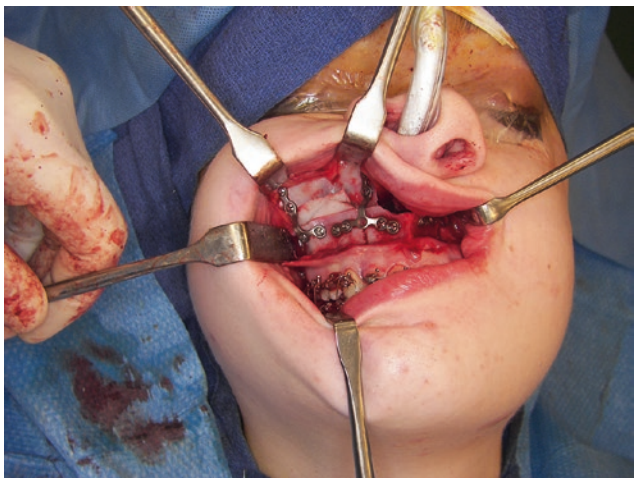


Fig. 25.15 Passive mini-plate adaptation and drilling holes center mass prevents plate or segment shifting during fixation

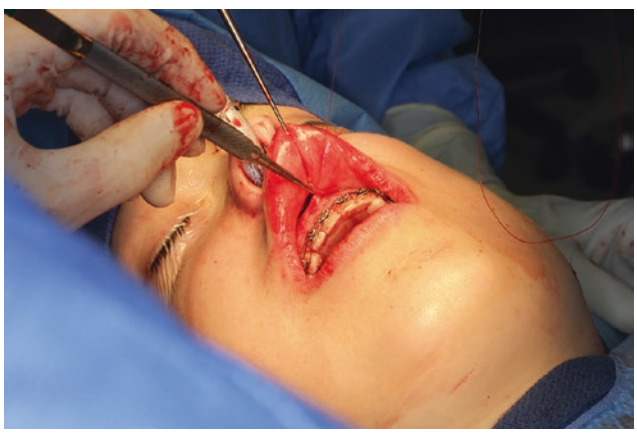


Fig. 25.16 V-Y closure minimizes lip shortening postoperatively. Superior retraction allows closure of the vertical midportion of incision first. This is followed by standard vestibular closure of remaining incision

25.4 Surgically Assisted Rapid Palatal Expansion (SARPE)

Transverse maxillary expansion by growth modification or surgical separation of the midpalatal suture is often used as a reliable correction for dentofacial deformities. The benefits in treating OSA using SARPE applications involve increasing the transverse width of the nasal and upper airway. The decision to perform SARPE versus rapid maxillary expansion (RME) is solely a question of growth potential. Surgical options are usually recommended beyond the age of 16 [45]. Although not considered first-line therapy of OSA treatment, it has been shown to be an effective alterna-

tive to traditional Lefort I surgery in patient with a mild AHI index and can be employed when medical management is not an option. In addition, a benefit to SARPE treatment is that it can often be employed as an outpatient setting.

The surgical technique of SARPE shares many of the same principles with Lefort I osteotomy. The surgery begins with injection of local anesthetic with a vasoconstrictor. A vestibular incision is made from first molar to the canine region. The incision is not carried across the midline. The same principles apply where a 5 mm cuff of mucosal tissue preserved superior to the mucogingival junction to allow for proper closure. Full thickness mucoperiosteal elevation is accomplished around the piriform, zygomaticomaxillary, and pterygomaxillary regions. The mucosa of the lateral nasal wall and floor of the nose is also elevated and protected. A horizontal osteotomy is then made using a reciprocating saw starting at the zygomaticomaxillary buttress area and extending medially through the lateral maxilla to the piriform region. A back cut through the tuberosity can also be made while protecting the pterygomaxillary area with an Obwegeser retractor. The same principle of keeping a safe distance of 5 mm from the teeth root apices applies during this horizontal osteotomy (Fig. 25.17). Next, a midline vertical incision is made over the anterior nasal spine and extended inferiorly into the keratinized tissue of the gingiva between the central incisors. Subperiosteal reflection identifies the piriform, anterior nasal spine, and tooth root eminences of the central incisors. A thin oscillating saw is used to make an osteotomy into the midpalatal suture using care not to perforate the palatal mucosa. A thin spatula osteotome is then used to complete the osteotomy driving the osteotome posteriorly through the palatal bone with a mallet using a finger intraorally for orientation (Fig. 25.18). Separation of the suture is verified by opening the Hyrax expander. If the segments are not mobilized, the decision may be made to separate the pterygomaxillary attachment using a curved osteotome. Following mobilization of the maxilla, closure is achieved with a 4.0 chromic suture.

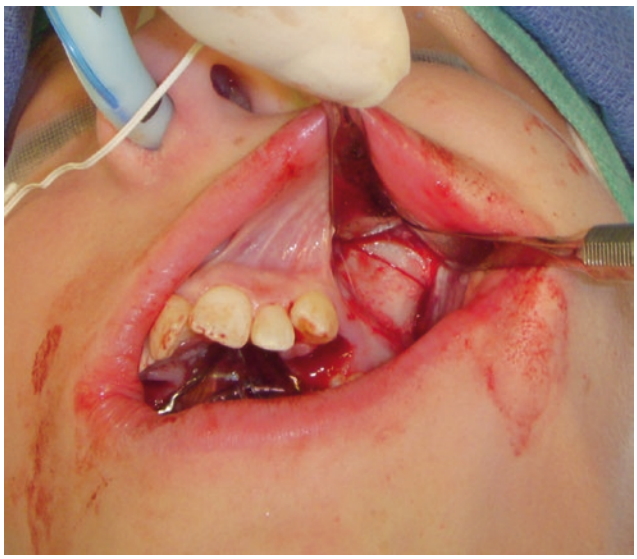


Fig. 25.17 SARPE: Horizontal osteotomy maxilla is performed 5 mm superior to apex of tooth roots

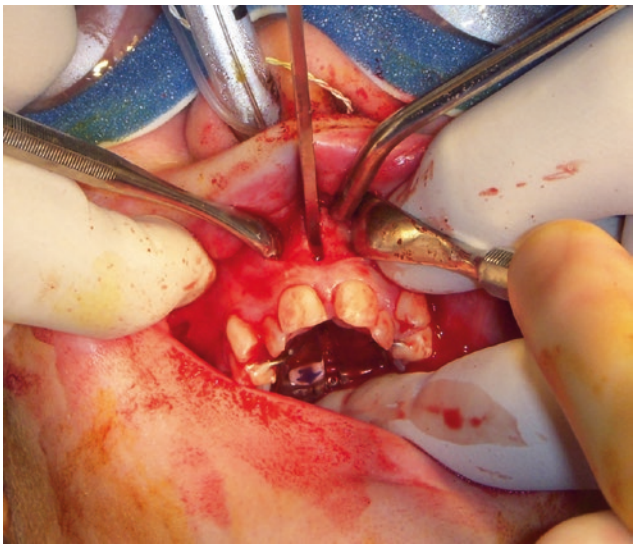


Fig. 25.18 SARPE: Sagittal osteotomy is performed through a vertical incision. The osteotomy is performed using an oscillating or reciprocating saw. An osteotome is placed in the osteotomy and directed posteriorly to complete division of maxilla into separate halves

25.5 Adjunct Procedures

In order to decrease the airway resistance, a number of modifications to the maxilla and surrounding structures may also be implemented. These include easy access for inferior turbinectomy, septoplasty, and nasal floor contouring. With the maxilla downfractured, the inferior turbinate is directly visualized and can be removed with a large hemostat and Dean scissors. In cases where septal deviation is encountered septoplasty may also easily be accomplished. Also as noted, OSA can be related to a constricted maxilla. In these cases, attention should be directed to the alar base and corresponding bony piriform which are likely also to be constricted. With surgical exposure of the piriform, bony contouring may be accomplished using a round bur or pineapple bur for osteoplasty or ostectomy with the objective of widening of the piriform to improve nasal airflow (Figs. 25.19a, b).

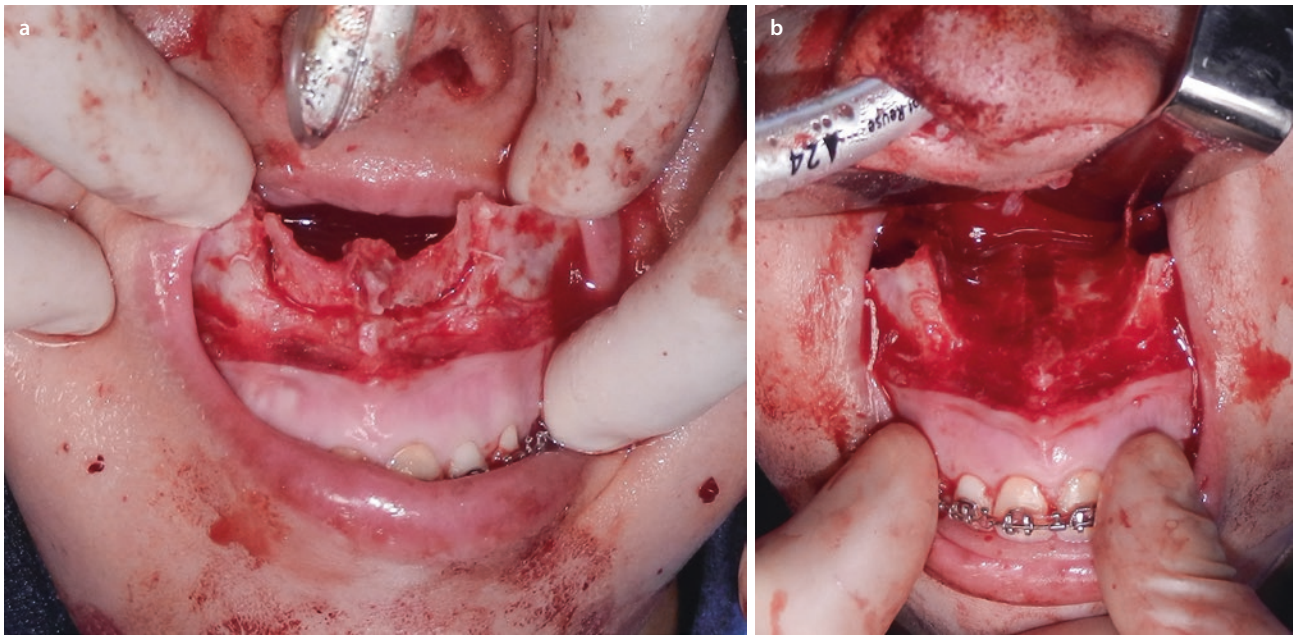


Fig. 25.19 Nasal aperture before and after widening using bur to contour bone at piriform aperture

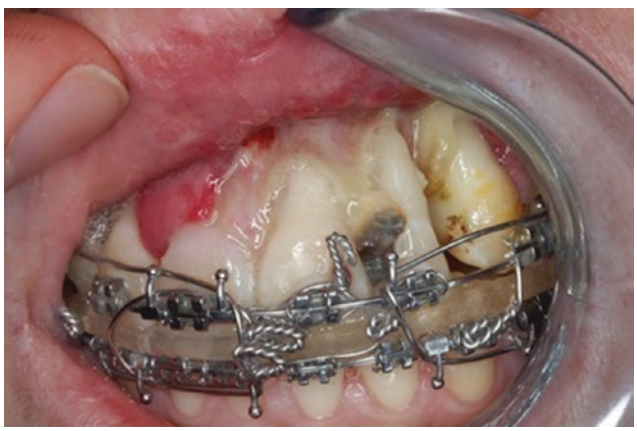


Fig. 25.20 Mild vascular insufficiency postoperatively results in necrosis of gingival soft tissue. These patients will have bony union but soft tissue defect which will require additional surgery for soft tissue grafting

25.6 Complications

Immediate complications may include bleeding, unfavorable fractures in the segmented maxilla, trauma to teeth or root apices, malocclusion from unidentified interferences, or septal deviation. With final positioning and plating of the maxilla, it is important to be mindful of the final position of the septum, as the final position of the maxilla may cause septal deviation. Septal position may even be altered during extubation.

Delayed complications may include bleeding from Pseudoaneurysm which can be severe requiring embolization via interventional radiology. Devitalization of teeth occurs when an osteotomy is performed too close to a tooth. It will often demarcate within weeks and require endodontic therapy. Hardware exposure can occur and will usually re-mucosalize with oral antibiotics, daily irrigation, and vigilant oral hygiene.

The two most difficult complications are infection and vascular insult. Mild infection can be treated as an outpatient with oral antibiotics and daily irrigation. More severe infection is an indication to return to the operating room for surgical irrigation or hardware exchange. Mild vascular insufficiency will result in gingival necrosis and may been seen in smoking patients or after ligation of descending palatine arteries in segmental osteotomy (Fig. 25.20). Maxillary hypoperfusion from arterial injury or impingement which is prolonged may result in maxillary segment loss. It can also result from vascular congestion due to injury to the venous drainage. Prompt identification is imperative. Reentry with hardware removal can allow for reperfusion. Hyperbaric oxygen therapy may also be considered if vascular insufficiency is identified early. Failure to identify this or inability to correct it may eventually result in bony necrosis (Fig. 25.21). Necrotic bone must be removed and grafting procedures are often required to correct the resulting defect.

Lastly, this patient population often presents with a constellation of medical issues from OSA. A sick patient with OSA and failed phase I therapy may not be a medically optimized patient for major surgery. Care should be exercised postoperatively where myocardial infarction, stroke, renal insufficiency, or other medical emergencies may be confronted.

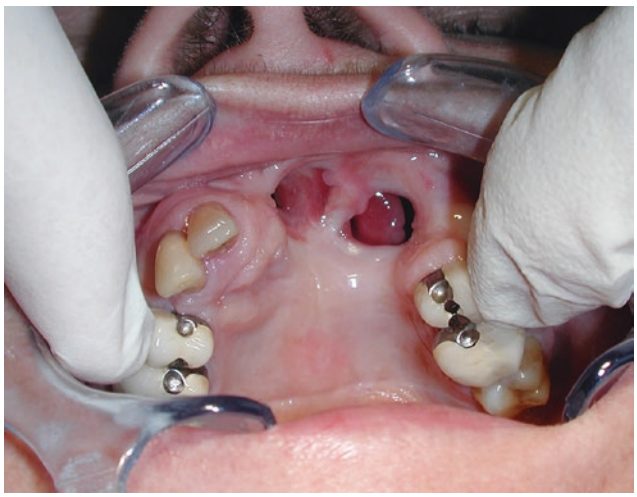


Fig. 25.21 Severe vascular insult will result in loss of teeth and or segment

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Mandibular Surgical Procedures

Larry Wolford

Contents

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Patients with obstructive sleep apnea (OSA) commonly have associated mandibular deformities, particularly mandibular hypoplasia (retrusion) that can create a decreased oropharyngeal airway contributing to OSA. The OSA patient may present with a retruded mandible (often associated with a retruded maxilla and a high occlusal plane angle) that can displace the tongue posteriorly into the oropharyngeal space, severely compromising the functional airway. OSA patients may benefit from orthognathic surgery to advance the maxillomandibular complex forward, usually in a counterclockwise rotation direction that predictably increases the oropharyngeal airway. Although combined maxillary and mandibular osteotomies for advancement are usually required for correction of OSA involving a decreased oropharyngeal airway, this chapter focuses only on various mandibular osteotomies that may be incorporated in the management of the adolescent and adult OSA patient. Maxillary osteotomies are presented in another chapter. Also, mandibular distraction procedures are not addressed in this chapter. Particular attention is directed to the mandibular ramus sagittal split osteotomy, as this is the primary osteotomy technique for advancing the mandible, so essential to predictable outcomes in the OSA patient.

The Oral and Maxillofacial Surgeon should have experience with mandibular and maxillary procedures and a thorough understanding of reasonable treatment goals in order to develop a plan that provides optimal functional and aesthetic results [1]. The surgeon must be aware of the potential risks and complications that can occur with each of the mandibular procedures. This knowledge enables the surgeon to develop an optimal treatment plan and alternative treatments according to his or her skill level. The surgeon should communicate to the patient the existing problems, the magnitude of these problems, the recommended treatment, alternative treatment options, and the potential risks and complications.

The surgical procedures that will be described include the following:

1. Genioplasty
2. Subapical osteotomies
3. Body osteotomies
4. Ramus osteotomies

26.1 Genioplasty Procedures

Genioplasty procedures can alter the position of the chin in all three planes of space. Chin position most commonly is changed by the use of a sliding horizontal osteotomy or by placement of an alloplastic implant. For OSA patients, an augmentation genioplasty may be indicated to advance the suprahyoid muscles, or enhance the facial balance.

26.2 Osseous Genioplasty

When the bony chin is to be repositioned, a soft tissue pedicle must be maintained to ensure viability to the osteotomized segment. The traditional horizontal osteotomy (Fig. 26.1a) can be used and stabilization can be achieved by wiring, bone screws, and/or bone plates.

26.2.1 Anteroposterior Augmentation

OSA patients may benefit from an osseous augmentation genioplasty. Not only can the aesthetic benefit occur, but advancing the chin will also increase tension on the supra-hyoid muscles that may have some positive effect, although relatively minor, of pulling the base of the tongue further forward. The usual limiting factor for chin advancement is the anteroposterior dimension of the symphysis, unless the osteotomized segment is tiered surgically. If the chin is narrow transversely, advancement tends to make the face appear more tapered. A-P soft tissue change is approximately 80–85% of the amount of bony chin advancement (Fig. 26.2a).

26.2.2 Surgical Procedure

The surgical procedure involves an anterior vestibular incision from cuspid to cuspid, and reflection of the mentalis muscles and periosteum off of the anterior and inferior border of the bony chin. A horizontal osteotomy is performed generally starting 4–5 mm below the mental foramen, at the inferior border, tapering forward to a level of 1–1 ½ cm above bony menton. At this level, the genial tubercles and associated muscles remain attached to the distal segment providing a vascular supply. Once the segment is mobilized and advanced, there are various methods to stabilize the segment, with the

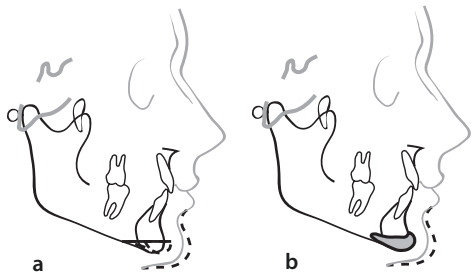

Fig. 26.1 **a** An osseous genioplasty can be used to augment the chin, move it posteriorly, alter its vertical position, or change the transverse position of the chin. **b** Alloplastic implants can be used to augment the chin anteriorly and laterally

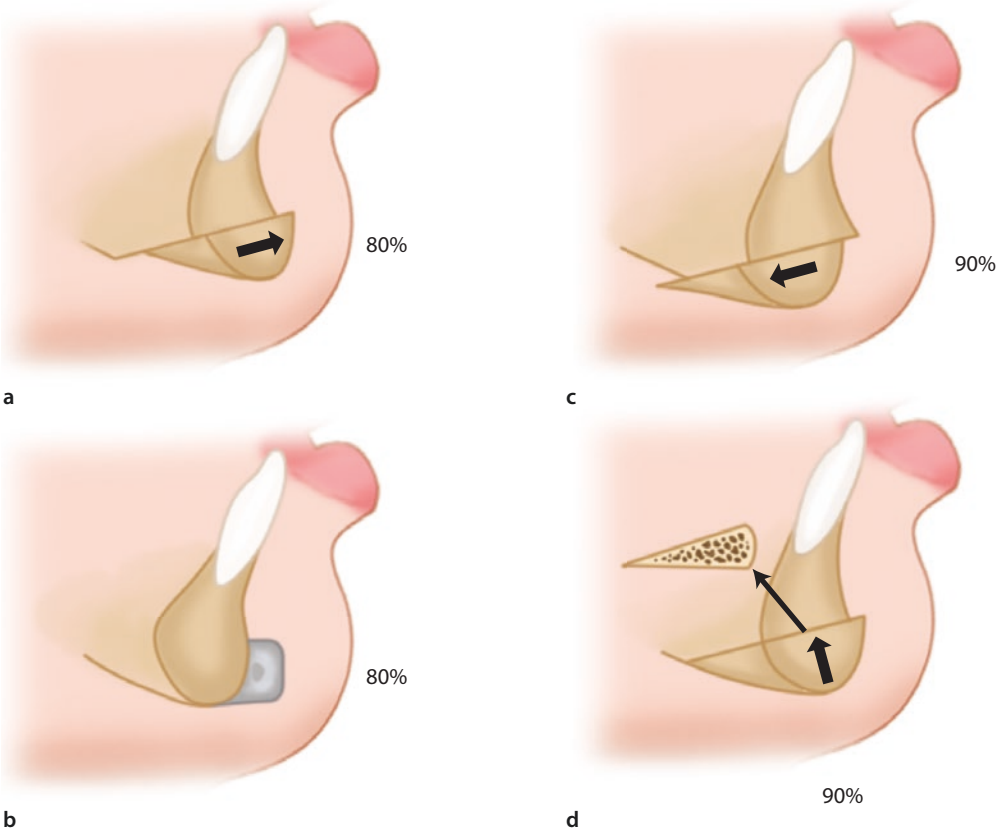

Fig. 26.2 Soft tissue changes associated with genioplasty procedures vary depending on the surgical method and direction of movement. **a** Bony augmentation genioplasty will change the soft tissue projection 80–85% of the amount of bony advancement. **b** Alloplastic augmentation to the chin will advance the soft tissues forward approximately 80–85% of the implant thickness. **c** Osseous genioplasty to position the chin posterior will result in approximately 90% soft tissue change relative to the amount of chin setback. **d** Vertical reduction of the chin by removing a bony wedge from above the horizontal genioplasty cut will create approximately a 90% vertical movement of the soft tissue relative to the amount of osseous vertical reduction

most common using a chin bone plate, available in various lengths (3, 5, 7, and 9 mm) that accommodates most chin advancement procedures (Fig. 26.3). The incision is closed in two layers, reattaching the mentalis muscles and then closing the mucosa.

26.2.3 Anteroposterior Reduction

In some cases, the chin may be too strong and require reduction to maximize the aesthetic result. In redo orthognathic cases, sometimes a previous excessively advanced genioplasty was done as a compensatory aesthetic procedure to increase the prominence of the chin, without properly correcting the dentofacial deformity and associated OSA. In this situation, the subsequent counterclockwise rotation of the maxillomandibular complex will render the chin too strong, requiring an A-P reduction genioplasty. The surgical approach is the same, but with repositioning the chin posteriorly. This may allow the tongue to settle posteriorly a minor amount. Optimal soft tissue change at pogonion is achieved by performing a horizontal sliding osteotomy and moving the chin and attached soft tissues posteriorly. The chin usually appears wider after this procedure, and the labiomental fold decreases. Soft tissue change, if soft tissue remains attached to the anterior and inferior aspect of the chin, is usually 90% of the anteroposterior bony reduction (Fig. 26.2c). Shaving of the anterior aspect of the bony chin to reduce the A-P prominence may result in only 20–30% posterior movement of the soft tissue in relation to the amount of bone removed, as the soft tissues tend to thicken following this approach.

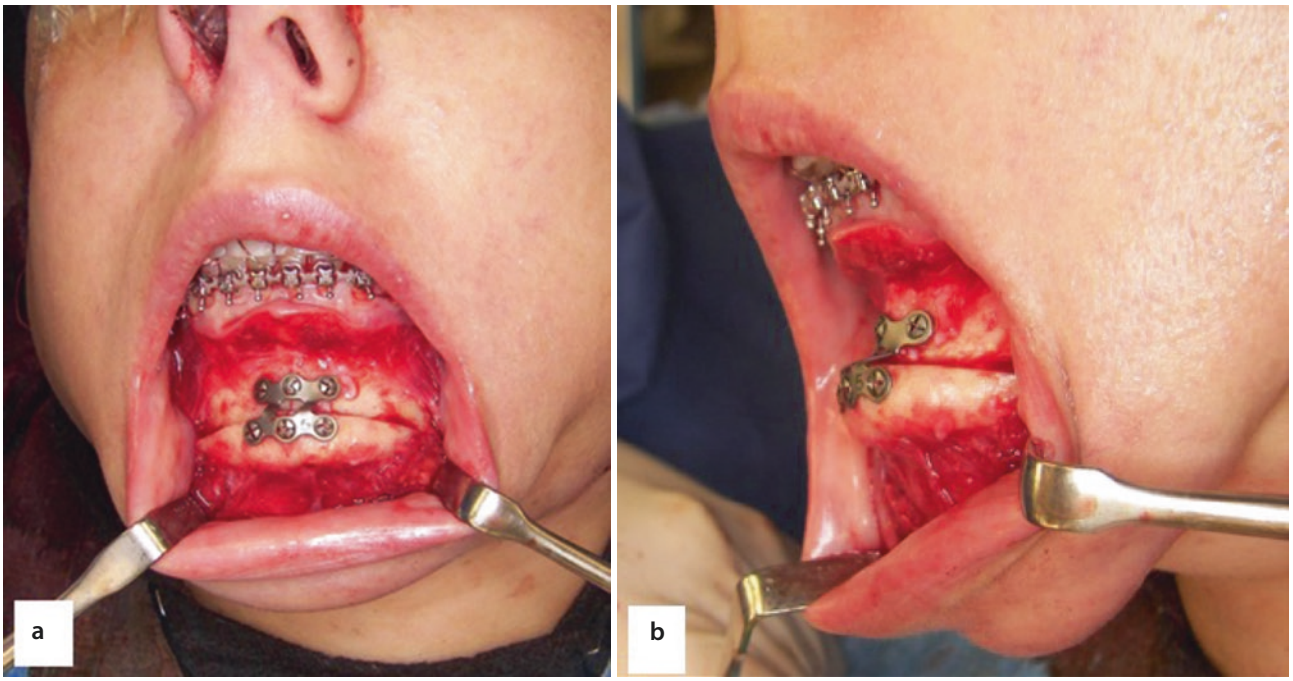

Fig. 26.3 **a** Specially designed chin plates can be used to support the advanced chin segment. The plates come in the sizes of 3, 5, 7, and 9 mm lengths. **b** Lateral view of a 5 mm chin advancement with a chin plate

26.2.4 Vertical Augmentation (Downgraft)

Vertical augmentation can be accomplished with a horizontal osteotomy and inferior repositioning of the chin segment, applying rigid fixation. This technique usually requires bone or synthetic bone grafting between the proximal and distal segments. Vertical soft tissue change is approximately 100% of the osseous change.

26.2.5 Vertical Reduction

The most predictable method to vertically reduce the chin height is with a wedge resection and rotation of the inferior chin segment superiorly. When the soft tissue remains attached to the inferior border, the soft tissue change is approximately 90% of the vertical osseous change (Fig. 26.2d). If the vertical reduction is performed by resecting and removing the inferior border, then the vertical soft tissue change is only 25–30% of the amount of bone removed.

26.2.6 Age for Osseous Genioplasty

Osseous genioplasty is best performed after 12 years of age to allow for eruption of the permanent mandibular canines and premolars to lessen possible damage to the roots.

26.3 Alloplastic Augmentations

Various synthetic materials have been used to augment the chin (Fig. 26.1b). Rigid stabilization of implants is important because mobility may result in malposition, bone resorption, and infection. Most infections of chin implants occur when there is improper fixation or inadequate soft tissue closure. The following recommended technique is safe and provides good long-term stability:

1. Perform the chin implant as the last step, after all other orthognathic procedures are completed and the associated incisions are closed.
2. After exposure and preparation of the implant area, thoroughly irrigate with sterile saline with a final irrigation with betadine solution.
3. Change gloves and wash off glove powder before handling the implant.
4. Stabilize the implant to the mandible to eliminate mobility and migration by using bone screws, plates, or intraosseous wiring.
5. Irrigate the surgical area thoroughly, use a final rinse of betadine, and close the incision in two layers with reapproximation of the mentalis muscle layer and tight mucosal closure.

Although many alloplastic materials have been used for chin augmentation, one currently recommended is Medpor (Porex, Newnan, GA); a porous polyethylene,

preformed implant with a selection of sizes and designs (Fig. 26.4a, b). It is recommended that alloplastic augmentation genioplasty be performed after eruption of the mandibular anterior teeth.

26.3.1 Surgical Procedure

The surgical procedure involves an anterior vestibular incision from cuspid to cuspid, and reflection of the mentalis muscles and periosteum off of the bony chin. The chin implant is contoured as necessary to provide the desired aesthetic result. It is inserted and stabilized with bone screws (Fig. 26.4c). The surgical area is thoroughly irrigated with saline and a final rinse of betadine solution. The incision is closed in two layers, reattaching the mentalis muscles and then a tight closure of the mucosa.

26.4 Genioplasty Complications

Several potential complications are associated with osseous and alloplastic chin augmentation:

- Loss of osteotomized bone segment
- Bone resorption
- Infection
- Loss of implant
- Displacement/malalignment
- Paresthesia/anesthesia of lower lip and chin
- Lower lip ptosis
- Mentalis muscle dysfunction
- Unsatisfactory aesthetic outcome

Loss of the osteotomized bone segment may occur following avascular necrosis. Avascular necrosis usually occurs because of loss of soft tissue attachment or infec-

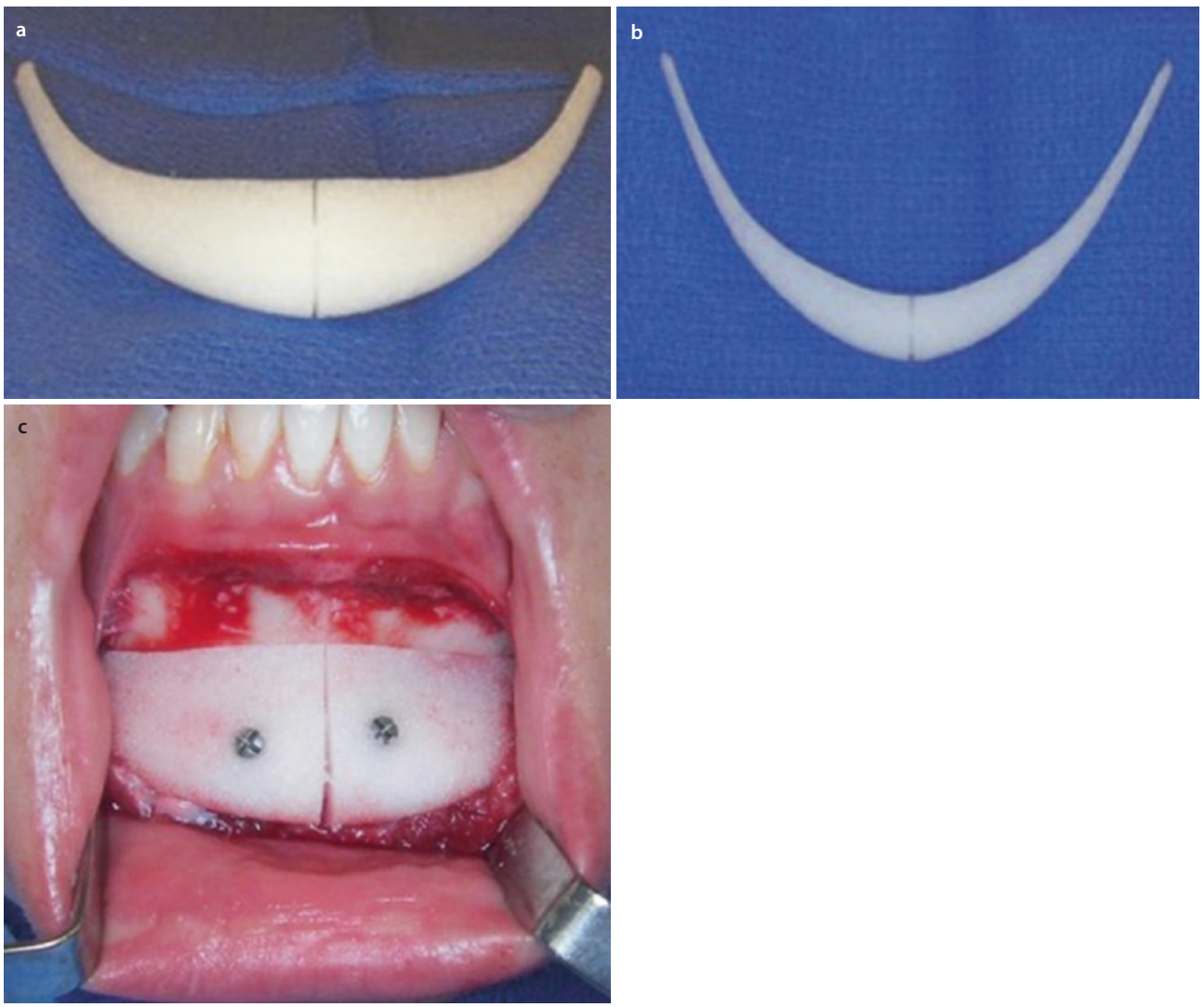

Fig. 26.4 **a**, **b** A Medpor chin implant to augment the chin anteriorly as well as enhance the lateral profile of the chin and mandible. **c** Chin implant in position stabilized with two bone screws. Soft tissues require closure in two layers including resuspension and attachment of the mentalis muscle and watertight closure of the mucosal layer

tion. Loss of the segment may require further alloplastic or bone graft reconstruction. A large amount of bone resorption can be expected if a free bone graft is used to augment the chin or if the soft tissue pedicle to the mobilized chin segment is lost. Pedicled osseous genioplasties usually undergo anterior bone resorption of about 10–20%. Infection most commonly is caused by avascular necrosis, contamination, and wound breakdown. Displacement of the alloplast may occur following trauma or inadequate stabilization. This may require additional surgery to restabilize the implant. Lower lip ptosis may be caused by inadequate positioning, resuspension, and stabilization of the mentalis muscle and associated soft tissues. Normally, when relaxed, the lower lip should be in level with the lower incisor edges. Correction of lower lip ptosis requires repositioning and resuspension of the mentalis muscles. Anesthesia or paresthesia of the lower lip and chin may result from trauma to the inferior alveolar and/or mental nerve branches from incision design, dissection, retraction, or direct injury when performing osteotomies. Nerve injury may be avoided by careful incision placement, careful dissection, minimal nerve retraction, and carefully planned bone cuts. If nerve transection is directly visualized, immediate repair is indicated.

26.5 Mandibular Subapical Procedures

These procedures are designed to alter portions of the mandibular dental alveolus and can be divided into three types: anterior, posterior, and total subapical osteotomies. Only the anterior subapical procedure will be discussed here as it can be used to correct a malpositioned anterior dental segment such as an accentuated or reverse curve of Spee involving the anterior mandibular arch.

26.5.1 Anterior Mandibular Subapical Osteotomy

Osteotomy design involves vertical interdental osteotomies joined by a horizontal osteotomy at least 5 mm below the apices of the associated teeth (Fig. 26.5). A mandibular horizontal vestibular incision is used for access. The vascularity to the mobilized segment is maintained by the lingual soft tissue pedicle. Indications for anterior mandibular subapical osteotomy include leveling the occlusal plane, changing the anteroposterior position of the mandibular anterior teeth, correcting asymmetries, and changing the axial angulation of the mandibular anterior teeth.

Presurgery orthodontics may be required to diverge the roots of teeth adjacent to the vertical osteotomies to minimize damage to the roots with the subsequent bone cuts. Bone screws, interosseous wiring, or bone plates can be used to stabilize the bone segments. Soft tissue closure is achieved by suturing the muscle layer first to resuspend the lower lip and then a tight mucosal closure.

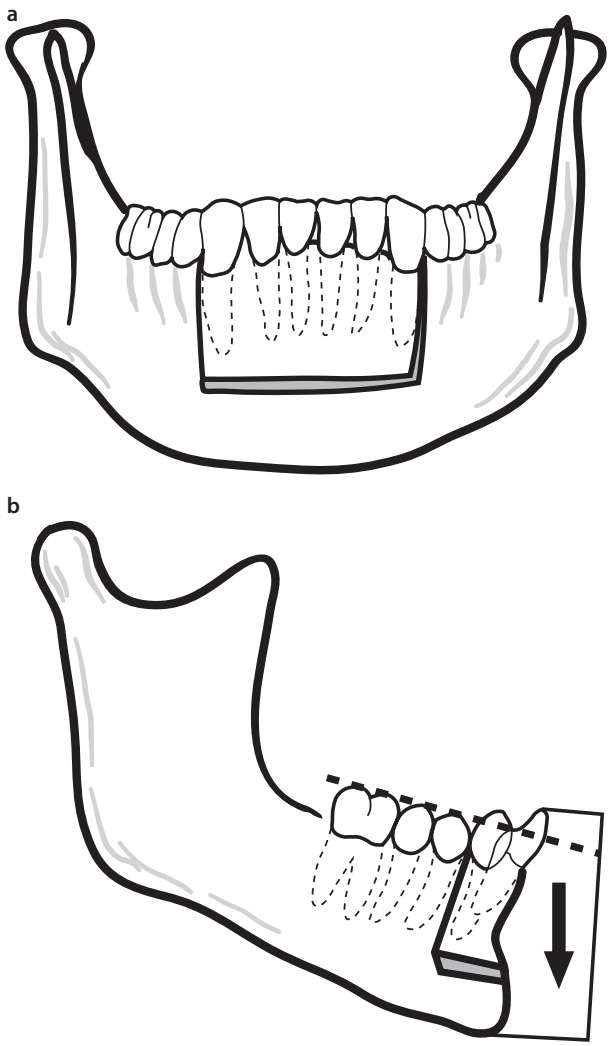

Fig. 26.5 ubapical osteotomy. **a**, **b** Vertical interdental osteotomies are performed with a connecting horizontal osteotomy or ostectomy positioned at least 5 mm below the apices of the teeth to minimize risk of dental devitalization. The segment can be stabilized with bone plates, interosseous screws, or wire fixation

26.5.2 Age for Surgery

Although no studies refer to the vertical growth effects of interdental osteotomies, it is recommended that surgery be performed in females after the age of 14 and males after the age of 16.

26.5.3 Possible Complications

Potential complications include root amputation, tooth ankylosis, periodontal defects, or loss of teeth and bone. Severe periodontal problems also may result from excessive removal of interdental bone. Major changes in vertical position may worsen preexisting periodontal problems in the area of vertical osteotomy. Additional complications include lower lip paresthesia/ anesthesia, lower lip ptosis, and pathological fracture. Anesthesia or paresthesia of the lip, teeth, and gingiva may result from trauma of the inferior alveolar or mental neurovascular bundle. This usually resolves in a few weeks to several months. If the neurological deficit lasts longer than 1 year, the prognosis for recovery is poor. If the nerve is severed, primary repair gives the best result. Teeth and gingiva in the subapical segment commonly exhibit an extended period of anesthesia or paresthesia.

26.6 Mandibular Body Surgery

Mandibular body surgery can be divided into anterior body and posterior body surgery. Anterior body surgery refers to osteotomies anterior to the mental foramen, including the symphysis area. Posterior body surgery involves osteotomies around and adjacent to the mental foramen area or further posterior in the body (Fig. 26.6). Posterior body surgery requires specific management of the inferior alveolar neurovascular bundle for its preservation. The basic indications for mandibular body osteotomies are (1) occlusal plane leveling, (2) mandibular setback, (3) removal of edentulous space or teeth and associated bone, (4) narrowing or widening of the mandible, (5) lengthening of the mandible, and (6) distraction osteogenesis. Contraindications include adjacent roots that are too close together and vascular compromise to adjacent soft tissue and bone. Perform the osteotomies so that there will be maximum bony interface following the repositioning of the segments. A significant bony gap created by the removal of too much bone may interfere with healing. Precise treatment planning in the model surgery and the prediction tracing is paramount for success in body osteotomies. Rigid fixation is preferred for stabilization of the segments.

26.6.1 Effects on Growth

Interdental osteotomies should not affect vertical alveolar growth, unless a tooth root is injured, resulting in dental ankylosis, which could result in deficient vertical dentoalveolar growth.

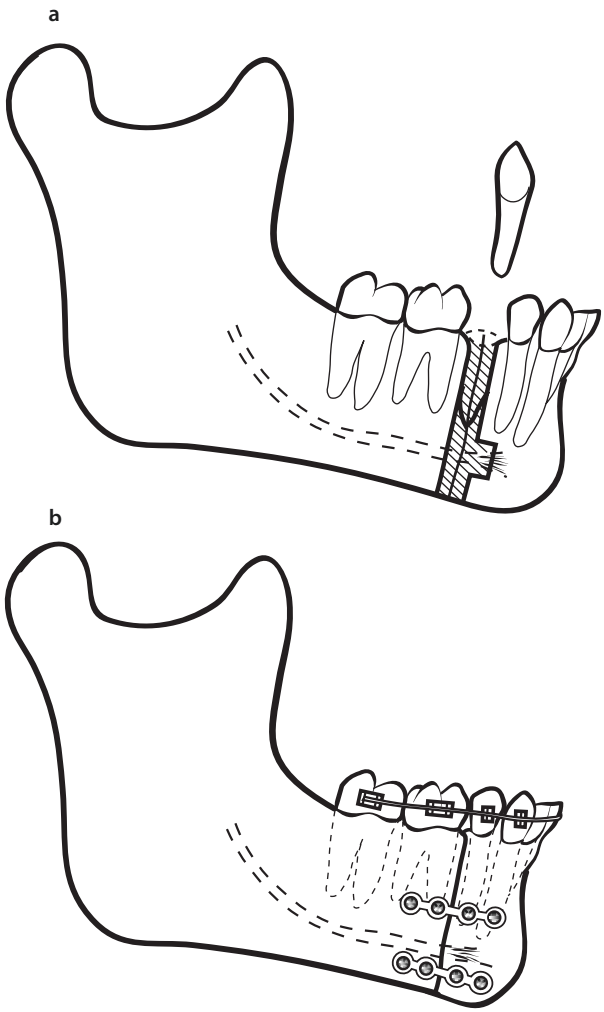

Fig. 26.6 **a** Vertical body osteotomies or ostectomies can be performed in any area of the mandible to move the anterior segment of the mandible posteriorly or to alter the vertical and transverse position. Combining body osteotomies and sagittal split osteotomies of the ramus allows flexibility in movement of the posterior and anterior segments independent of each other. **b** Rigid fixation improves the stability and facilitates healing

26.6.2 Age for Surgery

This surgery is recommended after the age of 14 in females and after age 16 in males.

26.7 Potential Complications for Mandibular Body Surgery

26.7.1 Nonunion or Malunion

Nonunion or malunion usually results from a poor bony interface, inaccurate position of the bony segments, or inadequate stabilization of the segments. Nonunion or

malunion may necessitate additional surgery to reposition and stabilize the segments and rigid fixation, with or without bone grafting.

26.7.2 Loss of Teeth and Bone

The loss of teeth and bone may occur because of vascular compromise, resulting in infection, osteomyelitis, or avascular necrosis. Vascular insufficiency can be devastating and may require hyperbaric oxygen treatment and secondary procedures that restabilize or reconstruct the bone segments.

26.7.3 Infections

Infection or osteomyelitis may require antibiotics and debridement. Infection is rare unless there is major damage done to the bone, teeth, and soft tissues during surgery. Hyperbaric oxygen therapy may be required as well as secondary reconstruction.

26.7.4 Periodontal Defects

Periodontal defects may occur as a result of vascular compromise, inadvertent removal of the cervical interdental bone, or major damage to the periodontal tissues. Defects also may occur by creating tears or vertical incisions in the osteotomy area.

26.7.5 Nerve Damage

Anesthesia or paresthesia of the lower lip, chin, and gums are the most common complications of mandibular body surgery. Generally, neurosensory deficit is temporary but can be permanent. Nerve damage usually is caused by edema and manipulation of the neurovascular bundle. In an anterior body ostectomy, where the anterior branch of the inferior alveolar nerve is sacrificed, the anterior teeth and gingiva may be numb for many months or permanently. If a major inferior alveolar or mental nerve injury is encountered during the surgery, immediate repair is indicated for the most predictable outcome.

26.7.6 Simultaneous Mandibular Ramus and Body Procedures

Simultaneous ipsilateral mandibular body and ramus procedures can be accomplished provided that the soft tissues are managed appropriately. Maintaining the integrity of the inferior alveolar neurovascular bundle, particularly in posterior segments, is important. Careful management and protection of the lingual tissues is also vital. When mandibular sagittal split ramus osteotomies are performed concomitant with mandibular body procedures, it is generally recommended to complete the sagittal split procedure before the body osteotomies. If the body osteotomies are performed first, even with rigid fixation, the prying forces necessary to complete the sagittal split may displace the body segments. If vertical oblique, or inverted L osteotomies are performed along with body osteotomies, either procedure may be completed first. Once the ramus and body osteotomies are completed, the occlusal splint can be used to align the segments appropriately for stabilization, preferably by rigid fixation. The body osteotomies are stabilized with rigid fixation first followed by the ramus rigid fixation.

26.8 Mandibular Ramus Surgery

Mandibular ramus sagittal split osteotomy is the most common mandibular orthognathic procedure for the OSA patient. This osteotomy technique originally was described by Trauner and Obwegeser in 1957 [2–4]. The bilateral sagittal split ramus osteotomy can be used for mandibular advancement or setback, correction of moderate asymmetries, control of the occlusion, and positioning of the condyle. The technique has undergone numerous modifications [5–10]. The procedure to be described herein, maximizes the bony interface by splitting the mandible at the inferior border, provides controlled positioning of the proximal segment and easy application of rigid fixation. (◘ Fig. 26.7) [11]. Even with large advancements, bone grafting rarely is required.

Indications for sagittal split ramus osteotomies include mandibular advancement, setback, and correction of mandibular asymmetries. Contraindications for mandibular ramus sagittal split osteotomy include severe decreased posterior mandibular body height, extremely thin medial-lateral width of ramus, severe ramus hypoplasia, absence of a condyle, and severe asymmetries.

Advantages include the following:

1. Healing is enhanced because of a good bony interface.
2. Can advance or set back the mandible, correct most asymmetries, and alter the occlusal plane, enabling counterclockwise rotation advancement of the maxillomandibular complex for the OSA patient.
3. Rigid fixation can be applied, eliminating the need for maxillomandibular fixation. Rigid fixation, when properly applied, significantly improves the stability and predictability of results. Bone plates with mono-

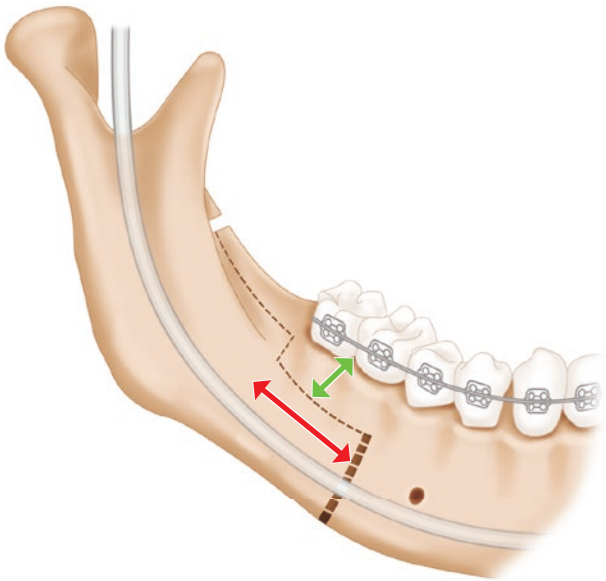

Fig. 26.7 Outline of the Wolford modification of the mandibular ramus sagittal split osteotomy. Green Arrow: Horizontal cut made 8–10 mm below the alveolar bone crest. Red Arrow: Horizontal osteotomy extends 8 mm longer than the amount of mandibular advancement to provide a bony interface between the proximal and distal segments

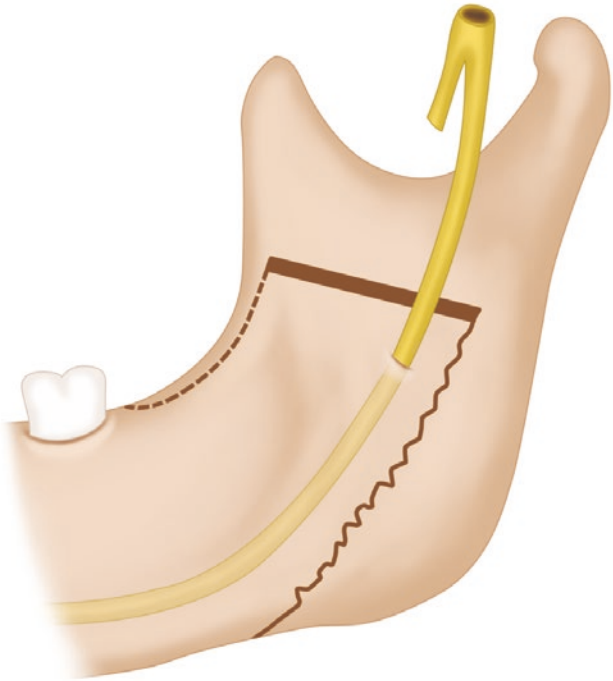

Fig. 26.8 Medial side of the ramus shows the position of the medial horizontal cut just above the lingula as well as the medial fracture line

cortical screws or bicortical bone screws can provide good stability.
4. Surgical modifications can maintain the angle of the mandible in the original spatial position, even in large advancements.
5. The muscles of mastication remain in their original spatial position.

Disadvantages include the following:
1. The incidence of nerve damage is increased compared to other techniques (i.e., vertical ramus osteotomies, inverted "L" osteotomies), although this is usually temporary.
2. Unfavorable splits may occur increasing difficulty with stabilizing.
3. Surgery must create a fracture on the lingual aspect of the ramus.
4. Severe asymmetries are more challenging to correct.

26.8.1 The Wolford Inferior Border Osteotomy Modification of the Mandibular Ramus Sagittal Split Procedure

This modification of the sagittal split procedure may address a number of the unfavorable features associated with the traditional osteotomy designs. This technique incorporates an inferior border osteotomy into the sagittal split design and provides a method to reposition the mandible with an effective means for easier splitting, greater advancement capabilities, decreased IAN involvement, positional control of the condyle and proximal segment, and easy application of rigid fixation [11]. The technique will be described.

1. The mandible is approached through an incision along the ascending ramus, extended forward to the first molar area, or further for larger mandibular advancements.
2. The medial monocortical osteotomy is performed through the lingual cortex just above the lingula, extending posterior to the lingula and mandibular foramen, using a short Lindenman bur (Fig. 26.8).
3. After initiating the vertical ramus cut with a #701 fissure bur, a thin bladed reciprocating saw is used to perform the osteotomy down the ascending ramus, adjacent to the buccal cortex, stopping just distal to the second molar (Fig. 26.9, yellow arrows).
4. A horizontal monocortical osteotomy is made with a #701 fissure bur, directed perpendicular to the buccal cortex, 8–10 mm below the alveolar crest (Fig. 26.9, green arrow, Fig. 26.10), and is extended anteriorly 8 mm greater than the amount of intended mandibular advancement (Fig. 26.9, red arrow). This cut subsequently creates a bony ledge on the distal segment that provides a vertical stop to control the position of the proximal seg-

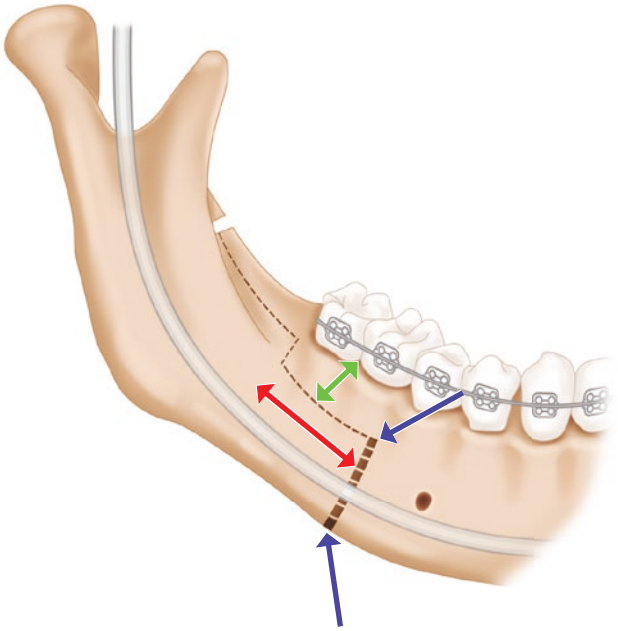

Fig. 26.9 The vertical buccal monocortical cut is completed with the osteotomy carried only halfway through the inferior border of the mandible. This cut is done with a 703 burr to create enough space to insert the sagittal splitting instruments

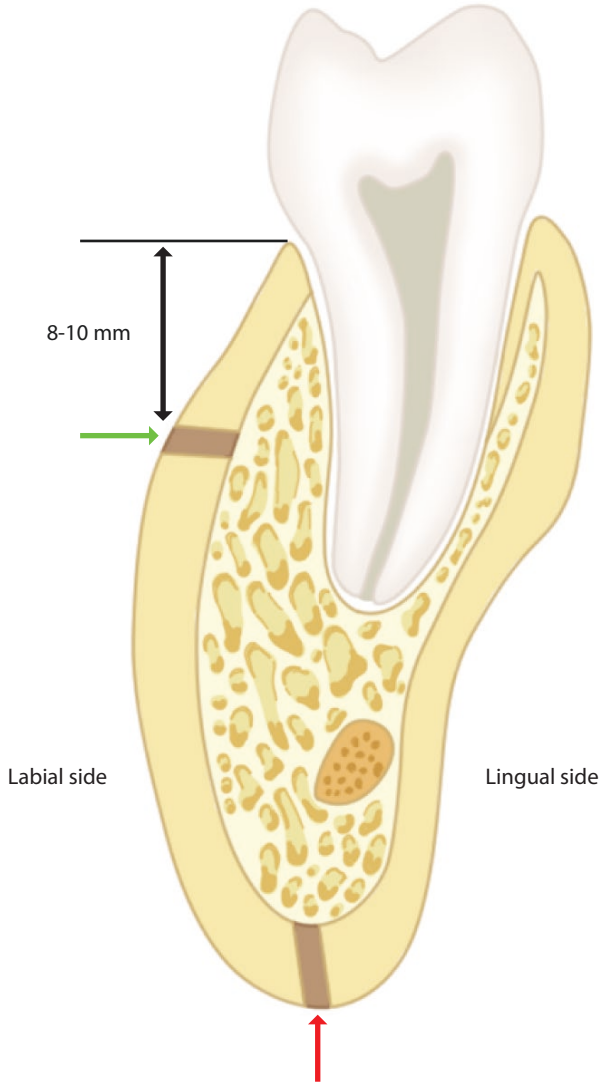

Fig. 26.10 Cross-sectional view through the distal of the first molar. Green arrow shows the horizontal bone cut perpendicular to the buccal cortex and at a level 8 mm below the alveolar bone crest. Red arrow points to the position of the inferior border osteotomy

ment and the area to apply a stabilizing bone plate. For counterclockwise rotation of the mandible, the buccal horizontal osteotomy can be curved to facilitate better approximation of the segments.

5. The posterior aspect of the horizontal cut is connected to the anterior aspect of the ascending ramus cut with a #701 fissure bur (Fig. 26.9).
6. A vertical cut is made through the buccal cortex from the anterior aspect of the horizontal cut, directed perpendicular to the inferior border of the mandible using a #703 fissure bur (Fig. 26.11). The inferior border is only cut halfway through, not completely through as in the traditional osteotomy design.
7. The inferior border osteotomy is performed with specially designed reciprocating inferior border osteotomy (IBO) saw blade (Fig. 26.12) that are manufactured by two companies: Stryker, Inc., Kalamazoo, Michigan and Hall Surgical Division of Zimmer, Largo, Florida. The osteotomy is initiated at the anterior vertical buccal cortical osteotomy and directed posterior to merge on the lingual side at the posterior aspect of the gonial notch (Figs. 26.13 and 26.14). This inferior border osteotomy significantly decreases the torque forces required for separation of the proximal and distal segments, reducing the risk of unfavorable fracture and provides a more predictable path of lingual fracture to occur decreasing involvement of the IAN (Fig. 26.8) 0.12 With the split at the inferior border of the mandible, as the mandible is advanced, the lingual cortex remains in place on the distal segment (Fig. 26.15, red arrow), thus eliminating the notching that commonly occurs with the traditional osteotomy design, particularly for larger advancements.
8. The proximal and distal segments are completely separated using the Smith angled separating instruments and a 3-prong Smith spreader (W. Lorenz Surgical, Jacksonville, Florida). No chisels or malleting are generally required in executing the sagittal split osteotomies, thereby minimizing the chances of unfavorable fracture or inadvertent nerve damage during instrumentation. In the unusual occurrence of the IAN remaining adherent to the proximal segment, it is usually

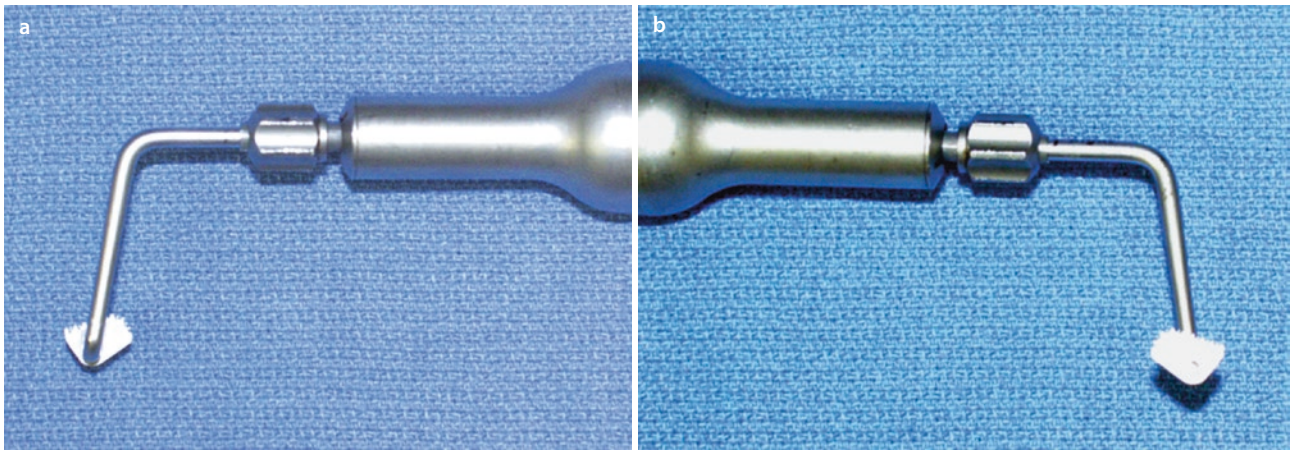

Fig. 26.11 The inferior saw blades are designed for the right side and the other for the left side. The saw is designed so that it will cut halfway across the inferior border of the mandible and has a 5 mm vertical stop to prevent injury to the inferior alveolar nerve

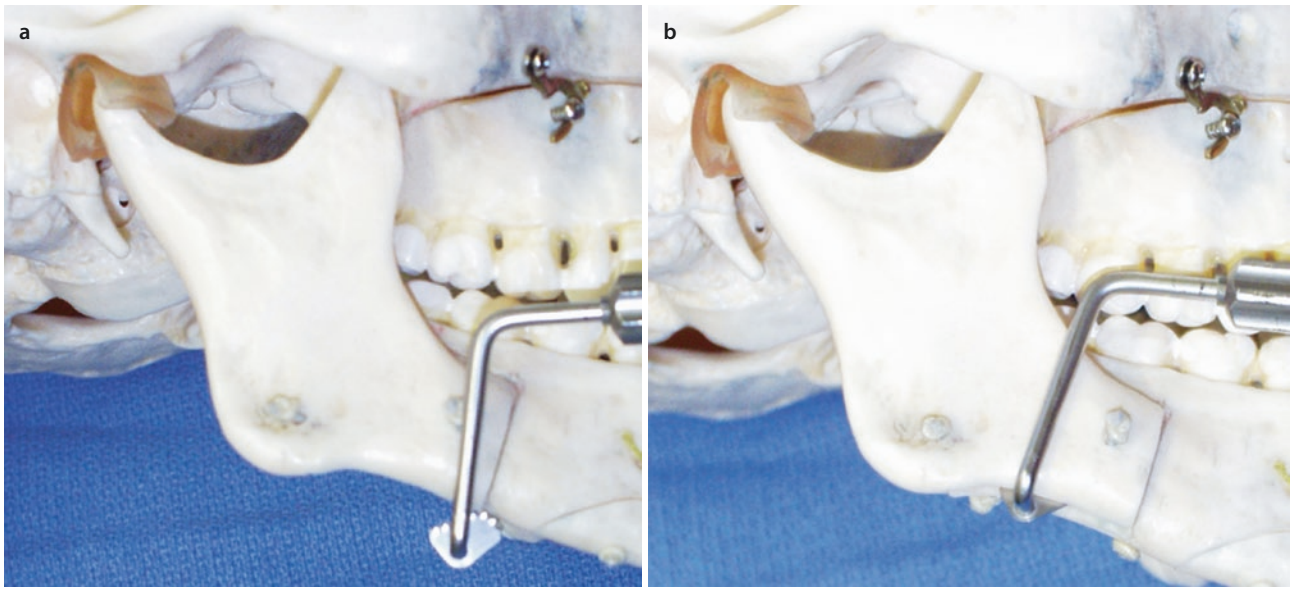

Fig. 26.12 **a** The inferior border osteotomy is initiated anteriorly adjacent to the vertical buccal osteotomy. The vertical shaft of the blade is positioned against the lateral cortical plate of the mandible and this should position the cutting aspect of the blade halfway across the inferior border of the mandible. The blade has a vertical stop and can only penetrate about 5 mm into the inferior border. **b** Once the saw blade is activated and enters into the bone, it is directed toward the lingual plate at the posterior aspect of the gonial notch

related to the cortical bone of the canal incasing the nerve. The IAN is carefully removed from the segment sometimes requiring removal of the residual canal bone.

9. If third molars are present, they are removed, whether impacted or erupted, after the split is complete.
10. The medial side of the proximal segment is smoothed with a reciprocating bone file, removing the remnants of the medullary bone and IAN canal to minimize subsequent injury to the inferior alveolar nerve when the segments are realigned.
11. The mandible is mobilized intermediate splint inserted, and maxillomandibular fixation (MMF) is applied.
12. The anterior aspect of the proximal segment is then positioned beneath the ledge of the distal segment (Fig. 26.15, red circle). This point of fulcrum allows easy setting of the condyle into the fossa with gentle pressure applied vertically at the angle of the mandible (Fig. 26.15, green arrow).
13. Rigid fixation is applied. A 6-hole "Z-plate" can be used (Fig. 26.16a, b), but the surgeon has several options to provide rigid fixation.

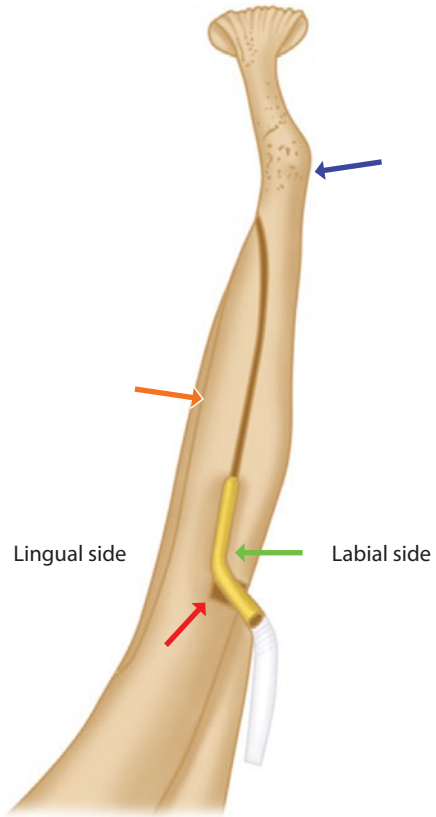

Fig. 26.13 View of the right inferior border of the mandible. Orange arrow points to the inferior border of the mandible. Blue arrow points at the angle of the mandible. Green arrow identifies the inferior border saw initiating the osteotomy anteriorly adjacent to the buccal vertical osteotomy (red arrow). The saw is guided toward the posterior aspect of the gonial notch and angled toward the lingual plate

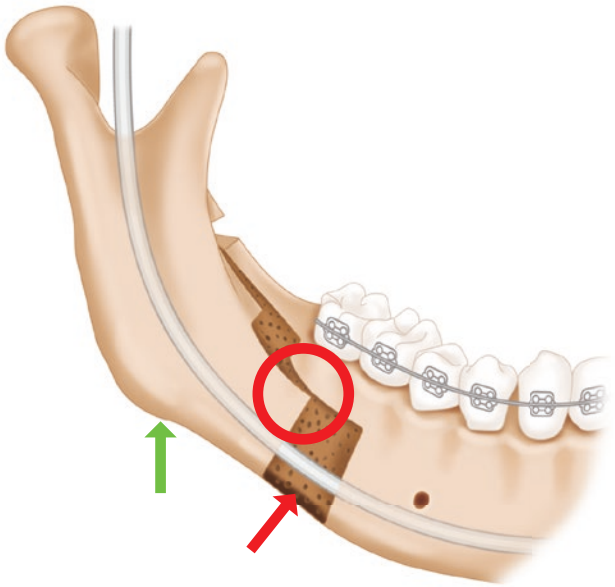

Fig. 26.14 The mandible has been advanced. The red circle shows the bony interface between the proximal and distal segments that controls the vertical position of the proximal segment. The red arrow points to the inferior border cortex that remains attached to the distal segment, eliminating the inferior border notching that occurs with the traditional sagittal split techniques. The green arrow indicates the direction of gentle upward pressure to seat the condyle into the fossa while rigid fixation is being applied

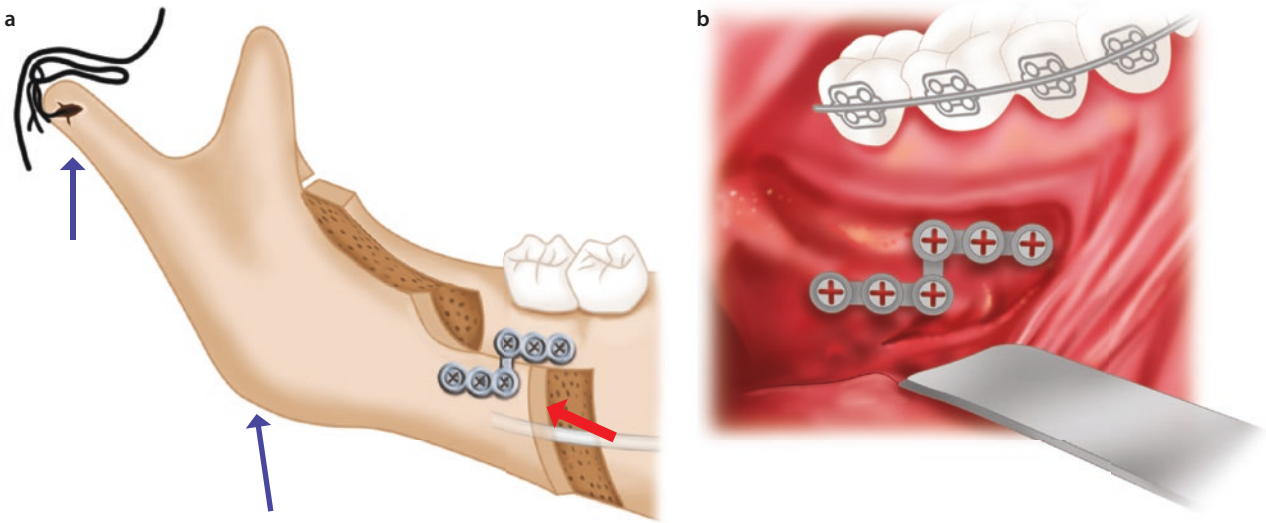

Fig. 26.15 **a** Once the segments are properly aligned, a 6-hole Z-plate can be applied to stabilize the segments as illustrated. If the bone is extremely thin or for large advancements greater than 10–12 mm, then additional supportive screws can be placed bicortical through the ascending ramus area to provide additional stability. **b** Clinical view of the applied 6-hole Z plate. Note the bony interface between the proximal and distal segments

14. Surgical areas are thoroughly irrigated with saline and a final rinse with betadine solution. The incisions are closed, and MMF and splint are removed.

With this modification, the proximal segment is positionally controlled because of the interface between the proximal segment and the ledge of the distal segment. These segments can then be stabilized with a bone plate or bone screws. In prognathic cases, bone will need to be removed from the anterior aspect of the proximal segment and along the anterior superior border of the proximal segment up toward the ascending ramus, for it to fit appropriately beneath the ledge of the distal segment (Fig. 26.17). Whether the mandible is set posteriorly or advanced 5 mm or 20 mm, it can usually be stabilized with a single bone plate positioned at the interface of the proximal segment with the ledge of the distal segment (Fig. 26.16). For larger mandibular advancements, or in the presence of thin cortical bone, 1–2 bone screws can be inserted bicortical along the anterior aspect of the ascending ramus for additional support.

One advantage of the inferior border osteotomy modification is that a significant lower amount of torque force is necessary to complete the sagittal split compared to the greater torque force required for the traditional design. This fact is supported with a recent study by Bockmann, et al. [12], where they performed an in vitro comparison of sagittal split osteotomy on 35 mandibles using the traditional Obwegeser/Dal Pont design with 35 sides without and 35 sides with the inferior border osteotomy. The torque used to split the mandibles was measured, and the fracture line position on the medial aspect of the mandibles was recorded. The average torque for the original technique without the inferior border osteotomy was 1.38 Newton-meters (Nm) or 1.02-foot pound force (ft.lbf) with the lingual fracture line along the mandibular canal, whereas the average torque required to split the mandible incorporating the inferior border cut was 1.02 Nm or 0.75 ft.lbf ($P < 0.001$) with the fracture line more parallel to the posterior ramus of the mandible. Bockmann et al. concluded that adding the inferior border osteotomy to the sagittal split osteotomy resulted in less torque needed to split the mandible and the fracture line was more favorable and predictable.

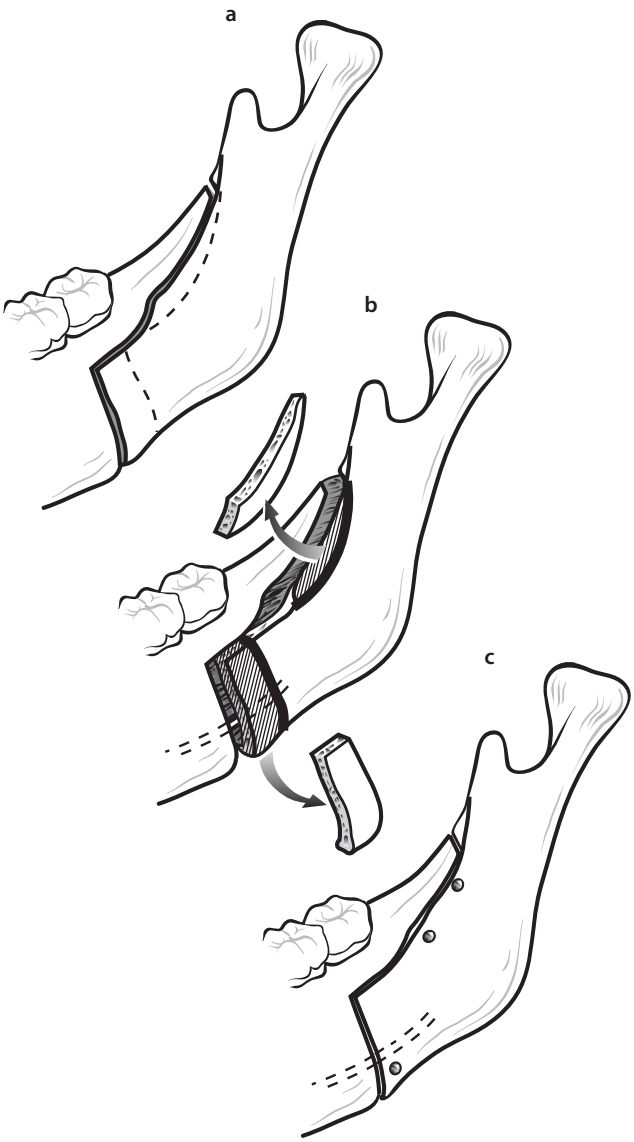

Fig. 26.16 For prognathic cases, this surgical design is very applicable as well, but does require additional ostectomy procedures. **a**–**c** For mandibular setback, the dotted lines outline areas requiring bone removal to eliminate bony interferences. For mandibular setback, usually bone removal is required at the vertical buccal osteotomy area of the proximal segment and also along the ascending ramus. When done appropriately, along with bone removal on the medial side, allows interdigitation of the two segments

26.8.2 Presence or Absence of Third Molars

Our published study [13] evaluated the outcomes of mandibular sagittal split osteotomies in two patient groups relative to the presence or absence of third molars. Group 1 consisted of 250 sagittal split osteotomies with concomitant removal of impacted third molars at the time of surgery, while Group 2 consisted of 250 sagittal split osteotomies with the absence of third molars, using the Wolford modified inferior border sagittal split technique. The surgical procedure and rigid fixation were performed in the same manner in both groups. The occurrence of unfavorable splits was 3.2% in Group 1 and 1.2% in Group 2, but no statistically significant difference between the two groups. In Group 1, unfavorable splits all occurred in teenagers, with seven of the eight fractures occurring at the poste-

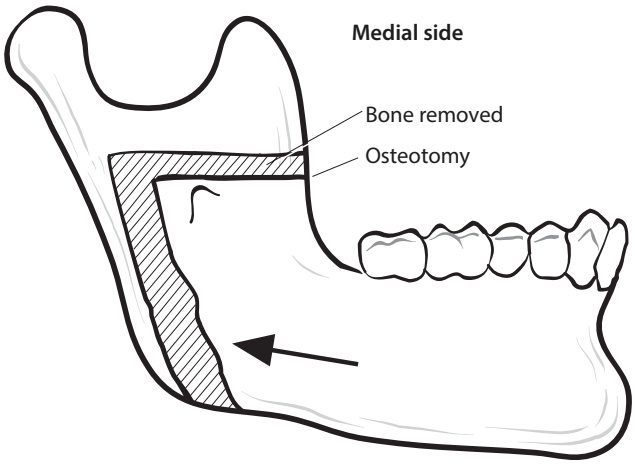

Fig. 26.17 The medial side of the ramus with the cross-hatched lines indicating areas on the proximal segment that will require bone removal of the lingual cortex to allow the segments to sit passively together

rior aspect of the distal segment through the third molar socket. This type of fracture is basically a nonissue with the Wolford method of rigid fixation (Fig. 26.16). Three fractures occurred in Group 2, all involving fracture of the buccal cortex of the proximal segment, but the sagittal splits were completed, the fractured buccal segment restabilized to the posterior part of the proximal segment with the bone plate, and the proximal and distal segments stabilized in the same manner as the patients with favorable splits. Outcomes were the same for all patients relative to stability, whether favorable or unfavorable splits occurred.

26.8.3 Neurosensory Evaluation of Inferior Alveolar Nerve

Our study [14] evaluated neurosensory outcomes on the IAN with sagittal split osteotomies using somatosensory evoked potentials (SEP) computer analysis as well as conventional two-point discrimination. Forty patients were evaluated who underwent bilateral mandibular ramus sagittal split osteotomies using the Wolford modification. All subjects were evaluated postsurgery at 2 weeks, 1 month,6months, and 1 year. At 2 weeks postsurgery, virtually all of the patients had abnormal IAN SEP recordings whereas at 3 months, 80% of the patients had complete return of sensation and at 1 year, 100% of the patients had full return. This study demonstrated that the Wolford modification performed properly and carefully, should have low morbidity for long-term deficit to the IAN.

Another of our studies [15] involved intraoperative SEP evaluations of 10 patients undergoing bilateral sagittal split osteotomies using the Wolford sagittal split modification. The anesthesia technique was standardized for all patients. SEPs were recorded in surgery to identify where potential IAN injury could occur during the operative procedure including: (1) prior to any bone cuts, (2) medial retraction for access for the medial bone cut, (3) cutting and splitting of the mandible, and (4) immediately after rigid fixation was applied. The greatest effect on the SEP was the medial retraction of the inferior alveolar nerve while performing the medial cut on the ramus. There was no significant noted nerve injury through the rest of the procedure.

The advantages to the Wolford inferior border osteotomy modification include the following:

1. Better bony interface between the segments enhancing healing.
2. Simultaneous removal of impacted or erupted third molars, if present, without significant risk of unfavorable split or fracture.
3. Accurate control of condylar position as well as the proximal segment.
4. No postsurgical MMF required providing better oral hygiene, speech, and nutrition.
5. The mandible can be advanced a significantly greater distance than with the traditional designs.
6. Ease of application of rigid fixation intraorally.
7. Temporomandibular joint (TMJ) surgery can be performed concomitantly with the sagittal split osteotomy [16–23].
8. Less risk of damage to the inferior alveolar nerve [14, 15].
9. Facilitates counterclockwise advancement-rotation of the maxillomandibular complex [24–27].

The disadvantages of this technique are as follows:

1. Added expense of purchasing the inferior border saws
2. Learning curve to master the inferior border osteotomy
3. Takes longer surgical time for treating mandibular prognathism compared to the vertical ramus or inverted “L” osteotomies

The mandibular ramus sagittal split osteotomy is a very good procedure for correcting mandibular hypoplasia (retrognathism), mandibular prognathism, and most asymmetries. The advantages of being able to correct the jaw alignment, have an excellent bony interface to promote primary bone healing, easy application of rigid fixation for stability, have accurate control of the condylar position, and the benefits of no postsurgery IMF make the SSRO a preferred osteotomy technique compared to others available. The Wolford

inferior border osteotomy technique also facilitates performing predictable redo mandibular sagittal split osteotomies on patients requiring repeat orthognathic surgery.

26

26.9 Vertical Ramus Osteotomy

This technique is rarely indicated in the OSA patient, but included here for completeness for ramus osteotomies. Extraoral or intraoral approaches can be used for the vertical ramus osteotomy. This procedure involves making a vertical cut from the sigmoid notch to the inferior border of the mandibular ramus, posterior to the lingula. (Fig. 26.18).

The following are indications for vertical ramus osteotomy:

1. Mandibular setback.
2. Small movements (unless temporalis, medial pterygoid, and masseter muscles are detached from the distal segment).
3. Asymmetries of mandible requiring setback.
4. Mandibular advancements may require coronoidectomies as well as bone grafting between the segments.

Stabilize segments with intraosseous wiring or rigid fixation will provide the most predictable results. This procedure is designed to allow the condyle and posterior border of the mandible to remain essentially in their original positions (although there is some rotation and torquing of the condylar head), while the mandibular ramus and body are moved posteriorly.

Contraindications for the vertical oblique osteotomy include the following:

1. Large setbacks (unless temporalis, medial pterygoid, and masseter muscles are detached from the distal segment).
2. Mandibular advancements,
3. Lengthening of the ramus (unless temporalis, medial pterygoid, and masseter muscles are detached from the distal segment).

Advantages of the vertical oblique osteotomy include the following:

1. Technically easy
2. Correction of mandibular prognathism or asymmetries

Disadvantages of this procedure include the following:

1. Unless segments are wired or rigidly stabilized, it may be difficult to control the position of the condyle. Condylar sag may result in anterior open bite postoperatively.
2. Healing time may be increased because of poor bony interface between segments.

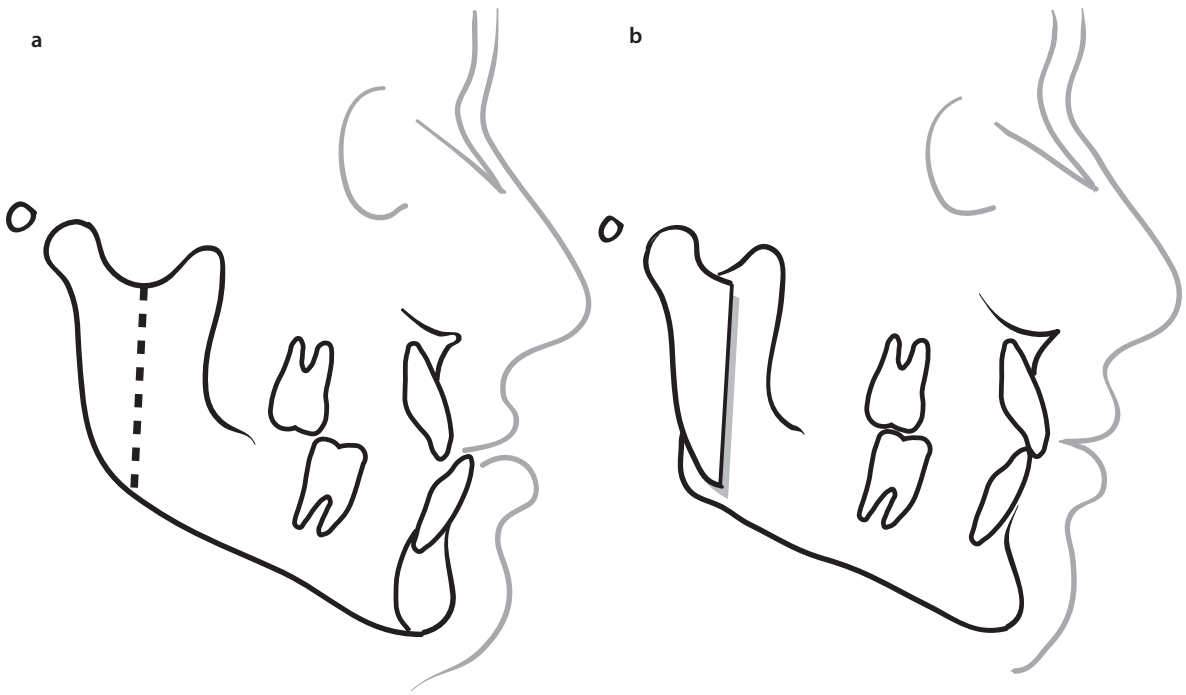

Fig. 26.18 **a** The vertical ramus osteotomy is illustrated, with the osteotomy cut extending from the sigmoid notch through the inferior border of the mandible posterior to the lingula. This osteotomy design may be indicated for prognathic cases but rarely indicated for mandibular advancements. **b** In mandibular setbacks, the proximal and distal segments overlap

3. Rigid skeletal fixation (i.e., bone screws) is difficult to use through an intraoral approach, so the procedure usually requires 4–8 weeks of MMF.
4. Procedure may require relatively long-term interarch elastics to control occlusion following removal of maxillomandibular fixation because of increased healing time and lack of condyle positional control.

26.10 Mandibular Ramus Inverted L-Osteotomy

Extraoral and intraoral approaches to perform the mandibular ramus inverted L-osteotomy are acceptable procedures for mandibular setbacks or advancements (Fig. 26.19). Indications include small or large setbacks, asymmetries, mandibular advancements, ramus lengthening (Fig. 26.20), presence of a thin ramus mediolaterally, and severe decrease in posterior mandibular body height. Contraindications include abnormal posterior location of the mandibular foramina and mandibular advancements without grafting.

Advantages of the procedure include the following:

1. Correct mandibular prognathism or asymmetries.
2. Coronoid process and temporalis muscle remain in original position.
3. Mandible can be set back a great distance.
4. Lengthen ramus or advance the mandible when used with bone or synthetic bone grafting.
5. Rigid skeletal fixation can be used.

Disadvantages include the following:

1. Requires bone or synthetic bone grafting for significant ramus lengthening or mandibular advancement.
2. Healing time may be increased compared with other techniques because of poor approximation of the segments when grafts are not used.

26.10.1 Effects on Growth

Ramus procedures have no significant affect on the rate of mandibular growth, providing growth is normal presurgery, but alteration of the position and orientation of the proximal segment can alter the vector of subsequent mandibular growth [28, 29].

26.10.2 Age at Surgery

Surgery can be performed predictably from the age of 12 years and older as long as normal mandibular growth is present and there is no preexisting TMJ pathology. With the sagittal split osteotomy, it is best to use the pro-

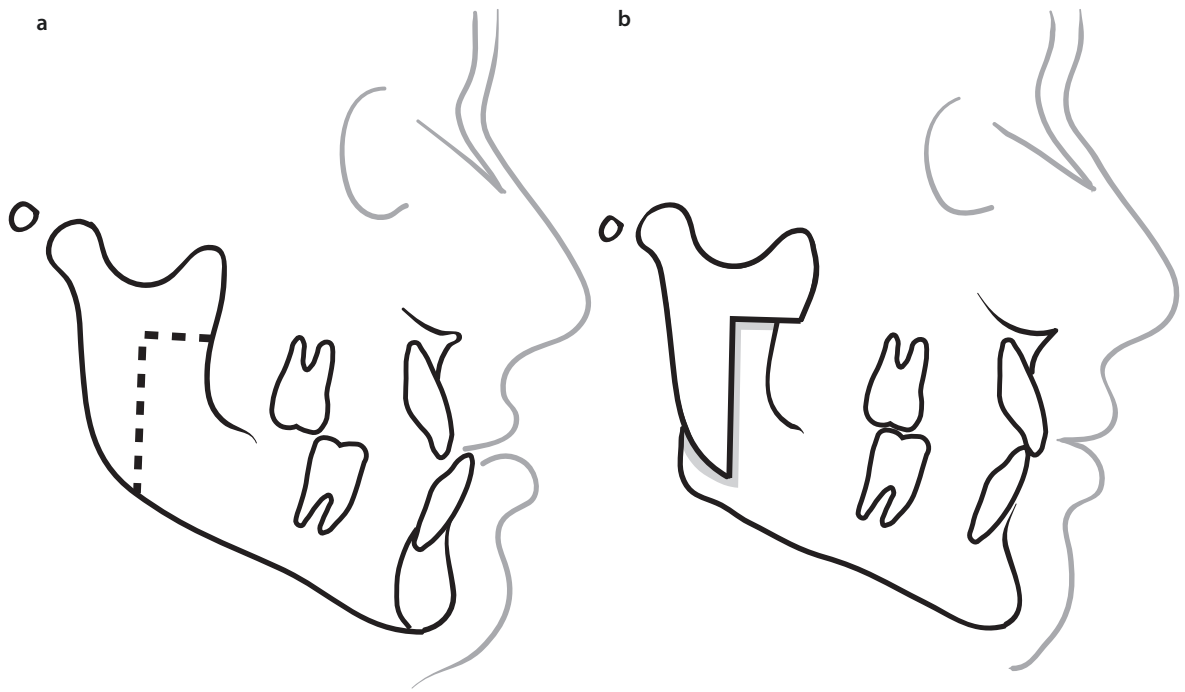

Fig. 26.19 The inverted L-osteotomy is a technique that can be applied to certain OSA patients. A horizontal cut is done superior to the lingula and the vertical cut is done posterior to the lingula. For prognathic cases, the distal segment moves posteriorly with the proximal segment overlapping

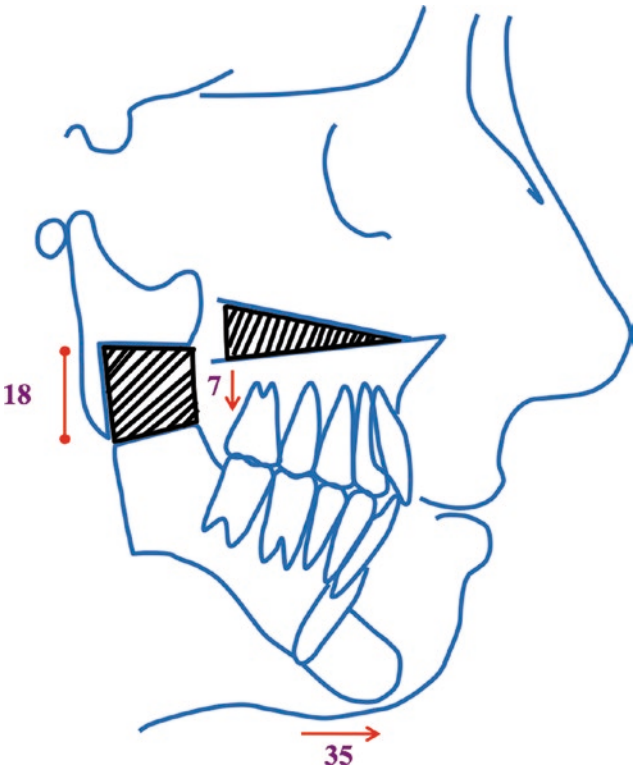

Fig. 26.20 Illustration of an actual case where the ramus was lengthened 18 mm with an inverted L osteotomy requiring a bone graft as well as a maxillary osteotomy to downgraft the posterior aspect also requiring a bone graft. Both areas require rigid fixation for stabilization

cedure after the second molars are erupted so that they are not injured by the procedure before eruption.

26.11 Complications of Mandibular Ramus Surgery

26.11.1 Early Relapse

Early relapse usually is related to improper condylar positioning or slippage between segments during the healing phase. Relapse is usually significantly less with rigid fixation compared with nonrigid methods.

26.11.2 Condylar Sag

Condylar sag is a complication of mandibular ramus surgery, usually caused by improper intraoperative positioning of the condyle by the surgeon, inadequate stabilization of the proximal segment, joint edema, or hemarthrosis such that the condyle is not seated fully in the fossa. If sag is not corrected, the mandible will shift posteriorly following the release of MMF, creating a Class II open bite. The preferred method of correction for condylar sag is to immediately reposition the mandibular segments and apply fixation to secure the position of the proximal segment and condyle. Condylar sag can be avoided by careful surgery, proper seating of the condyles at surgery, and stabilization of the segments with screws, plates, and/or wires.

26.11.3 Temporomandibular Joint Hemarthrosis or Edema

Other possible complications of mandibular ramus surgery are TMJ hemarthrosis and edema. This may displace the condyle downward and forward. Hemarthrosis and joint edema are caused by traumatic surgery and fluid effusion into the bilaminar tissues or joint spaces. This can occur with wire or rigid stabilization. For edema, several minutes of firm upward pressure on the proximal segment helps to express the majority of the fluid from the bilaminar tissue, allowing the condyle to seat more appropriately. The application of rigid fixation and careful surgical techniques prevent these complications.

26.11.4 Unfavorable Splits or Fractures

Unfavorable splits or fractures most commonly occur at the buccal cortex of the proximal segment or vertically through the third molar area of the distal segment. Management requires careful completion of the split and stabilization of the segments with bone plates and screws. When these unfavorable splits occur, proper stabilization of the segments will provide equally stable results as compared to favorable splits.

26.11.5 Extrusion of Teeth

Extrusion of teeth also may complicate ramus surgery. This is fairly common when interosseous wiring and maxillomandibular fixation are used for stabilization of the mandibular segments. Extrusion also may occur with postsurgical elastics, particularly when there is an associated postsurgical malocclusion. Causes of tooth extrusion include improper skeletal stabilization, advancement greater than 5 mm, short tooth roots, mobile teeth, condylar sag, and periodontal disease. If extrusion occurs, there is a potential for orthodontic relapse. Treat extrusion by extensive orthodontics or reoperate later. Prevent extrusion by careful orthodontics and surgery and adequate skeletal stabilization.

26.11.6 Periodontal Defects

Periodontal defects are commonly caused by extrusion or protrusion of teeth, particularly in the lower arch. Poor hygiene also may contribute to the development of postsurgical periodontal defects. Preexisting periodontal problems can worsen with orthodontics and surgery. Improperly performed interdental osteotomies may result in vascular and periodontal compromise. The difficulty of postsurgical dental hygiene in the presence of numb teeth and gums may predispose a patient to periodontal disease. Treatment should include frequent professional dental hygiene visits with special attention to home care regimens. The prevention of periodontal disease should include appropriate presurgical periodontal management, good presurgical orthodontics, careful surgery, adequate skeletal stabilization, and proper oral hygiene techniques.

26.11.7 Temporomandibular Joint Dysfunction

Preexisting TMJ conditions/pathologies are common in the OSA patient population and may contribute to postsurgical complications such as TMJ instability, dysfunction, pain, malocclusion, and surgical relapse. These complications may result from several situations:

- Preexisting TMJ conditions such as internal derangement, adolescent internal condylar resorption, reactive arthritis, connective tissue/autoimmune diseases, and other end-stage TMJ disorders [16–24].
- Intraoperative or postsurgical joint trauma.
- Overloading the TMJ related to mandibular advancement, opening the bite posteriorly at surgery with splints and then using posterior vertical elastic mechanics after splint removal to close the bite, and Class III elastics.
- Long-term maxillomandibular fixation, required with interosseous wiring (no rigid fixation), interferes with normal nutritional factors and function of the disc and articular cartilage, resulting in an increased potential for degenerative changes.
- Uncontrolled postsurgical muscle dysfunction such as, trismus, bruxism, and clenching.
- Preexisting medical conditions such as malnutrition, malabsorption, diabetes, smoking, and immunodeficiencies that can interfere with subsequent healing.

Prevention of postsurgical TMJ complications is based on proper presurgical evaluation, diagnosis, and management of patients before and after orthognathic surgery [1]. Pretreatment and presurgical evaluation of the TMJs should include appropriate clinical and imaging examination. Identify and manage preexisting TMJ disease appropriately. Do not overload the joints by using excessive forces such as Class III elastics. Patients with a history of nocturnal clenching and bruxism may require medications postsurgically to decrease the overloading effects of these habitual patterns. Mandibular advancements increase the resting pressures within the joints until the soft tissues have a chance to reequilibrate with the mandibular alignment. Especially important is to maintain a closed bite posteriorly with surgery unless the surgeon is expecting a significant vertical relapse in the area. Surgically creating a posterior open bite may require vertical elastics to close the open bite, which can overload the joint. Careful surgery minimizing the loading forces on the joint and appropriate management of the TMJ preoperatively, intraoperatively, and postoperatively minimizes TMJ complications.

26.11.8 Nerve Injury

Several nerve complications also are encountered commonly with mandibular ramus surgery. The inferior alveolar nerve or its branches may be injured during ramus, body, subapical, and chin procedures [14–16].

Neuropraxia (type I) nerve injury is not uncommon with these procedures and is usually temporary. The cause may be edema, manipulation, stretching, or mild pinching of the neurovascular bundle. If this problem occurs, recovery may take from 2 weeks to several months.

Axonotomesis (type II) injury is caused by a crushing or significant stretching of the nerve. This can cause degenerative changes within the distal portion of the nerve and may take from 3 months to 2 years to recover, depending on the severity and location of the injury.

Neurotmesis (type III) nerve injury is a result of severance or resection of the nerve. Recovery is unpredictable. The best chance for successful recovery is an immediate direct anastomosis. Delays in surgical management may result in atrophy of the distal portion of the nerve that will decrease the quality of recovery significantly.

If the inferior alveolar nerve is severed in a prognathic correction, generally the nerve can be repaired directly without any significant tension on the nerve. However, if the nerve is cut during a mandibular advancement, the appropriate method for management when performing a primary or secondary repair may require decortication of the lateral aspect of the mandible overlying the neurovascular bundle up to and including the mental nerve area. The anterior portion of the inferior alveolar nerve can be cut to allow posterior repositioning of the distal portion of the inferior alveolar nerve and mental nerve. The repair must be com-

pleted with minimal tension. Primary repairs yield the best results. Secondary repair yields poorer results, especially with long delays (more than 6 months). The result of the repair depends on the type and extent of nerve injury, the length of time since the injury, the quality and type of repair, the amount of tension on the repaired nerve, and the vascularity of the area where the repair is being performed. If a nerve graft is required, the size, length, and fascicular pattern affect the results. With a nerve injury requiring a delayed surgical repair, the complete return of normal sensation is unlikely [14–16].

26.11.9 Infections

Infections usually occur because of breakdown of an incision with contamination or avascular necrosis. Indicated treatment includes culture and sensitivity, appropriate antibiotics, conservative debridement, and copious and frequent irrigation with saline. The most common surgical area to become infected is the mandibular sagittal split incision area. If properly managed, there is little to no consequence. *Candida albicans* infections are also common intraorally in some patients. Systemic antibiotics may increase the incidence of *Candida* infection.

26.11.10 Nonunion

Nonunions usually are caused by poor segment alignment, inadequate bony contact or mobility, and inadequate stabilization. Nonunions are best treated early by providing stability and adequate bony contact between segments. A long-term nonunion may require reoperation with possible bone or synthetic bone grafting. Nonunions can be prevented by careful surgery, appropriate immobilization of segments using rigid fixation, and adequate bone contact between segments.

26.11.11 Bleeding Problems

In ramus osteotomies, the most common major vessels involved in bleeding problems include the inferior alveolar, facial, retromandibular, masseteric, and maxillary vessels. In mandibular body osteotomies, bleeding involvement may include the inferior alveolar, lingual, and facial vessels. Hemorrhage control initially is performed with pressure packing, identification of the causative vessels, and hemostasis by cauterization, Avitene (microfibrillar collagen hemostat), other hemostatic agents, or ligation. Secondary bleeding is rare but can be controlled by local tamponade, reexploration of the wound, or embolization.

In summary, the Oral and Maxillofacial Surgeon involved in treating OSA patients should understand the application of the various mandibular osteotomy procedures, posses the skills to execute the procedures when indicated, and know the potential complications and how to manage the complication if it occurs. For OSA patients, mandibular osteotomies to advance the mandible are often combined with maxillary osteotomies for counterclockwise rotation of the maxillomandibular complex as well as other adjunctive procedures, such as partial turbinectomies, septoplasty, uvulopalatopharyngoplasty, and genioplasty, to eliminate airway obstruction, establish a good functional occlusion, eliminate pain, and enhance facial balance. The goal is to render the optimal outcome for the OSA patient.

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Counterclockwise Rotation of the Maxillomandibular Complex for the Correction of Dentofacial Deformities and Sleep Apnea

Larry Wolford

Contents

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27.1 Occlusal Plane Alteration

The correction of dentofacial deformities often requires double jaw orthognathic surgery to achieve a quality functional and aesthetic result. An often ignored but important cephalometric and clinical inter-relationship in the diagnosis and treatment is the occlusal plane angle (OPA). The OPA is defined as the angle formed by the Frankfort horizontal plane (a line through infra-orbitale tangent to the superior aspect of porion) and a line tangent to the cusp tips of the lower premolars and the buccal groove of the second molar (Fig. 27.1). The normal value for adults is 8 ± 4 degrees. An increased or high occlusal plane angle (HOP) is reflected in an increased mandibular plane angle (dolichocephaly), and a decreased or low occlusal plane angle (LOP) correlates with a decreased mandibular plane angle (brachycephaly). The focus of this chapter will be on the HOP facial morphology since it is commonly associated with sleep apnea and will describe the diagnostic characteristics, treatment protocols, and outcome results.

The traditional methods used by most clinicians for surgical management of the OPA in double jaw surgery are usually addressed by one of the following three methods: (1) maintaining the presurgical OPA, (2) establishing the OPA by autorotation of the mandible (usually in an upward and forward direction) when correcting vertical maxillary hyperplasia, or (3) selectively increasing the OPA relative to Frankfort horizontal plane (FHP) to "improve stability" (regardless of the steepness of the original OPA) by the posterior intrusion of the maxilla and vertically shortening the posterior height of the mandible. Although these methods may achieve an acceptable relationship of the teeth in centric relation, they may not provide the optimal functional and aesthetic relationship of the musculoskeletal structures, dentition, and airway dimensions. As the OPA increases in steepness and begins to approach the slope of the TMJ articular eminence, certain functional problems can develop, including (1) loss of canine protected occlusion, (2) loss of incisal guidance, and (3) development of working and nonworking posterior dental functional interferences. If the clinician believes in the protected occlusion philosophy, there may be concern over the application of the traditional treatment methods of increasing the angulation of the OPA in patients that initially present with an HOP facial morphology.

In addition, the steepness of the OPA can have a profound adverse effect on the dimensions and volume of the oropharyngeal airway. The steeper the OPA, there is generally a reduction in the dimensions and volume of the oropharyngeal airway that can contribute to upper airway resistance syndrome and sleep apnea.

27.1.1 History

The philosophy and implementation of deliberate alteration of the OPA by counterclockwise rotation (CCWR) or clockwise rotation (CWR) of the maxillomandibular

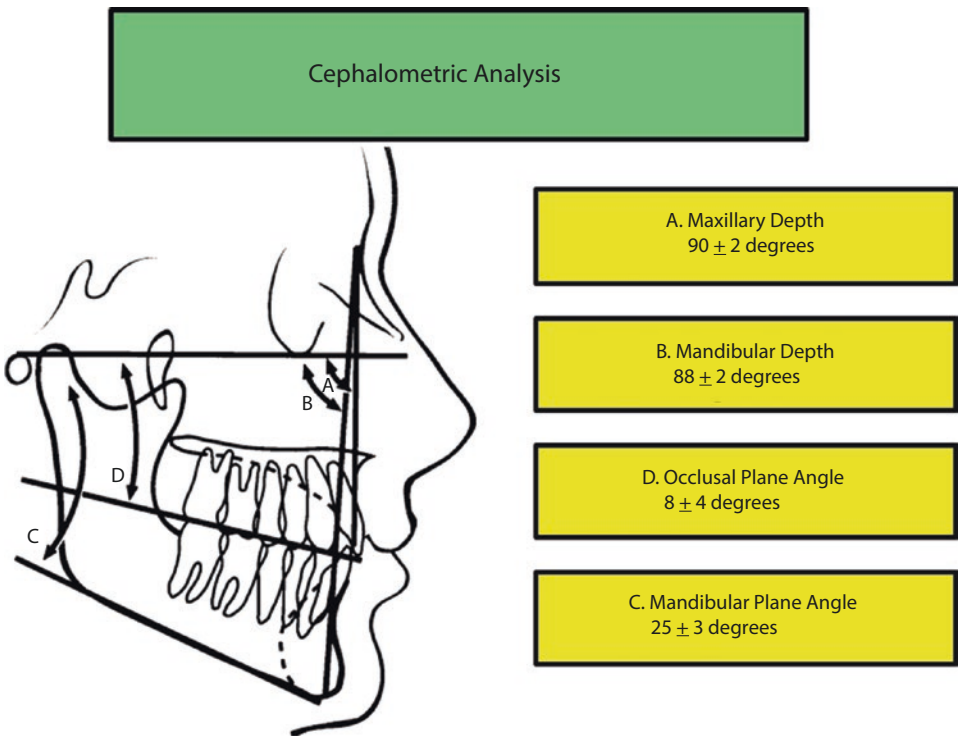

Fig. 27.1 Cephalometric analysis is an important aspect for patient diagnosis and treatment planning. An often overlooked but essential inter-relationship is the occlusal plane angle (OPA) relative to the Frankfort horizontal reference plane. The OPA can have a profound effect on jaw and occlusal function, facial esthetics, and airway. When orthognathic surgery is considered for a patient, alteration of the occlusal plan may be necessary to achieve the best treatment outcome

complex (MMC) was developed by Wolford in 1981 with the first known, successfully performed double jaw CCWR of the MMC by vertical lengthening of the posterior maxilla and mandible. This procedure was done prior to the availability of rigid fixation. The first published reference to the philosophy and surgical planning of CCWR of the MMC was by Wolford et al. [1] in 1985. The second reference to CCWR of the OPA was by Wolford and Hilliard [2] in 1987 where the first known case to have CCWR of the MMC was published (surgery performed in 1981). The patient's surgery was carried out specifically to correct her severe sleep apnea and severe facial deformity. Thus, the concept and application of CCWR of the MMC as an effective means to maximize functional, esthetic, and airway outcomes in patients with HOP facial deformities was introduced.

Wolford et al. [3, 4]. published detailed descriptions of the clinical and radiographic characteristics of the HOP facial type that could benefit from CCWR of the MMC, as well as presented the protocols for surgical management. Kortebein and Wolford [5] in 1991 demonstrated the significant and positive effect that CCWR of the MMC had on increasing the oropharyngeal airway as well as the improvement of facial balance in treating sleep apnea patients. Chemello et al. [6] in 1994 published a comparative stability study between CCWR and CWR of the MMC, demonstrating good stability for both in the presence of healthy TMJs.

Nevertheless, postsurgical stability has remained a major concern for CCWR of the MMC by many clinicians because of numerous reports of mandibular relapse related to condylar resorption with simple surgical mandibular advancements, plus the misperceived problems related to increasing the posterior facial height; stretching of the suprahyoid, pterygoid, and masseteric muscles; and adverse effects on the TMJs [7–21]. It has been reported that the skeletal stability after CCWR of the MMC is comparable to other mandibular surgical procedures [6, 22, 23], but to obtain the acceptable level of stability after CCWR, appropriate preoperative orthodontic treatment, proper execution of the surgical procedures, and the presence of healthy and stable TMJs are essential factors. But Proffit et al [24] found that surgically decreasing the anterior facial height by CCWR of the mandible jeopardizes the stability of the results. However, with the development of rigid fixation, improvement in surgical techniques, as well as recognition of preexisting TMJ pathology and its appropriate management, CCWR of the MMC has proved to be a very stable procedure [6, 22, 23].

27.2 Corrected Frankfort Horizontal Plane

There are often times when the cephalometric analysis does not correlate to the clinical assessment of the patient's facial morphology. This can be related to an aberrantly positioned FHP because of vertical malposition of porion or orbitale and/or anteroposterior malposition of nasion compared to the "normal" anatomy. In this situation, it can be helpful to reorient the FHP (i.e., correct the FHP reference line) so that the cephalometric values for maxillary and mandibular anteroposterior (A-P) positions correlate with the clinical impression of the patient. This provides application of the normal cephalometric values to assist in diagnosis and treatment planning (Fig. 27.2). Cephalometric analysis tempered with good clinical judgment can be valuable tools in establishing the most appropriate orthodontic and surgical treatment plans.

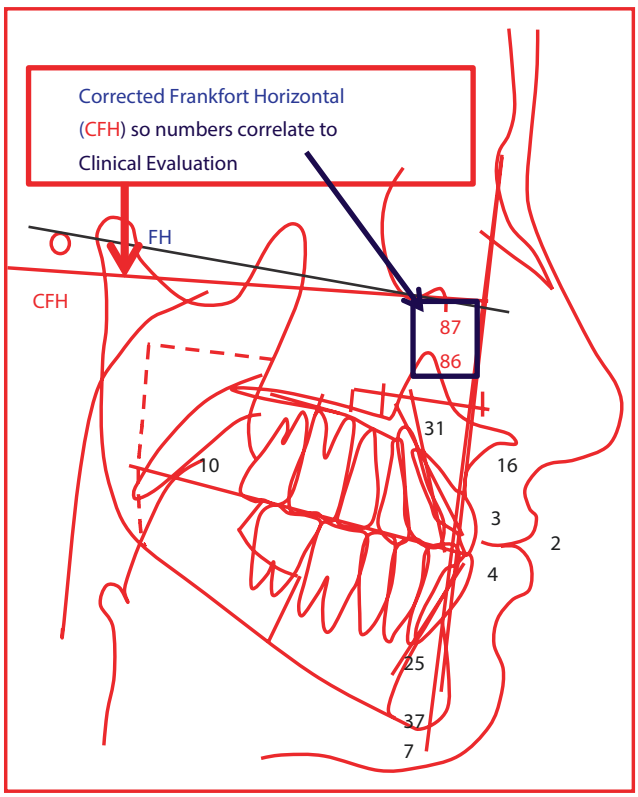

Fig. 27.2 The cephalometric analysis of numerical values in reference to the A-P projection of the maxilla and mandible may not represent the clinical impression of the patient. In this situation, it can be helpful to reorient the FHP (i.e., correct the FHP reference line) so that the cephalometric values for maxillary and mandibular anteroposterior (A-P) positions correlate with the clinical impression of the patient. This provides application of the normal cephalometric values to assist in diagnosis and treatment planning

27.3 High Occlusal Plane (HOP) Facial Type

HOP facial morphological types are the most common facial form associated with sleep apnea (Figs. 27.3a, b, 27.4a, b, and 27.5a). Common characteristics of the HOP facial type generally include some or all of the following: (1) increased OPA (12 degrees or greater); (2) increased mandibular plane angle; (3) anterior vertical maxillary hyperplasia, and/or posterior vertical maxillary hypoplasia, as well as transverse hypoplasia; (4) increased vertical height of the anterior mandible and/or decreased vertical height of the posterior mandible; (5) decreased projection of the chin (A-P microgenia); (6) A-P and posterior vertical mandibular and maxillary hypoplasia; (7) decreased angulation of maxillary incisors, although overangulation can occur; (8) increased angulation of mandibular incisors; (9) occurrence of Class II occlusion, which is common, although Class I and Class III occlusions can also occur; (10) presence of anterior open bite that may be accompanied by an accentuated curve of Spee in the upper arch; (11) hypertrophied turbinates, septal deviation, and nasal airway obstruction; (12) loss of incisal guidance, loss of canine protected occlusion, and the presence of working and nonworking posterior dental interferences in more pronounced cases in which the OPA approaches the slope of the articular eminence; and (12) decreased oropharyngeal airway where the more severe cases may demonstrate moderate to severe sleep apnea symptoms as a result of the tongue base and soft palate displaced posteriorly and constricting the oropharyngeal airway. Normal oropharyngeal airway space measured from the posterior pharyngeal wall to the soft palate and to the base of tongue is 11 ± 2 mm.

27.3.1 Orthodontic Considerations for HOP Facial Type

In the HOP facial type, decreasing the angulation of the maxillary incisors below normal and increasing the lower incisor angulation during the presurgical orthodontic phase may be indicated so that when the OPA is decreased surgically, the same amount of angulation change occurs with an increase in the maxillary incisor angulation and a decrease in the mandibular incisor angulation (Fig. 27.6).

If the maxilla is segmentalized at surgery in the HOP facial type, then the presurgical orthodontic goals, relative to the maxillary incisor angulation, are not as critical as they are for a one-piece maxilla. If the maxilla is sectioned bilaterally, between the lateral incisors and canines, the following movements can be accomplished: (1) optimal maxillary incisor angulation in the final surgical position; (2) adjustments for tooth size discrepancies between the maxillary and mandibular anterior teeth; (3) corrections of transverse, vertical, and anteroposterior arch discrepancies; and (4) leveling the curves of Spee and Wilson.

27.3.2 Surgical Decrease of the OPA

In the HOP facial type, the indicated surgical correction should include a CCWR of the MMC. In open bite cases or deep bite cases, the maxillary OPA and the mandibular OPA may be different from each other and should be evaluated independently. For illustrative purposes, a Class I occlusion case is used with the maxillary incisor edge as the center of rotation (Fig. 27.6). The anatomical changes that occur with CCWR of the MMC include the following: (1) OPA decreases; (2) mandibular plane angle decreases; (3) maxillary incisor angulation increases (the same amount of degrees that the maxillary OPA decreases); (4) mandibular incisor angulation decreases (the same amount of degrees that the mandibular OPA decreases); (5) projection of the chin increases relative to the lower incisor edges; (6) posterior facial height may increase; (7) prominence of the mandibular angles may increase; (8) perinasal area moves posteriorly relative to the maxillary incisor edges; (9) incisal guidance and canine protected occlusion improves, and posterior working and nonworking interferences are eliminated; and (10) oropharyngeal airway increases.

The center of rotation affects the aesthetic relationship of the jaws with the other facial structures. If the center of rotation is at the maxillary incisor edge, as in Fig. 27.6, the perinasal area, subnasale area, and the nasal tip move posteriorly and the chin comes forward. If rotation is around point A or higher, then the perinasal area and the nose are less affected, but the maxillary incisor edges come forward, increasing the A-P support to the upper lip, and the chin also comes further forward. When decreasing the OPA for CCWR and advancing the mandible, the oropharyngeal airway increases substantially. There is a significant aesthetic improvement that decreasing the OPA can make with the most notable change in forward projection of the mandible and chin.

27.3.3 Mandibular Surgery First: Sequencing with Healthy TMJs

When the OPA is surgically decreased, it is usually easier to perform the mandibular osteotomies first, creating bilateral posterior open bites as the posterior mandible is moved downward and usually forward to its new posi-

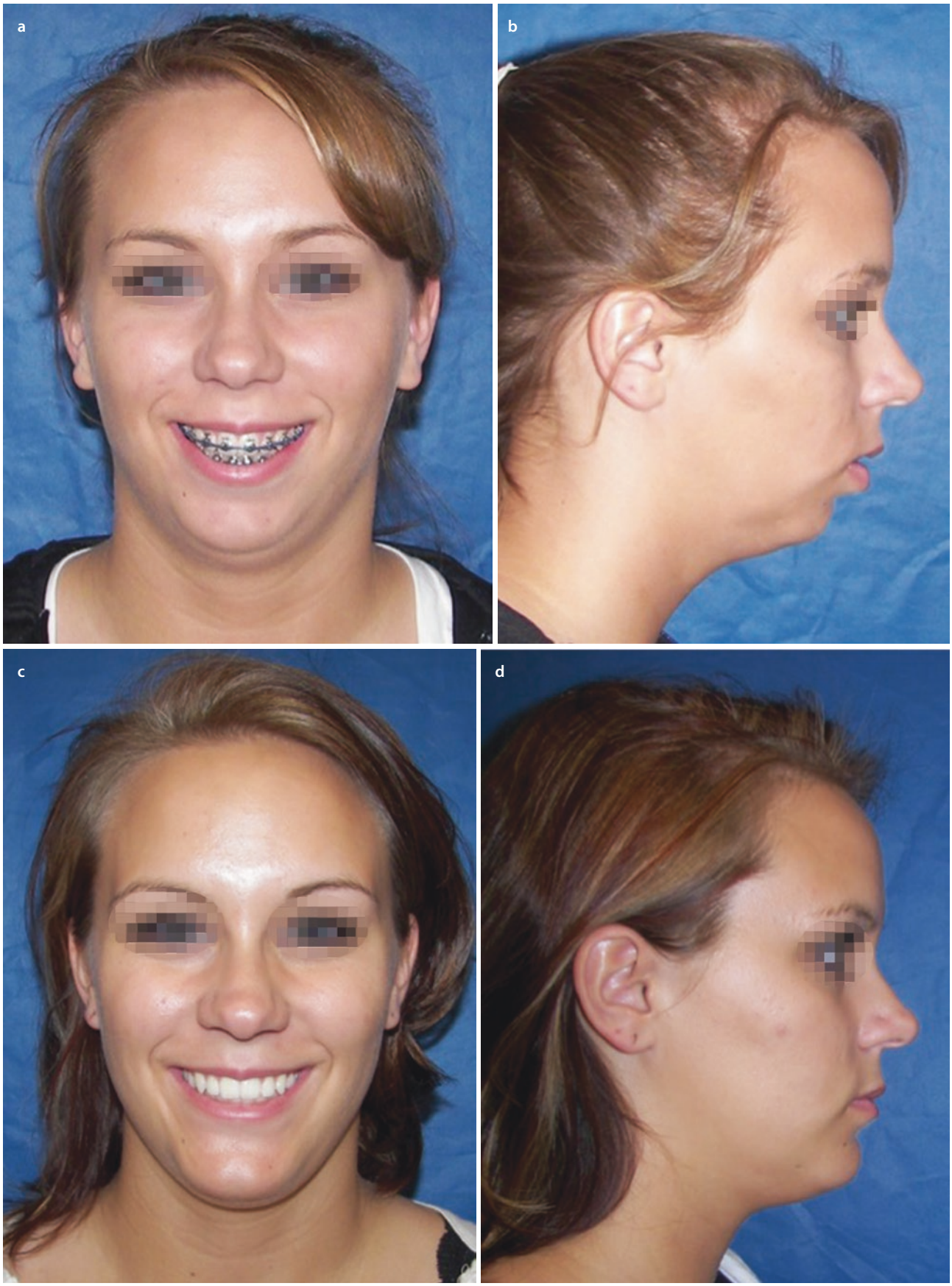

Fig. 27.3 **a** This 18-year-old female with AICR demonstrates good frontal facial symmetry. **b** In profile, the retruded mandible and HOP facial morphology are evident. **c**, **d** The patient is seen three years postsurgery demonstrating good facial balance

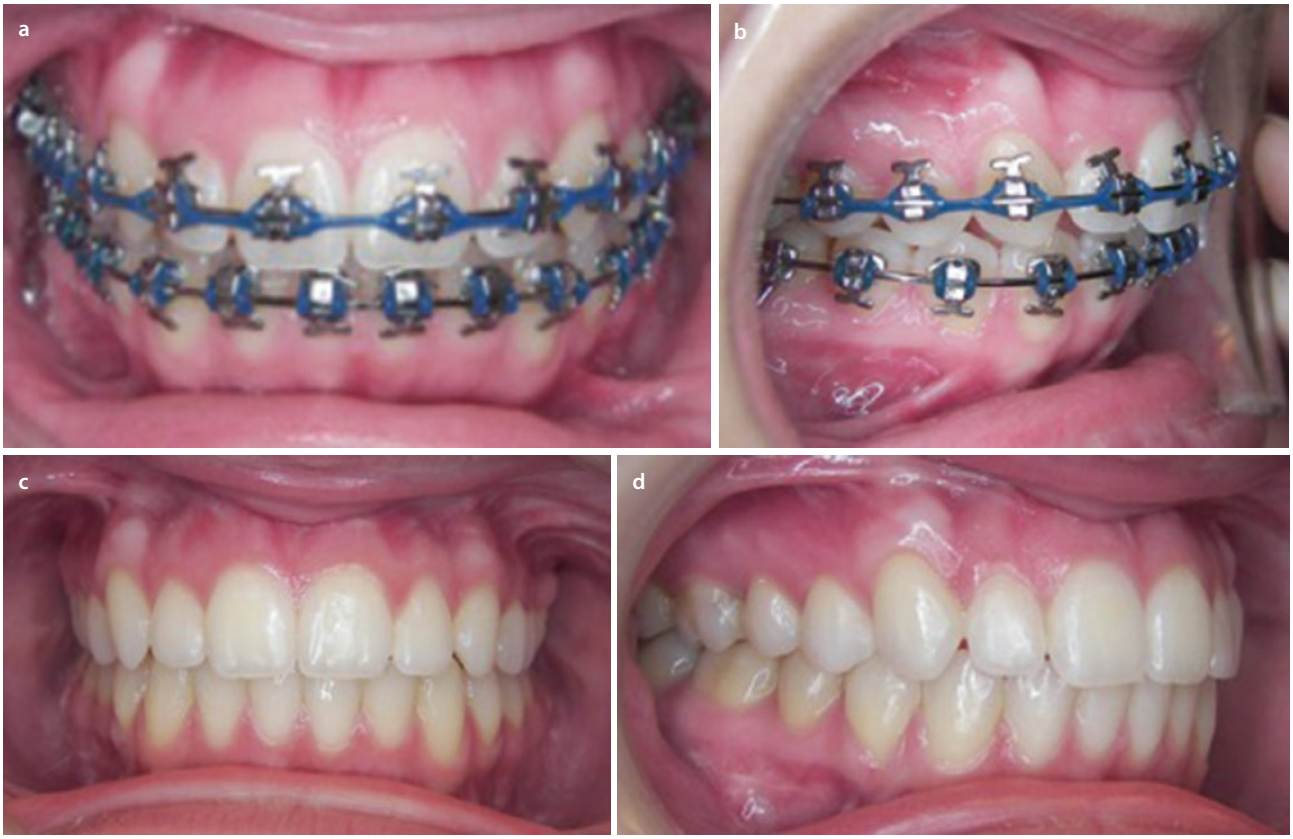

Fig. 27.4 **a**, **b** The Class II end-on occlusal relationship is noted that has been getting progressively worse. **c**, **d** At three years postsurgery, the patient is noted to have a good stable occlusal relationship

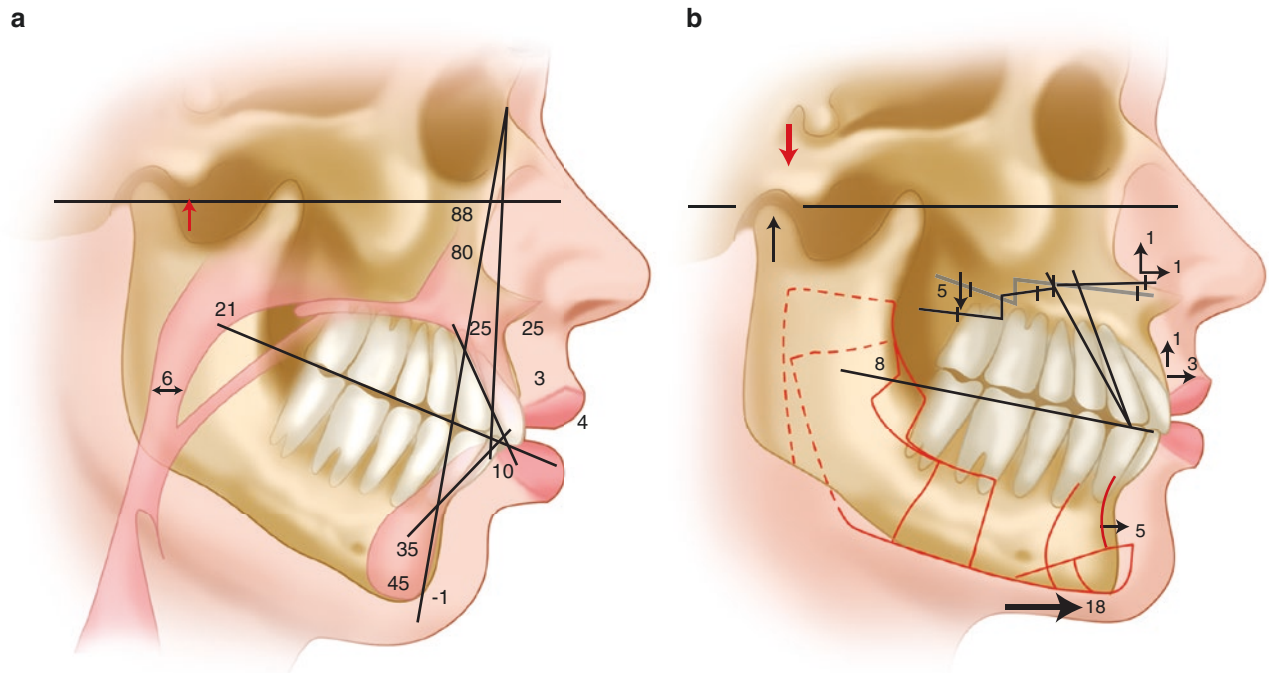

Fig. 27.5 **a** Presurgery cephalometric analysis demonstrates the HOP facial morphology with the retruded mandible. **b**, the surgical prediction tracing illustrates the counter-clockwise rotation of the maxillomandibular complex as well as repositioning the articular discs and augmentation genioplasty

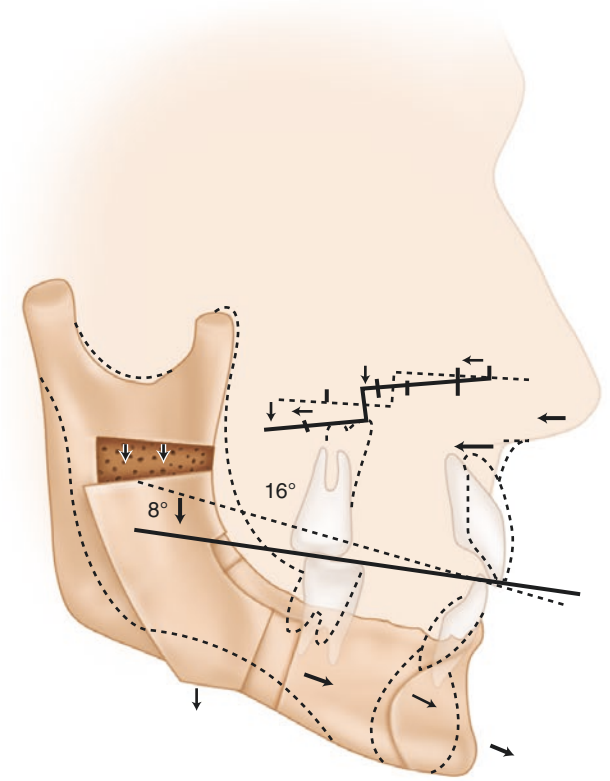

Fig. 27.6 For illustrative purposes, a Class I occlusion case is used with the maxillary incisor edge as the center of rotation. The anatomical changes that occur with CCWR of the MMC include the following: [1] OPA decreases; [2] maxillary incisor angulation increases (the same amount of degrees that the maxillary OPA decreases); [3] mandibular incisor angulation decreases (the same amount of degrees that the mandibular OPA decreases); [4] projection of the chin increases relative to the lower incisor edges; and [5] perinasal area moves posteriorly relative to the maxillary incisor edges

tion with an intermediate surgical splint. Although many surgeons prefer to perform the maxillary osteotomies first, this sequencing makes the surgery much more difficult as then a significant anterior open bite must be developed as the posterior maxilla is repositioned downward to its new position, rotating the mandible downward and backward with the intermediate splint creating a substantial anterior open bite. Then, the mandibular osteotomies are completed, but the subsequent CCWR of the mandible and application of MMF may place excessive stress on the maxilla and could cause some maxillary displacement, even in the presence of rigid fixation, resulting in a suboptimal outcome.

Thus, sequencing the mandible first can be a significant advantage for CCWR procedures and would progress as follows in the presence of healthy TMJs: (1) bilateral mandibular ramus sagittal split osteotomies and removal of third molars if present; (2) application of intermediate splint and MMF; (3) application of mandibular rigid fixation; (4) removal of MMF and intermediate splint; (5) maxillary osteotomies, mobilization, removal of third molars if present, segmentation if indicated, and application of palatal splint; (6) intranasal partial turbinectomies, septoplasty, etc., if indicated; (7) maximization of occlusal fit and placement of MMF; (8) application of maxillary rigid fixation and bone grafting if indicated; (9) removal of MMF; and (10) ancillary procedures if indicated such as genioplasty and rhinoplasty.

When the OPA is decreased, it is much easier to set the mandible first into its final position, with bilateral mandibular ramus sagittal split osteotomies, creating bilateral posterior open bites. An intermediate splint will align the mandible in its new position, and then rigid fixation is applied to the mandible. Usually a six-hole Z-plate with 2-mm-diameter monocortical screws provides adequate stability for mandibular setbacks and for most mandibular advancements (Fig. 27.7). However, for large advancements, one or two bone screws can be placed in the ascending ramus for additional stability. Performing the mandibular surgery first makes the maxillary surgery much easier with better positional accuracy. Stabilization of the maxilla is achieved with four bone plates and grafting with bone or porous block hydroxyapatite to fill any osseous defects. In some cases, the vertical height of the ramus may be increased. However, because most of the cases requiring CCWR are skeletal and occlusal Class II malocclusions, the distal segment moves inferior but anterior to the pterygoid-masseteric sling. In Class III HOP skeletal and occlusal relations, because the ramus portion of the distal segment must move down through the sling, the pterygoid-masseteric sling can be split to allow the posteroinferior aspect of the distal segment to rotate down through the sling. The bone eventually remodels back up to the height of the sling. With these techniques of CCWR of the MMC, the muscles of mastication are not lengthened and remain in their original positions. Rigid fixation eliminates the requirement for postsurgery MMF, and usually light-guiding elastics are all that are necessary to control the occlusion after surgery.

27.3.4 TMJ Evaluation and Treatment Considerations

Evaluation of the status of the TMJs before surgery is very important for outcome stability, particularly when surgery is contemplated to decrease the OPA. Surgical

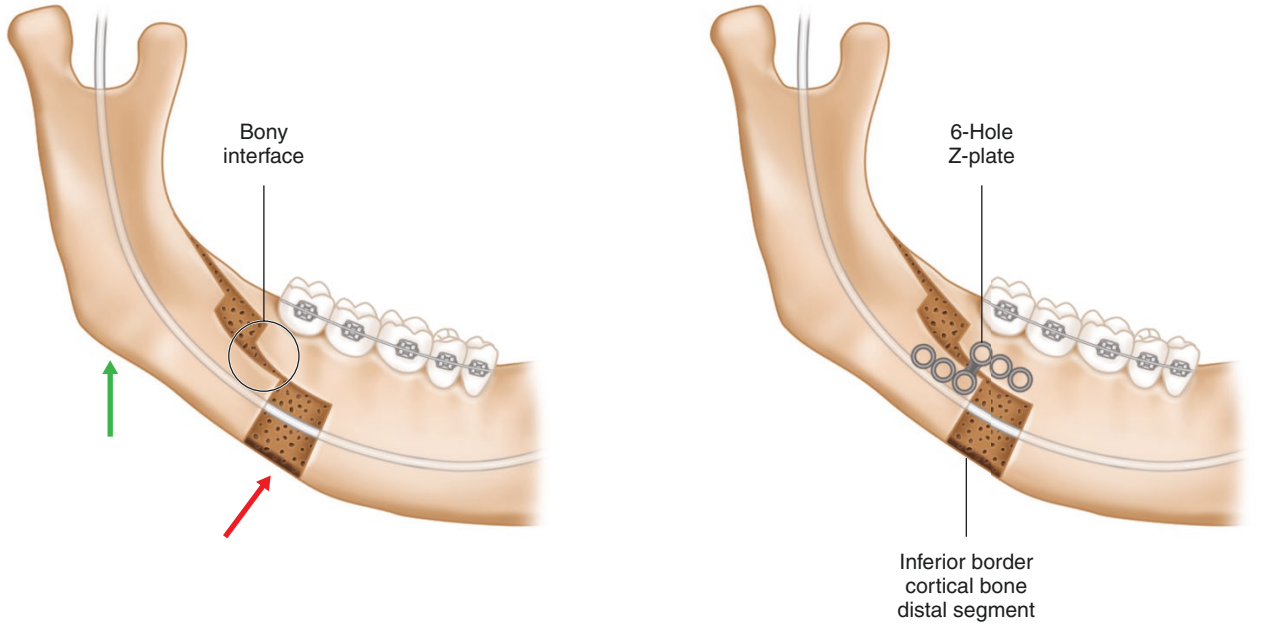

Fig. 27.7 The Wolford modification of the mandibular ramus sagittal split osteotomy maximizes the bony interface between the proximal and distal segments, provides a vertical stop between the segments, and allows the application of a six-hole Z-plate to stabilize the segments

CCWR of the MMC lengthens the functional moment arm (mandible), thereby increasing loading to the TMJs as a result of stretch and tension of the suprahyoid muscles, periosteum, skin, and other soft tissue elements. It may take several months for the soft tissues to adapt and reestablish a state of equilibrium. If the TMJs are healthy and stable, they should be able to withstand the increased loading through the adaptation phase. If TMJ pathology is present, then skeletal and occlusal stability are at risk. Comprehensive assessment and appropriate management of patients with preexisting TMJ disorder is so important so that the joints can be properly treated and will be stable when the surgery is completed.

Al-Moriassi and Wolford [22] published a systematic review and meta-analysis comparing the stability of CCWR to CWR in the correction of dentofacial deformities and showed that these techniques are equally stable and predictable orthognathic surgical procedures when the TMJs are healthy and stable. In the presence of uncorrected TMJ pathology, orthognathic surgery outcomes for CCWR or CWR may be unpredictable relative to stability, function, and pain factors.

Al-Moriassi and Wolford [23] also publish a systematic review and meta-analysis comparing outcome stability of CCWR of the MMC in the presence of healthy TMJs or pathological TMJs. The result of this meta-analysis suggests that the CCWR of the MMC is a stable procedure for patients with healthy TMJs and patients undergoing concomitant TMJ reconstruction with the Mitek anchor technique or patient-fitted total TMJ prostheses. Surgical results may be unstable in the presence of untreated TMJ disc displacement and when TMJ status is not assessed.

27.3.5 TMJ Conditions That Can Affect Surgical Outcomes for CCWR of the MMC

Temporomandibular joint (TMJ) disorders or pathology and dentofacial deformities commonly coexist. The TMJ pathology may be the causative factor of the jaw deformity, or develop as a result of the jaw deformity, or the two entities may develop independent of each other. Common TMJ pathologies that can coexist with or create HOP facial morphologies include (1) articular disc dislocation; (2) adolescent internal condylar resorption (AICR); (3) reactive arthritis; (4) condylar hyperplasia; (5) ankylosis; (6) congenital deformation or absence of

the TMJ; (7) connective tissue and autoimmune diseases; (8) trauma; and (9) other end-stage TMJ pathologies [25–28]. These TMJ conditions can be associated with dentofacial deformities, malocclusion, TMJ pain, headaches, myofascial pain, TMJ and jaw functional impairment, ear symptoms, decreased oropharyngeal airway, nasal airway obstruction, sleep apnea, etc. HOP patients with these conditions may benefit from corrective surgical intervention, including TMJ and orthognathic surgery.

Many clinicians choose to ignore the TMJ pathology and symptoms, preferring to perform only orthognathic surgery for these types of cases, but this treatment philosophy can result in continuation or exacerbation of the presurgery TMJ pathology, pain, recurrence of jaw deformity, malocclusion, and other adverse outcomes. Although most TMJ patients have associated symptoms, approximately 25% of patients with significant TMJ pathology/disorders may be asymptomatic relative to pain, TMJ noises, and jaw dysfunction presurgery. These patients are diagnostically challenging when undergoing orthognathic surgery because the TMJ pathology may not be recognized. Failure to recognize and properly treat the TMJ pathology in symptomatic or asymptomatic patients will commonly result in poor treatment outcomes including potential redevelopment of the skeletal and occlusal deformity by continued condylar resorption or condylar overdevelopment, initiation of or worsening pain, headaches, jaw and TMJ dysfunction, as well as other TMJ symptoms. However, there are clinical and imaging factors that can indicate the presence of TMJ pathology in the asymptomatic as well as the symptomatic patient.

Patients with TMJ pathology and coexisting dentofacial deformities can be corrected with concomitant TMJ and orthognathic surgery (CTOS) in 1 surgical stage or separated into 2 surgical stages. The 2-stage approach requires the patient to undergo 2 separate operations (one surgery to correct the TMJ pathology and a second operation to perform the orthognathic surgery) and two general anesthetics, with a significantly lengthened overall treatment time. Performing CTOS in a single operation significantly decreases treatment time, provides better outcomes, but requires careful treatment planning and surgical proficiency in the 2 surgical areas. Virtual surgical planning (VSP) can be a significant advantage in developing the treatment plan, surgical sequencing, and projected outcome.

Many patients diagnosed with sleep apnea have HOP facial morphologies with accompanying decreased oropharyngeal airways and nasal airway obstruction, but also have TMJ issues that need to be addressed at the same time or before the orthognathic surgery is performed to provide a stable, predictable

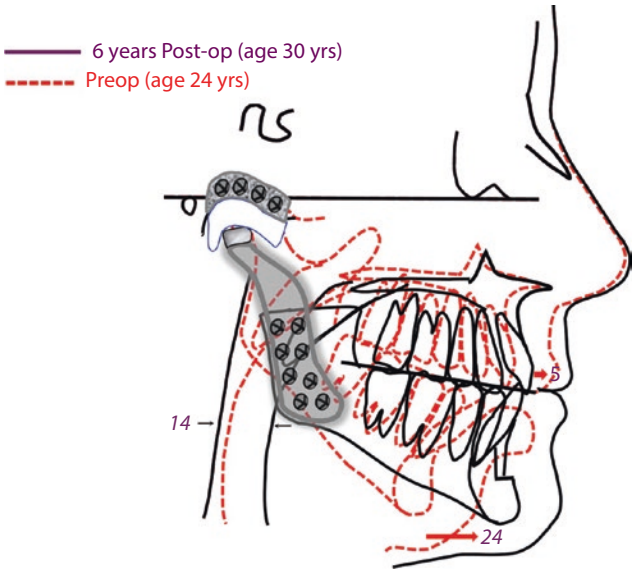

Fig. 27.8 Presurgery (red dotted lines) and 6-year postsurgery (black solid lines) super-imposed tracings of a severe rheumatoid arthritis patient with severe sleep apnea, demonstrates the postsurgical airway changes following CCWR of the MMC including bilateral TMJ reconstruction and CCWR of the mandible using TMJ Concepts total joint prostheses and concomitant maxillary osteotomies and bony genioplasty with pogonion advancing 24 mm. At the oropharyngeal area, the distance between the red line and posterior black line (2 mm) demonstrates the severe presurgical airway constriction. The long-term postsurgical changes are seen between the black lines with the airway dimension of 14 mm. The normal A-P dimension in this area is 11 mm ± 2 mm

outcome and decrease preexisting pain. The normal cephalometric A-P dimension from the posterior pharyngeal wall to the soft palate and posterior pharyngeal wall to the base of the tongue is 11 ±2 mm. In HOP patients who have a retruded maxilla and mandible, this airway may be significantly decreased. Advancing the MMC in a CCWR direction improves facial balance and the oropharyngeal airway opens significantly, to improve breathing (Fig. 27.8). Our studies [5, 29–33] have shown that double jaw surgery with CCWR of the MMC will increase the oropharyngeal airway approximately 65–70% for the first 10 mm of mandibular advancement. With 10–15 mm of advancement, the oropharyngeal airway continues to open, but at a lesser percentage of the amount of mandibular advancement of about 55–60% of the mandibular advancement. When the mandible is advanced 15–20 mm, the oropharyngeal airway continues to open, but only about 40–45% of the amount of mandibular advancement [34].

There are a triad of factors that commonly go together in HOP patients and they include (1) a high OPA facial morphology associated with a retruded maxilla and mandible with an accompanying decreased oropharyngeal airway, (2) nasal airway obstruction related

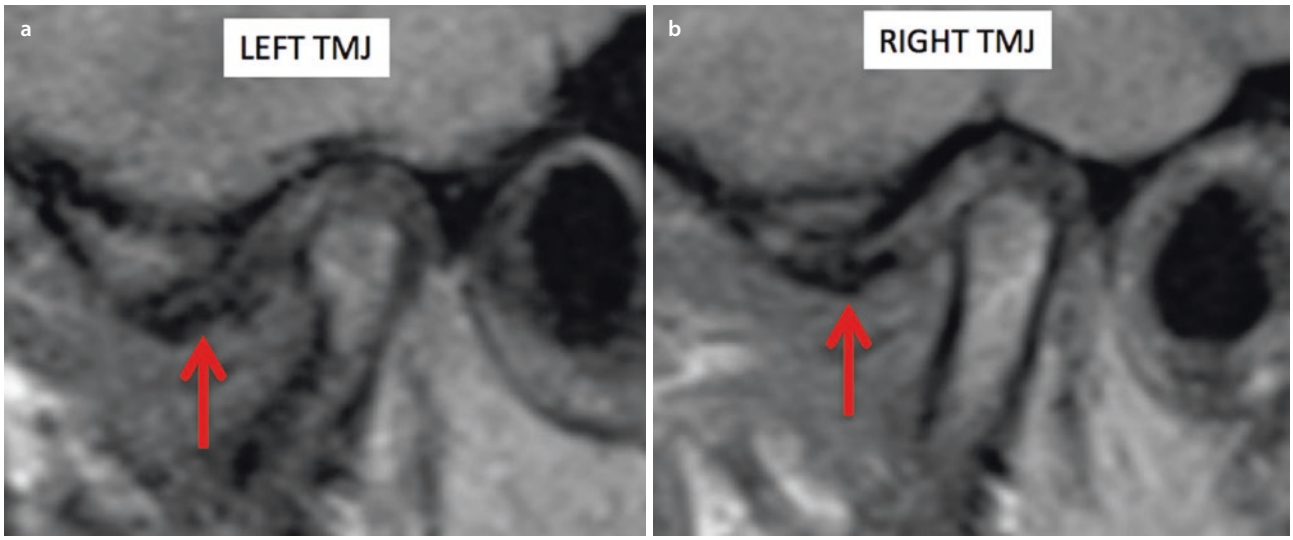

Fig. 27.9 Bilateral MRIs of a patient with AICR. The condyles generally appear small in size and the cortical bone on top of the condyle may be somewhat thin. The articular discs are anteriorly displaced. In AICR patients, the articular discs may or may not reduce on opening

to hypertrophied turbinates and/or nasal septal deviation or spurring, and (3) TMJ pathology. Our study [35] evaluated 1234 consecutive patients referred to the author for orthognathic surgery requiring at least maxillary osteotomies. There were 603 patients (49%) with hypertrophied turbinates that required partial turbinectomies and 278 patients (23%) required nasal septoplasty. For patients requiring partial turbinectomies ($n = 603$), 84% had maxillary hypoplasia, 72% had mandibular hypoplasia, 69% had a high occlusal plane angle, and 49% of the patients required CTOS. A strong correlation was established between hypertrophied inferior turbinates, hypoplastic maxilla and mandible, as well as a steep occlusal plane. Our findings correlate with other studies evaluating the facial morphology of mouth breathing and nasally obstructed patients [36–38]. Therefore, patients with the HOP facial morphology with a retruded maxilla and mandible should be assessed for nasal airway obstruction, decreased oropharyngeal airway and sleep apnea, as well as TMJ pathology (even if asymptomatic).

27.3.6 MRI Evaluation

Magnetic resonance imaging (MRI) is one of the most important diagnostic tools that we have to evaluate, diagnose, and treatment plan for TMJ pathology as it allows evaluation of bone and soft tissue structures, TMJ disc position, morphology, mobility, extent of joint degenerative changes, inflammation, the presence of connective tissue/autoimmune diseases, and so forth (Fig. 27.9). MRI can help in the diagnosis of TMJ disorders in the silent joint in which disc displacement and degenerative changes can be present, may not make noise or cause pain, but may contribute to poor outcomes if only orthognathic surgery is performed.

27.3.7 TMJ Disc Displacement

When discs are anteriorly displaced, there is a 4-year window from onset of the disc displacement to perform the repair for a predictable outcome, providing there is no coexisting reactive arthritis, connective tissue/autoimmune disease, metabolic disease, etc., that could continue a degenerative process within the TMJ with resultant failure of the disc repair. TMJ conditions that respond well to disc repositioning include disc displacement and adolescent internal condylar resorption (AICR) with or without reduction of the disc on opening as long as the disc and condyle are in good condition without significant adhesions, degeneration and the afore-mentioned TMJ diseases, and less than 4 years from the onset of the disc displacement (Fig. 27.9). The Mitek anchor technique is the only proven method to predictably reposition the disc onto the condyle (Fig. 27.10). Disc repositioning with the Mitek anchor can be done concomitantly with orthognathic surgery [22, 23, 25–28].

However, after 4 years, the discs may become non-reducing, deformed with loss of the intermediate zone, thickening of the posterior and anterior bands, degenerative changes within the disc, and vascular invasion. Displaced discs initiate a cascade of events that lead to TMJ arthritis. When discs are displaced and become non-reducing, the degenerative process of the disc progresses more rapidly as compared to displaced discs that reduce. When discs advance to a certain level of defor-

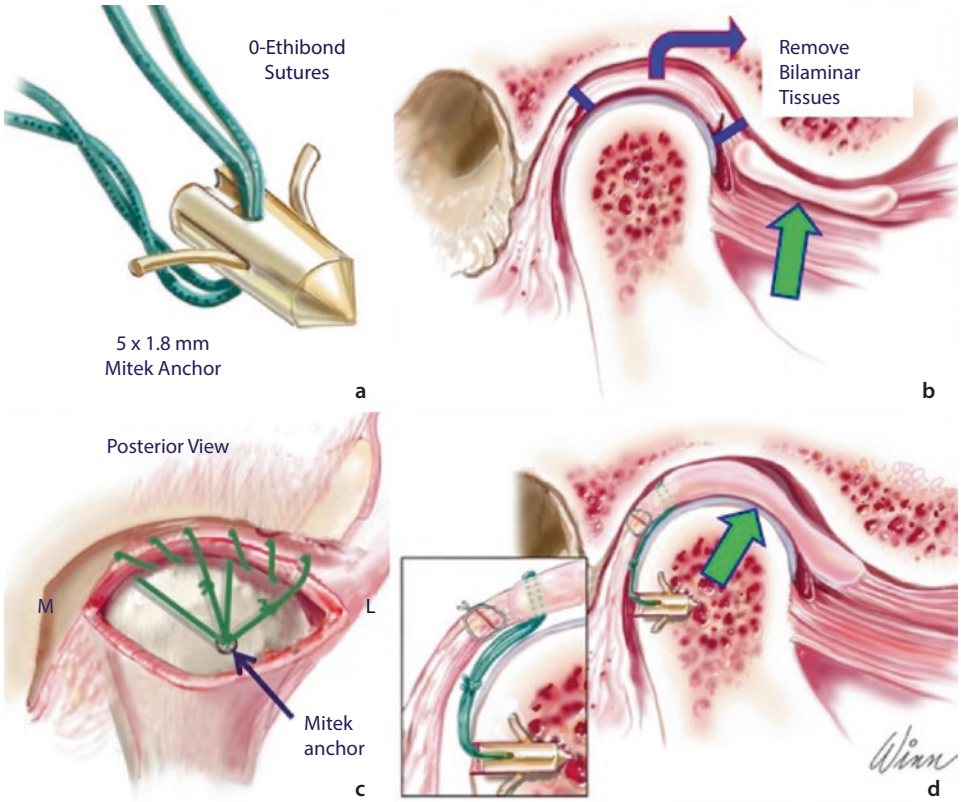

Fig. 27.10 Mitek anchor technique: **a** The Mitek mini anchor is 5 x 1.8 mm in dimension with an eyelet to support 2 artificial ligaments (0-Ethibond suture). **b** Bilaminar tissues are excised and disc mobilized. **c** The disc is passively positioned over the condyle and Mitek anchor placed in the lateral aspect of the posterior head, about 8 mm below the top of the condyle. **d** The sutures are attached to the posterior band of the disc and secured

mation and degeneration, they become non-salvageable requiring TMJ reconstruction with patient-fitted total joint prostheses. Concomitant TMJ reconstruction and orthognathic surgery may be indicated to produce to most predictable and high-quality outcomes.

27.3.8 Adolescent Internal Condylar Resorption (AICR)

Adolescent internal condylar resorption (AICR) is a condition that develops usually during pubertal growth between the ages of 11 and 15 years, predominantly in females (ratio 8:1 females to males) [39, 40]. Clinically, the mandible will be noted to slowly retrude into a Class II occlusal and skeletal relationship with a tendency toward anterior open bite. These patients all have HOP facial morphological profiles. On the MRI, these cases present with a condyle that is slowly becoming smaller in size in all 3 planes of space and the disc is anteriorly displaced similar to (Fig. 27.9. In some cases, there is significant thinning of the cortical bone on top of the condyle contributing to the inward collapse of the condylar head in this pathological process. The articular discs are anteriorly displaced and may or may not reduce on opening. Commonly, the disc becomes non-reducing relatively early in the pathological progression. Non-reducing discs will degenerate and deform at a more rapid rate as compared to discs that reduce.

Our studies [39, 40] demonstrate that AICR is arrested if the articular discs are put back into position on top of the condyle and stabilized with the Mitek anchor technique. It is so predictable for stopping the resorption that the indicated orthognathic surgery can be done at the same operation. Results of this treatment protocol are best for AICR if the TMJ surgery for disc repositioning is performed within 4 years of the onset of the pathology. After 4 years, the discs may become non-salvageable and condyles significantly resorbed with the indicated treatment transitioning to patient-fitted total joint prostheses to repair the TMJs and CCWR the MMC with concomitant orthognathic surgery.

27.3.9 Reactive Arthritis

Reactive arthritis is commonly caused by bacterial or viral entities [41–47] and may show a localized area of inflammation (synovitis) with erosion of the condyle and/or fossa. It also can present as a more profuse inflammatory process through the bilaminar tissues, capsule, surrounding the disc but can progress to the destruction of the disc and condylar resorption. The most common bacteria causing reactive arthritis in the knees and TMJs are the following: Chlamydia trachomatis and Chlamydia pneumoniae, as well as Mycoplasma pneumoniae and Mycoplasma genitalium [46–52]. These are non-culturable, non-motile, obligate

intracellular bacteria that stimulate the production of pro-inflammatory/pain mediators: TNFα, cytokines, chemokines, substance P, etc., with subsequent breakdown of cartilage and bone and generation of pain. They have an anti-apoptosis effect on host cells (including monocytes and macrophages) and create immunodysfunction. Standard antibiotic therapy can be effective for urinary tract, genital, ocular, respiratory, and GI infections involving these bacteria, but are not effective for synovial infections, making it very difficult to eliminate these bacteria from joints including the TMJs [46–52]. Currently, there are no predictable methods to conservatively treat reactive arthritis involving these particular bacteria. However, when the infection is confined to a small portion of the synovial and bilaminar tissues, in the TMJ, debridement may be indicated, but prognosis is guarded. Surgical indication may include removal of the nidus of inflammation and reposition the articular disc if salvageable. When significant TMJ involvement and particularly with extensive destruction of the TMJ tissues, a total joint prosthesis may be indicated.

27.3.10 Connective Tissue/Autoimmune Diseases

The MRI presentation of connective tissue/autoimmune diseases is fairly pathognomonic. These diseases include rheumatoid arthritis, idiopathic condylar resorption, juvenile idiopathic arthritis, psoriatic arthritis, Sjogren's syndrome, lupus, scleroderma, etc. In these conditions, the articular disc oftentimes is in a relatively normal position but surrounded by a reactive pannus. There is usually progressive condylar resorption, "mushrooming" of the remaining condyle, and often resorption of the articular eminence, with slow but progressive destruction of the articular disc. This presentation almost always indicates the requirement of total joint prostheses for jaw reconstruction to eliminate the pathologic process in the joint as well as concomitant orthognathic surgery to correct the associated jaw deformity and malocclusion, eliminate pain, establish a good airway, and provide good facial balance [25–28]. Use of autogenous tissues in this scenario could result in the disease process attacking the autogenous tissues placed into the joint with subsequent failure.

27.3.11 Other End-Stage TMJ Pathologies

Other end-stage TMJ pathologies may include (1) ankylosis, (2) absence of condyle or TMJ secondary to trauma or congenital deformities such as hemifacial microsomia and Treacher Collins syndrome, (3) tumor, (4) metabolic joint diseases, (5) failed autogenous grafts, (6) failed alloplastic TMJ implants, etc. These conditions will generally have the best outcomes with custom-fitted TMJ total joint prostheses.

27.3.12 Repositioning the Mandible First with Concomitant TMJ Surgery (Salvageable Discs)

The TMJ surgery must be performed first since the surgery may include disc repositioning and/or high or low condylectomies (if active condylar hyperplasia present), which will alter the spatial position of the mandible. Repositioning the mandible following the TMJ surgery will place the mandible into its final position, no matter how much mandibular positional change occurred with the TMJ surgery. The only effect will be a difference in the amount of mandibular positional change resultant from the TMJ surgery to the final mandibular position. The maxillary surgery then follows. For patients with high OPAs, this is the best surgical sequencing. When the mandible is repositioned first before the maxilla, the surgical sequencing is as follows: (1) TMJ surgery that may include disc repositioning and high or low condylectomies, or other intra-capsular procedures; (2) mandibular sagittal split osteotomies, removal of third molars if indicated, repositioning the mandible with an intermediate splint, application of intermaxillary fixation (MMF), and application of rigid fixation; (3) maxillary osteotomies and mobilization, removal of third molars if indicated and segmentalization if necessary; (4) intranasal procedures such as turbinectomies and nasoseptoplasty if indicated; (5) application of the palatal splint, MMF, rigid fixation for the maxilla, and appropriate bone grafting, if indicated, with bone or porous block hydroxyapatite. Release MMF and check occlusion; and (6) other ancillary procedures (i.e., genioplasty, rhinoplasty, etc.).

27.3.13 Repositioning the Maxilla First with Concomitant TMJ Surgery (Salvageable Disc)

Some surgeons prefer to reposition the maxilla first in these cases, so the sequencing is different. With sequencing the maxillary surgery before the mandibular surgery, the maxilla must be repositioned before the TMJ surgery since the TMJ surgery will change the position of the mandible, which would then create a malposition of the maxilla if the TMJ surgery was done first. If it is decided to do the maxillary surgery first, then the sequencing changes as follows: (1) completion of maxillary osteotomies and mobilization, removal of third molars if indicated and segmentalization if necessary;

(2) intranasal procedures such as turbinectomies and nasoseptoplasty if indicated; (3) application of the palatal splint and the intermediate splint, MMF, rigid fixation to the maxilla, and appropriate bone grafting, if indicated, with bone or porous block hydroxyapatite; (4) release of MMF and checking of occlusion with intermediate splint, removal of intermediate splint; (5) TMJ surgery that may include disc repositioning and high or low condylectomies, or other intra-capsular procedures; (6) mandibular sagittal split osteotomies, removal of third molars if indicated, reposition into the best occlusal fit, placement of MMF, and application of rigid fixation, release of MMF and check occlusion; and (7) other ancillary procedures (i.e., genioplasty, rhinoplasty, etc.).

27.3.14 Repositioning the Mandible First with Concomitant TMJ Total Joint Replacement

When the condyles and discs are non-salvageable with end-stage TMJ pathology, then TMJ replacement surgery is usually indicated. The most predictable treatment method is with a TMJ total joint prosthesis system. There are 2 basic types of TMJ total joint prostheses: stock and patient-fitted devices. Stock devices have different choices of prefabricated components including fossa components and mandibular components. The surgeon picks the size and shape of fossa and mandibular components that best fit the patient's anatomy. The patient-fitted devices are custom designed to fit the patient's specific anatomical requirements. When mandibular advancement is indicated, patient-fitted devices can be constructed to advance the mandible with the TMJ prostheses. When the orthognathic and TMJ surgeries are performed concomitantly, the surgical sequencing is as follows: (1) through an endaural or preauricular incision, condylectomy, discectomy, and joint debridement; (2) coronoidectomy with detachment of the temporalis muscle if the mandible is to be significantly advanced or vertically lengthened; (3) through a submandibular incision, detach the masseter and medial pterygoid muscles, mobilize the mandible to obtain the new position of the mandible; (4) apply intermediate splint and MMF; (5) place TMJ prostheses; (6) harvest fat graft from the abdomen (or from elsewhere if preferred) and pack around articulating area of TMJ prostheses; (7) remove MMF and intermediate splint; (8) maxillary osteotomies, mobilization, segmentation if indicated, application of palatal splint; (9) intranasal procedures such as turbinectomies and nasoseptoplasty if indicated; (10) maximize occlusal fit, apply MMF, rigid fixation to the maxilla and appropriate bone grafting, if indicated, with bone or porous block hydroxyapatite. Release MMF and check occlusion; and 11) other procedures (i.e., genioplasty, facial augmentation, or rhinoplasty).

27.4 Case 1 (Figs. 27.3, 27.4, and 27.5)

This 18-year-old female reported the onset of her TMJ symptoms at about the age of 13 when her joints started to click and pop. By the age of 16, the clicking stopped but her pain involving the TMJs and headaches had significantly increased. She was referred for treatment at the age of 18 years. Although she had good facial symmetry in the frontal view, in profile she had the HOP facial morphology commonly seen with AICR with the retruded mandible and chin as well as an end-on Class II occlusion (Figs. 27.3a, b, 27.4a, b, and 27.5a). On a scale of 0 to 10, where 0 equals no pain and 10 the worse pain imaginable, she rated her headaches at 6, TMJ pain at 7, and myofascial pain at 8. She had significant difficulties eating and chewing related to her pain issues and was on a relatively soft diet. She rated her disability at 7, where 0 indicates no disability and 10 means totally disabled. She had previous extensive orthodontics with an unstable outcome. She was in orthodontic treatment for a second time, at the initial surgical evaluation. Her diagnoses consisted of the following: (1) bilateral TMJ AICR; (2) maxillary anteroposterior (AP) and posterior vertical hypoplasia; (3) mandibular AP and posterior vertical hypoplasia; (4) Class II end-on occlusion; (5) high occlusal plane angle; (6) impacted third molars x 4; (7) hypertrophied turbinates with nasal airway obstruction; and (8) TMJ pain, myofascial pain, and headaches. The single-stage surgical treatment consisted of the following: (1) bilateral TMJ articular disc repositioning and ligament repair with Mitek anchors (Fig. 27.10); (2) bilateral mandibular ramus osteotomies to advance the mandible in a counterclockwise direction; (3) multiple maxillary osteotomies to down graft the posterior aspect; (4) anterior mandibular horizontal osteotomy to augment the chin (Fig. 27.5b); (5) removal of impacted third molars x4; and (5) bilateral partial inferior turbinectomies.

The patient was evaluated three years postsurgery with the following findings: no TMJ pain, headaches, myofascial pain was observed; incisal opening was 43 mm (presurgery was 28 mm); excursion movements of 5 mm in each direction were observed; good jaw function and no disability were noted; and good facial balance (Fig. 27.3c, d) and stable occlusion (Fig. 27.4c, d) were observed.

27.4.1 The Importance of the Articular Disc in Orthognathic Surgery Stability

Goncalves et al. [53] reported a retrospective study that evaluated the records of 72 patients who underwent CCWR of the MMC. The sample was divided into three groups to address the influence of TMJ health and articular disc surgical repositioning relative to postoperative stability. Group 1, with healthy TMJs, underwent double jaw surgery only. Group 2, with articular disc dislocation, underwent articular disc repositioning using the Mitek anchor technique concomitantly with orthognathic surgery. Group 3, with articular disc dislocation, underwent orthognathic surgery only. Preoperative characteristics for all patients included high occlusal plane angle, maxillary and mandibular retrusion, and increased anterior facial height. All 3 patient groups had similar dentofacial deformities and underwent orthognathic surgical procedures performed by the same surgeon in the same manner with rigid fixation. Each patient's lateral cephalograms were traced, digitized twice, and averaged to estimate surgical changes and postoperative stability. The MMC was advanced with CCWR similarly in all 3 groups; approximately 13 mm measured at menton. Postoperatively, the occlusal plane angle increased in Group 3 (37% relapse rate), but remained stable in Groups 1 and 2. Postoperative mandibular changes in the horizontal direction demonstrated a significant A-P relapse in Group 3 at menton (28%), B point (28%), and lower incisor edge (34%), but remained stable in Groups 1 and 2. MMC advancement with CCWR of the OPA is a stable procedure for patients with healthy TMJs and for patients undergoing simultaneous TMJ disc repositioning using the Mitek anchor technique. Those patients with preoperative TMJ articular disc displacement who underwent double jaw surgery and no TMJ intervention experienced significant relapse.

Chemello et al. [6] and Satrom et al. [54] reported that mandibular advancement in double jaw surgery (with or without CCWR) using rigid internal fixation with healthy TMJs is a stable procedure over the long term, with a mean A-P relapse at point B of 6% regardless of the amount of surgical advancement performed. On the other hand, Wolford et al. [55] evaluated 25 consecutive patients (23 females and 2 males) with jaw deformities and displaced articular discs (confirmed by MRI) who were treated with orthognathic surgery only, including mandibular advancement, and stabilized with rigid fixation. The average postoperative relapse at point B was 36% of the mandibular advancement, and the average distance from the condyle to point B decreased by 34%, indicating condylar resorption. Six patients (24%) demonstrated significant postoperative condylar resorption (3–8 mm), resulting in Class II anterior open bite malocclusion. The increased loading of the TMJs as a result of the mandibular advancement most likely stimulated the resorption process. New onset or aggravation of TMJ symptoms (e.g., pain, TMJ dysfunction) occurred at an average of 14 months after surgery. At the completion of the study, 48% of the patients required TMJ and repeat orthognathic surgery. Before surgery, 36% of the patients complained of pain or discomfort, but at 2.2 years postoperatively, and 84% of the patients reported pain with a 75% increase in pain intensity compared with the preoperative pain level. Only 4 of the 25 patients (16%) had a stable outcome without pain. This study clearly demonstrates the problems associated with performing orthognathic surgery only on patients with coexisting TMJ articular disc dislocations.

27.5 Case 2 (Figs. 27.11, 27.12, and 27.13)

This 20-year-old female presented with juvenile idiopathic arthritis (JIA) with onset at approximately age 5 but first noted clinically to be affecting the TMJs at age 12, with progressively worsening facial deformity related to condylar resorption (Figs. 27.11a, b, 27.12a, b, and 27.13a). She had polyarthritis involving the neck, hands, feet, and TMJs, and moderate pain issues around the head and neck (4 on a scale of 0 to 10), with jaw function rated at 3 and diet at 2. Her incisal opening was 38 mm and excursions 6 mm to the right and left. Her diagnosis included (1) bilateral TMJ JIA; (2) maxillary A-P and posterior vertical hypoplasia; (3) mandibular A-P and posterior vertical hypoplasia; (4) Class II occlusion with anterior open bite; (5) microgenia; (6) decreased oropharyngeal airway (A-P dimension 3 mm) with significant sleep apnea symptoms; and (7) hypertrophied turbinates creating nasal airway obstruction. The MRIs demonstrate the severe destruction of the condyles resorption of the articular eminences, and reactive pannus surrounding the discs (Fig. 27.14a, b). Presurgical orthodontics prepared the patient for surgery.

Surgery included (Fig. 27.13b): (1) bilateral TMJ reconstruction and counterclockwise rotation of the mandible with TMJ Concepts patient-fitted total joint prostheses; (2) bilateral coronoidectomies; (3) bilateral TMJ fat grafts packed around the articulating area of the prostheses, harvested from abdomen; (4) multiple maxillary osteotomies for counterclockwise rotation and advancement; (5) anterior mandibular horizontal osteotomy to augment the chin; and (6) bilateral partial inferior turbinectomies.

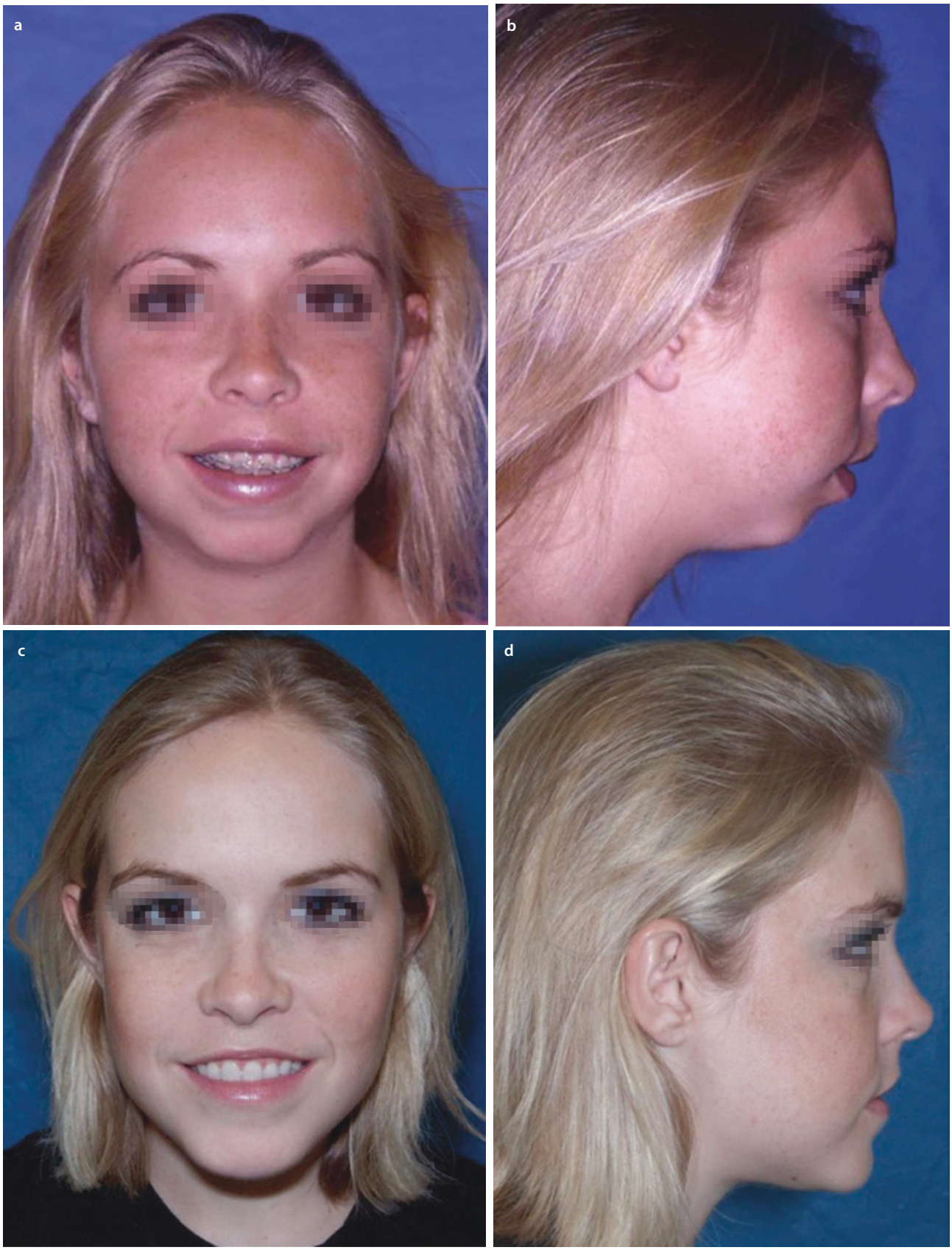

Fig. 27.11 Case 2: **a**, **b** a 20-year-old female with JIA and grossly resorbed mandibular condyles, retruded mandible and maxilla, posterior maxillary vertical hypoplasia, high occlusal plane angle facial morphology, decreased oropharyngeal dimension (3 mm) and sleep apnea symptoms as well as hypertrophied turbinates and difficulty breathing through the nose. **c**, **d** The patient is seen 4 years postsurgery demonstrating significantly improved facial balance and function with a stable occlusion

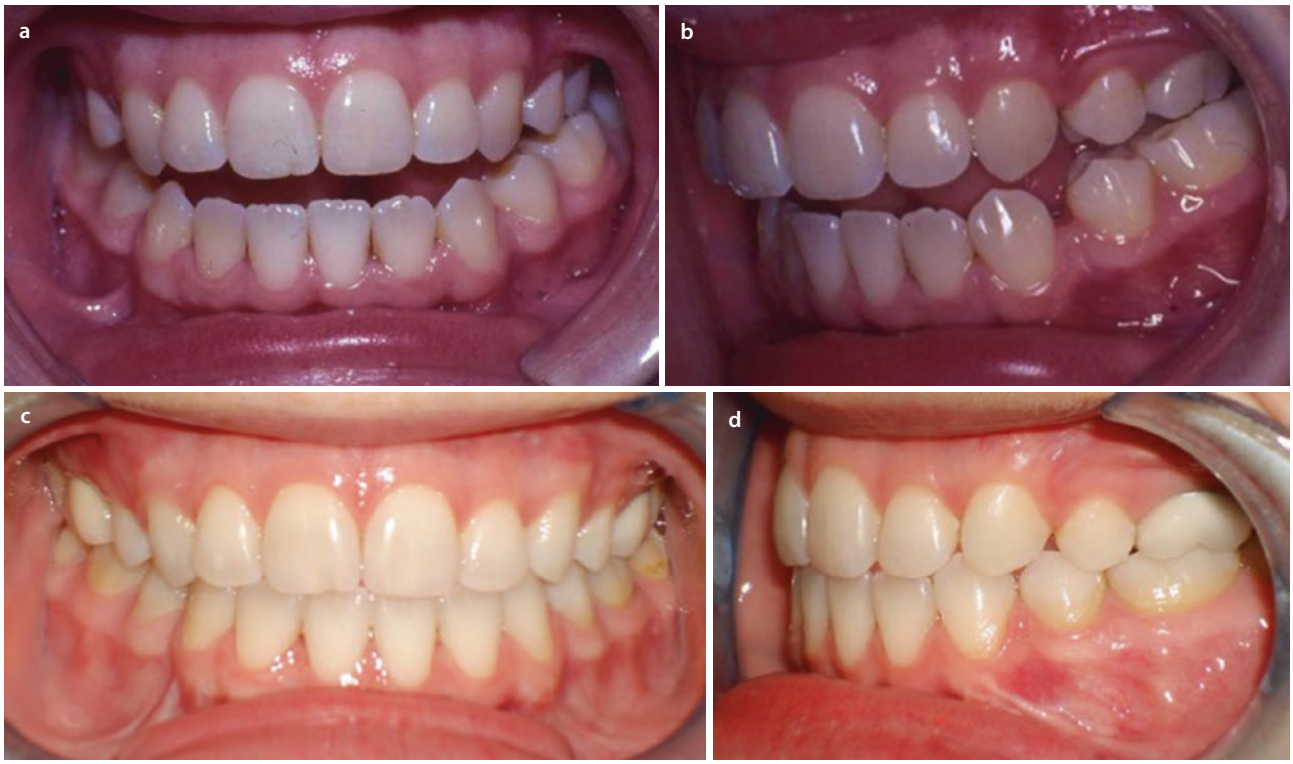

Fig. 27.12 Case 2 **a**, **b** The patient has a Class II occlusion presurgery with an anterior open bite. **c**, **d** At 4 years postsurgery she demonstrates a stable Class I occlusion

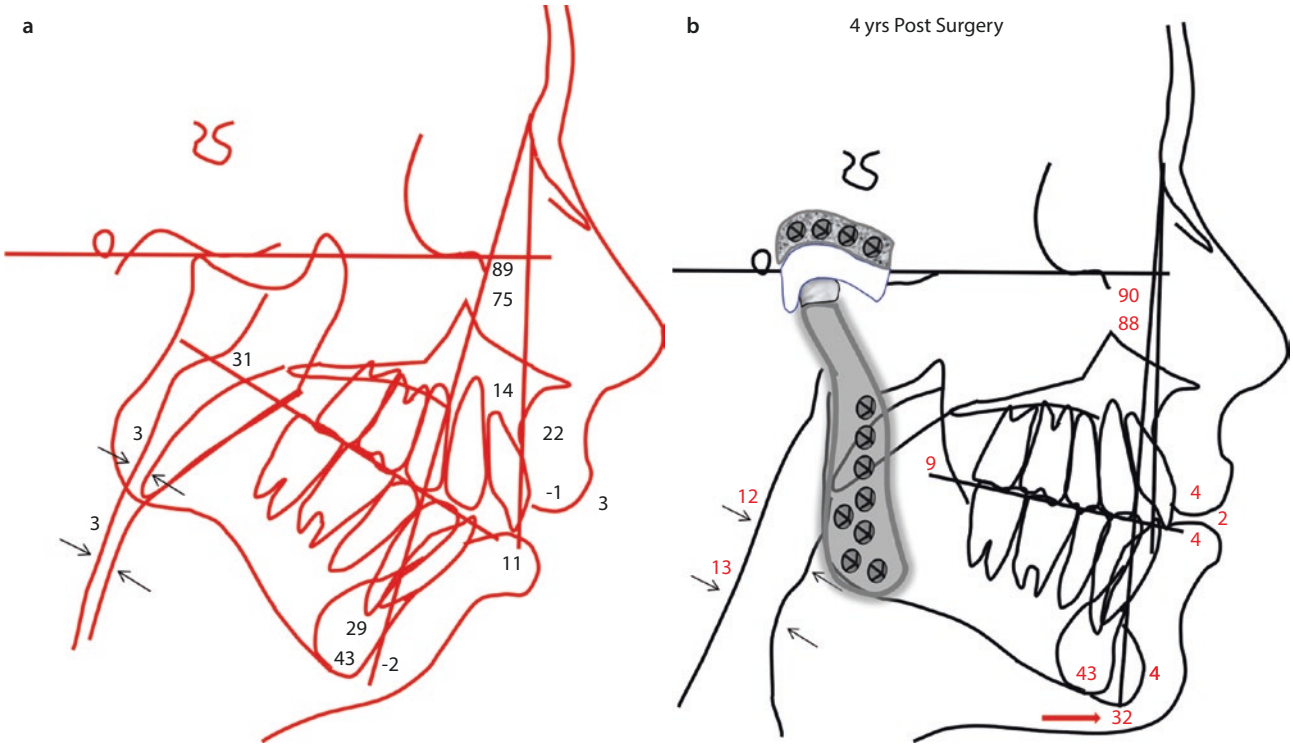

Fig. 27.13 Case 2: **a** Cephalometric analysis shows severe jaw deformity with retruded maxilla-mandible, high occlusal plane angulation, and decreased oropharyngeal airway. **b** The 4-year postsurgery cephalometric analysis demonstrates the counter-clockwise rotation of the maxillomandibular complex with TMJ Concepts total joint prostheses. The chin is augmented with a bony genioplasty. Maxillary incisors advanced 4 mm, Pogonion advanced 32 mm, and the occlusal plane decreased 22 degrees, creating improved function and facial balance. There is a normal oropharyngeal airway of 13 mm

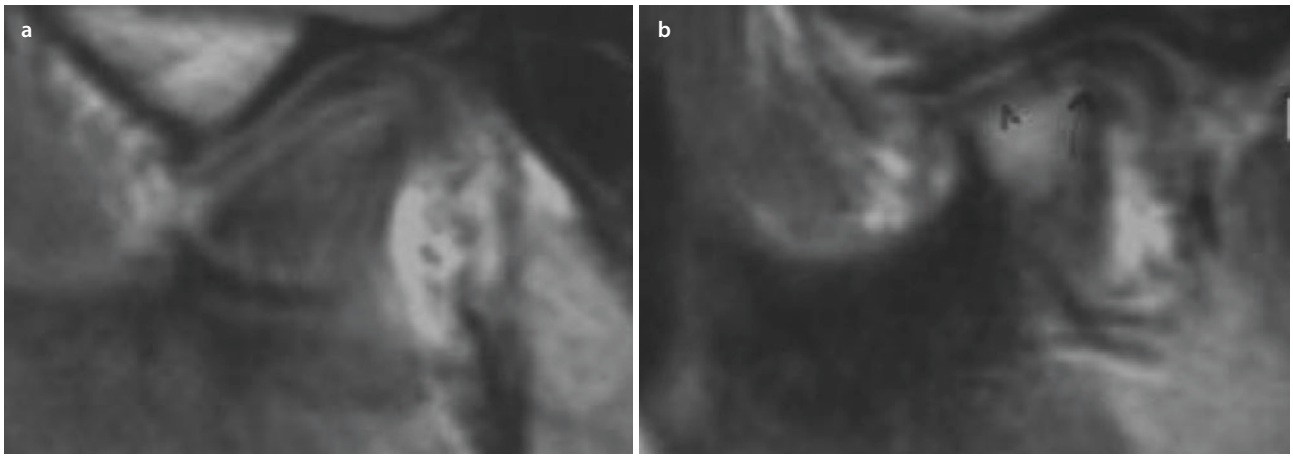

Fig. 27.14 Case 2: MRIs of the TMJs; **a** right TMJ sagittal view, **b** left TMJ sagittal view showing the destruction of the condyles and articular eminences, common in JIA cases. Notice the "mushrooming" of the remainder of the condylar neck process. The articular discs are in a relatively normal position but surrounded by a reactive pannus

At four years post-surgery she was pain-free and the following were observed: incisal opening at 42 mm, excursive movements 3 mm bilaterally, stable Class I occlusion, improved facial balance, normal diet, good nasal and oropharyngeal airway, and elimination of sleep apnea symptoms (Figs. 27.11c, d and 27.12c, d).

27.5.1 Outcome Stability with TMJ Concepts Patient-Fitted Total Joint Prostheses

Dela Coleta et al. [56] evaluated 47 female patients for surgical stability following bilateral TMJ reconstruction using TMJ Concepts patient-fitted TMJ total joint prostheses, TMJ fat grafts, and CCWR of the MMC with Menton advancing an average of 18.4 mm and the OPA decreasing an average of 14.9 degrees. Average follow-up was 40.6 months. Results demonstrated minor maxillary horizontal changes while the mandibular measurements remained very stable.

Pinto et al [57] evaluated the same 47 female patients relative to pain and dysfunctional outcomes. Patients were divided into two groups based on the number of previous surgeries: Group 1 had 0–1 previous surgeries while Group 2 had 2 or more previous surgeries. Significant improvements (37–52%) were observed for TMJ pain, headaches, jaw function, diet, and disability. MIO increased 14%. Group 1 patients had better pain and jaw function results than Group 2 patients. For patients who did not receive fat grafts around the prostheses and had previous failure of alloplastic TMJ implants, more than half required secondary surgery including TMJ debridement for removal of foreign-body giant-cell reaction, fibrosis, and/or heterotopic bone formation. The Dela Coleta et al. and Pinto et al. studies demonstrated that end-stage TMJ patients could be treated in one operation with TMJ Concepts patient-fitted TMJ total joint prostheses, fat grafts, and MMC CCWR for correction of an associated dentofacial deformity with good stability and improvement in pain and TMJ function.

Although the life expectancy of this device is unknown, Wolford et al [58] published a 20-year follow-up study of 56 patients who had received the Techmedica total joint prostheses between 1989 and 1993. There were statistically significant improvements in all parameters including incisal opening, jaw function, TMJ pain, and diet, with 85.7% of the patients reporting significant improvement in their quality of life. The greater the number of previous TMJ surgeries, patients reported a lower degree of subjective improvement, but they did report increased objective mandibular function and improved quality of life. There were no reports of device removal due to material wear or failure.

Wolford et al [56–65], Mercuri et al [66–72], and others [73–76] have published numerous studies in reference to outcome data using patient-fitted TMJ total joint prostheses. A summary of these publications have produced the following facts in reference to the TMJ Concepts total joint prostheses: (1) TMJ Concepts prostheses are superior to autogenous tissues for end-stage TMJ reconstruction relative to subjective and objective outcomes; (2) after two previous TMJ surgeries, autogenous tissues have a very high failure rate, whereas patient-fitted total joint prostheses have a high success rate; (3) no donor site morbidity; (4) increased number of previous TMJ surgeries produces a lower level of improvement related to pain and function outcomes compared to patients with 0 to 1 previous TMJ surgeries; (5) failed TMJ alloplastic reconstruction

(i.e., P/T, silastic, metal-on-metal articulation, etc.) can create a foreign-body giant-cell reaction and/or metallosis, best treated by joint debridement and reconstruction with patient-fitted total joint prostheses; (6) fat grafts packed around the articulating area of the prostheses improve outcomes relative to decreased pain, improved jaw function, and decreased requirement for repeat surgery [77–80]; (7) osseo-integration of the TMJ Concepts fossa and mandibular components occur and is important for long-term stability; (8) posterior stop on the fossa component is important to stabilize the joint, jaw position, and occlusion; (9) concomitant orthognathic surgery can be performed at the same time as the TMJs are reconstructed; and (10) 20-year follow-up study [58] demonstrated improvements in pain, jaw function, diet, incisal opening, and quality of life.

27.6 Summary

CCWR of the MMC is highly predictable for quality treatment outcomes in orthognathic surgery in the presence of healthy and stable TMJs. If the TMJs are not stable and healthy, CCWR for orthognathic surgery may be unsatisfactory relative to function, esthetics, skeletal and occlusal stability, as well as pain. The oral and maxillofacial surgeon should be suspicious of possible TMJ problems in the following types of patients: (1) high occlusal plane angle facial morphologies with retruded maxilla and mandible; (2) progressive development of anterior or lateral open bites; (3) progressively worsening occlusal and jaw relationships; (4) facial asymmetry, particularly with progressive worsening; and (5) patients reporting headaches, TMJ pain, myofascial pain, history of clicking and popping of the TMJs, and/or ear symptoms. The surgeon should not ignore these symptoms. With one or more of these symptoms, patients should be evaluated for possible TMJ pathology. An MRI of the TMJs can aide in the identification of the specific TMJ pathology. Failure to recognize and treat these conditions can result in significant orthognathic surgery relapse, increased pain, and a greater complexity of subsequent treatment for patients.

During the past 30 years, major advancements have been made in TMJ diagnostics and the development of surgical procedures to treat and rehabilitate the pathological, dysfunctional, and painful TMJ. Research has clearly demonstrated that TMJ and orthognathic surgery that involves CCWR of the MMC can be safely and predictably performed at the same operation, but it does necessitate the correct diagnosis and treatment, as well as requires the surgeon to have expertise in both TMJ and orthognathic surgery. The surgical procedures can be separated into 2 or more surgical stages, but the TMJ surgery should be done first. With the correct diagnosis and treatment plan, combined TMJ and orthognathic surgical approaches provide complete and comprehensive management of patients with coexisting TMJ pathology and dentofacial deformities requiring CCWR of the MMC.

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Maxillomandibular Advancement

Reza Movahed

Contents

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Overview

Obstructive sleep apnea (OSA) is a common sleep and respiratory disorder characterized by episodic airway collapse during sleep with either a partial or a complete blockage of airflow. Insufficient sleep and its associated daytime sleepiness are common symptoms that can be significantly disruptive to a patient's work life and other activities of daily living (ADL), rendering intervention for OSA an important objective for clinical medicine, which include CPAP, a range of symptom-relieving devices, and adjunct treatments, such as oral appliances, pharmacotherapy, and surgical interventions, including most notably maxillomandibular advancement (MMA), which has demonstrated excellent effectiveness and long-term reliability up to and including a cure for OSA.

The existence of OSA as a discrete pathological disorder was first described by physician William Osler in 1918, a physician who suggested OSA patients were "Pickwickian," a term he coined that referenced a character named Joe in the Charles Dickens's novel *The Pickwick Papers,* denoting a patient who is hungry, red-faced, and consistently suffering from daytime sleepiness characteristically interrupting some task [1, 2]. In 1956, Bickelmann et al. [3] published one of the first known descriptions of OSA and used "Pickwickian syndrome" to describe patients with daytime sleepiness (n = 346). Since then, several noninvasive and surgical techniques have been developed to address OSA, each with varying success. This chapter describes the landscape of OSA and the rationale and approach for MMA to treat and even cure this widespread syndrome.

28.1 The Growing Problem of Obstructive Sleep Apnea

OSA affects a significant proportion of the population. Young et al. estimated in 2002 that mild OSA may be found in at least 1 in 5 adults and moderate OSA in at least 1 in 15 adults [4]. The Wisconsin Sleep Cohort [5] defined OSA as an apnea–hypopnea index (AHI) of ≥ 5 events per hour and found a prevalence in women and men aged 30–60 years of age of 9% and 24%, respectively. In a 2013 reanalysis of the Wisconsin Sleep Cohort by Peppard et al. [6], the authors adjusted data based on more recent rates of overweight and obese individuals, which predicted a significantly marked increase in OSA prevalence that was estimated to impact 17.4% of women and 34% of men between 30 and 70 years of age (with the same expected AHI of ≥ 5 events per hour).

28.1.1 Symptoms

The most common symptoms of OSA are insufficient sleep and daytime sleepiness. However, these may (or may not) be accompanied by a range of other predominant symptoms associated with OSA, such as dry mouth, nocturia, morning headache, cognitive impairment/brain fog, mood irritability, and longer lasting psychiatric impact (e.g., cognition and memory loss), all of which may lead to a very noticeable impact on quality of life (QOL) [7–10] and be a cause of personal distress. OSA is associated with lower workplace productivity [11] and a greater risk of occupational injuries, as well as injuries sustained in motor vehicle accidents up to and including fatality [12–14]. OSA represents a formidable public health burden associated with significant costs [15] to individuals and the health-care system if OSA is left untreated, particularly in the form of a range of comorbid conditions that accompany it.

28.1.2 Comorbid Associations with OSA

There is a lack of awareness OSA in not only the general public [16–18], but primary care providers, as well [19]. Therefore, comorbid conditions are also likely underrecognized. However, detecting comorbidities offer the opportunity for clinicians to recognize and explore potential links between OSA and its many reported associations, the presence of which could potentially warrant screening for and ruling out of OSA. Several medical conditions may lead to or exacerbate OSA, chief of which are metabolic disorders, e.g., metabolic syndrome and obesity, including a greater risk for OSA in individuals with a greater body mass index (BMI) and neck circumference [20–28].

28.1.2.1 Obesity and Neck Circumference

Obesity is one of the most common major risk factors contributing to the natural course of OSA and is directly associated with OSA severity [29]. Many OSA patients exceed a BMI of 30 and therefore necessitate specialized education and additional care, and additional postoperative nasopharyngolaryngoscopy has been recommended for obese patients [30]. A disproportionate number of patients have a higher BMI or greater neck circumference [31], with the latter identified as a predictor of treatment failure [31]. In 2014, Cizza et al. reported that neck circumference was associated with OSA and metabolic syndrome in both obese men and premenopausal obese women sleeping less than 6.5 hours per night [26]. Katz et al. [32] reported that children in the 95th percentile of neck circumference adjusted for age and sex had a significantly increased risk for OSA and

suggested that neck circumference is a validated screening tool for pediatric OSA. Evidence suggests that treating OSA reduces obesity, but the reciprocal may also be true [33]. Obesity or patients of advanced age who have received a mandibular setback only are at higher risk for developing sleep-disordered breathing. Therefore, a two-jaw procedure has been advocated for these patients with skeletal class III patients to anticipate pharyngeal space reduction known caused by mandibular setback [34].

28.1.2.2 Other Important Comorbidities: Cardiovascular, Circadian, Immune/Autoimmune, Cancer, Nutrition, and Psychiatric/Cognitive

OSA has also been positively linked to cardiovascular disease [35], including extensive evidence of comorbid hypertension [23, 35–45], stroke (independent OSA association potentially leading to stroke as well as impacting outcomes and recovery for patients following stroke [46–60]), and other life-threatening events, circadian dysregulation [21, 61] (which has been suggested to be the "next frontier" in OSA research strongly suspected to be linked to the cardiopulmonary system [62]), immune (including autoimmune) disorders and the inflammatory response [20, 63–65], increased risk of cancer [66], nutrition (e.g., higher alcohol consumption, other dietary patterns and risk factors, and potential lifestyle modifications aimed at weight loss and to address nutritional deficiencies [10, 67–71]), psychiatric disturbances (e.g., depression, anxiety, and schizophrenia [10, 72–76]), and cognitive impairment [53, 57, 77–79].

28.1.3 Traditional OSA Treatments Have Mixed Success

If risk factors and OSA can be positively diagnosed, a variety of treatments exist to treat and relieve OSA symptoms and sequelae [13, 80–82]. Many patients begin with what has been considered one of the go-to "first-line" treatments of continuous positive airway pressure (CPAP), which has demonstrated effectiveness in improving airway patency, AHI, and associated reductions in daytime sleepiness [8, 13, 81, 83, 84]. Because of its discomfort, however, CPAP is a modality with which many patients struggle to remain adherent [85–87] and, in many cases, become intolerant and ultimately discontinue, and therefore is not a universally reliable intervention for OSA.

28.1.3.1 Noninvasive Treatments

Other noninvasive lifestyle modifications have also been used as first-line interventions for OSA, but each has limited success, as reported in the 2013 systematic review and meta-analysis by Araghi et al. [88], who evaluated seven randomized controlled trials (n = 519) evaluating weight reduction initiatives. These programs included both exercise and weight management with the intention to decrease body fat percentage, smoking cessation to reduce smoking-related risk factors, simple changes in sleep position to improve airway patency, and avoidance of sedatives and alcohol. The authors concluded that while various lifestyle modifications may yield some improvements of OSA, particularly for patients with severe symptoms, they are ultimately insufficient to normalize them [88].

Over the years, several oral appliances have been marketed and chosen by providers and patients because of their noninvasiveness and ease of use. However, these adjuncts are also not ideal because they are only palliative and do not address the root cause of airway obstruction; they are also prone to varying rates of nonadherence and failure [89]. A recent 2-year follow-up study of CPAP vs. oral appliance therapy found that there were no significant differences in treatment success for mild-to-severe OSA patients [90]. Despite modest effectiveness of the aforementioned treatments to alleviate some measures of OSA-related symptoms, they do not address the root pathogenic cause of OSA itself and ultimately fail to provide a permanent solution.

28.1.3.2 Surgical Interventions

A variety of surgical interventions have been explored to attempt to modify airway characteristics (see Table 28.1).

Tracheotomy/tracheostomy has long been considered a potentially curative approach. From the late

Table 28.1 Surgical techniques for OSA (Fleisher and Krieger, 2007 [91])

	Technique	Location
Phase 1	Nasal surgery (septoplasty, turbinectomy)	Nose
	Uvulopalatopharyngoplasty (UPPP)	Oropharynx (retropalatal airway)
	Genioglossal advancement	Oropharynx (retrolingual airway)
	Radiofrequency ablation	Oropharynx (retrolingual airway)
	Modified genioplasty	Oropharynx + hypopharynx
	Hyoid myotomy suspension	Oropharynx + hypopharynx
Phase 2	Maxillomandibular advancement	Nasopharynx/oropharynx/hypopharynx
	Tracheostomy	Trachea
	Bariatric surgery	Gastric

1960s to the early 1980s, tracheostomies were the primary surgical approach of choice for OSA subjects who had failed in prior attempts at traditional medical management. One of the first subjects treated by tracheostomy was reported by Valero and Alroy in 1965 [92], who detailed a thin patient who sustained traumatic micrognathia and who was observed to have recurrent apneas during hospitalization. Tracheostomy was performed and resolved the patient's hypersomnia. Another early documented tracheotomy for upper airway obstruction was described by Kuhlo in 1969 [93] of a classical "Pickwickien" patient.

In 2014, Camacho et al. [94] performed a systematic review and meta-analysis of tracheostomy for the treatment of OSA. From the included 18 studies, the reviewers found that tracheostomy was a highly effective intervention showing both clinically significant and statistically significant decreases in the mean observed apnea index (AI) regardless of BMI, improved oxygen desaturation indices, reduced sleepiness, and significant reductions in mortality. In 2015, Camacho et al. [95] performed another systematic review and meta-analysis of tracheostomy vs. MMA for morbidly obese OSA patients (34 MMA patients and 14 tracheostomy patients). Because of the small samples, the authors reported they could not draw any definitive conclusions and called for higher-level studies. Camacho et al. [96] also suggested in 2016 that mini-tracheostomies, some as small as 4 mm, likely have a place in the short-term treatment of upper airway obstruction.

While highly effective and even curative, the tracheotomy or tracheostomy procedure is not appealing for a majority of patients for a variety of reasons, such as frequent coughing up of mucus and plugging of the tube, risk of trachea-innominate fistula, aspiration, susceptibility to infection (e.g., pneumonia), formation of granulation tissue, unappealing appearance, inability to swim, and vocal cord paralysis [97–99]. At present, there are few ideal tracheostomy candidates and it is generally considered a procedure of last resort. However, some patients may benefit and be considered potential candidates if they have failed medical management, refused MMA, or have conditions that otherwise contraindicate MMA, as well as contraindications/refusal for other competing soft tissue procedures.

Various other procedures have been considered and performed for OSA patients, including procedures that target the nasal airway (e.g., septoplasty, partial inferior turbinectomy, use of spreader batten grafts), interventions in the retropalatal region (uvulopalatopharyngoplasty [UPPP], targeting the soft palate, among other procedures), and procedures that target the retrolingual region (genioglossus advancement (for base of tongue obstruction [100]), midline glossectomy, hyoid suspension, and hyoidmyotomy suspension, amongst others). However, the success of these procedures is dismal at best and only targets a specific anatomical site.

28.1.4 MMA as First-Line Therapy

While there are a variety of surgical procedures to address OSA, a growing body of evidence-based data (including from systematic reviews and meta-analyses) demonstrates that surgical intervention, specifically MMA, a well-established orthognathic procedure for OSA with a relatively low rate of major complications (1%) [101], currently appears to be the most effective, long-term solution for OSA. In 1993, Riley et al. put forward their two-phase Stanford Protocol [102] (Fig. 28.1), phase 1 of which consisted of presurgical evaluation up to and including numerous targeted procedures.

Genioglossus advancement, glossectomy, and hyoid suspension were previously considered a phase 1 treatment for OSA. However, over time these treatments have failed to yield acceptable rates of success. For example, UPPP has a success rate of 40–50% in the short term [103], and long-term outcomes show an even lower rate of success after 4-year follow-up (35%) [104]. Laser-assisted uvulopalatoplasty (LAUP), an outpatient procedure for snoring, requires multiple procedures and may result in significant postoperative pain [105]. It has been suggested that palatal implants may be needed to improve results following LAUP [106]. A 2003 randomized trial of patients treated with LAUP vs. untreated patients showed that there was no difference between groups in measures of excess daytime and QOL [107]. The authors concluded that their results do not support the use of LAUP for OSA. Partial glossectomy also has a questionable rate of success and is known to be accompanied by several complications, such as dysphagia, odynophagia, loss of taste sensation, and reduced tongue mobility [108, 109]. Hyoid suspension also has a success rate as low as 17% [110]. A postoperative polysomnography would ultimately be performed after these procedures to evaluate the procedure success. If this step failed to yield success, the patient would move on to phase 2: MMA.

Given the improved success of current treatments optimizing postoperative healing, recovery, and QOL, the current treatment approach is to move directly to MMA if patients are not tolerant of CPAP therapy. For some patients, the effectiveness of CPAP decreases over time. As a result, MMA has become the first surgical line for OSA therapy. MMA can result in significant enlargement of pharyngeal and hypopharyngeal airway volume by expanding the anatomic area. Several sleep studies have demonstrated that MMA has been shown to lead to significant decreases in the apnea–hypopnea index (AHI) and is a long-lasting intervention that achieves positive change to alleviate OSA by dramatically increasing airway dimensions. It is also important to note that fluid dynamic studies have shown that MMA is responsible for reversing drops in negative pressure and airway collapse (drop in pharyngeal walls) [111] and that less effort is required to respirate post-

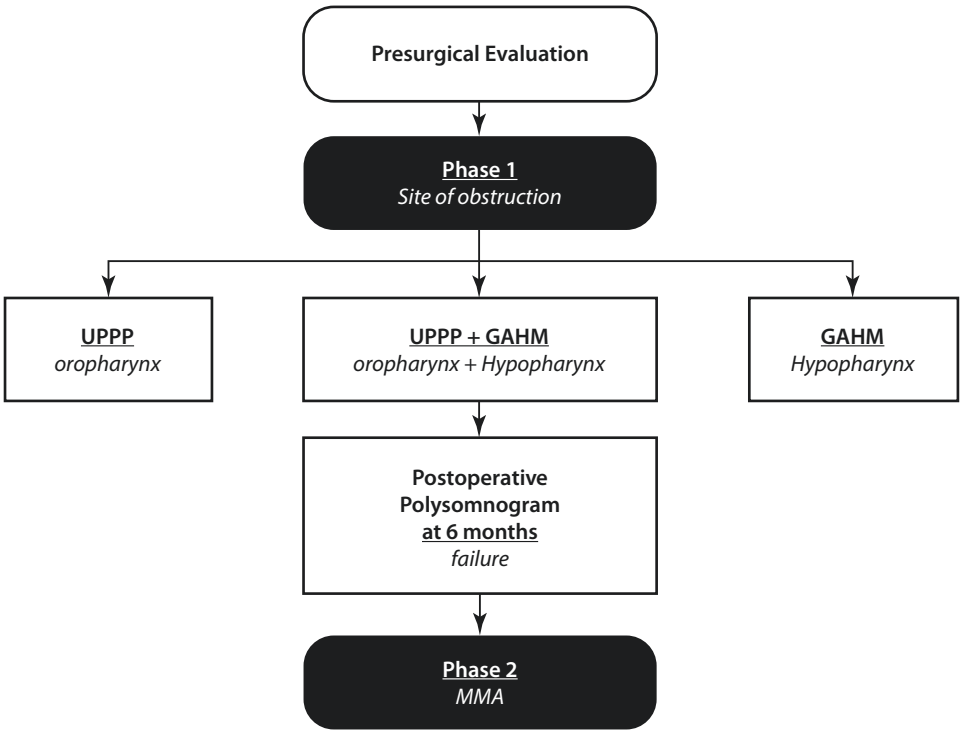

Fig. 28.1 Surgical protocol for OSA initially proposed by Riley et al. in 1993 [102]

MMA [112], that MMA improves multiple health-related functional outcomes and QOL [113–117], and the positive airway changes facilitate normal respiration during sleep leading up to and including an ostensible cure of the condition [80, 101, 111, 113, 118, 119].

Regardless of the procedure selected, whether it be noninvasive or surgical, including tracheostomy, or another upper airway surgery—especially MMA—individual patients should always be assessed on a case-by-case basis. They should be closely evaluated based on their medical history and concurrent medical conditions, pharmacotherapy, and fitness for treatment. For surgical interventions, there are likely other factors that may influence or even dictate selection of surgery, including surgeon training, confidence, and preference, institutional resources, available imaging, surgical equipment, staff, and, ultimately, patient choice.

28.2 MMA as a Definitive Intervention for OSA

28.2.1 Postoperative Stability of MMA: Short- and Long-Term Safety and Effectiveness

In the past 25 years, there has been no shortage of evidence demonstrating that MMA is both safe and effective in improving OSA outcomes and QOL and that it is a viable first-line therapeutic alternative to continuous positive airway pressure (CPAP) for many patients [120]. Makovey et al. confirmed in 2017 that MMA for OSA was highly effective for their group of patients treated for moderate-to-severe OSA, showing a 2.5-fold volume in airway increase in their sample of 20 patients (mean age, 48.8 ± 12.3 years). Furthermore, a 2017 overview of 11 systematic reviews concluded that MMA reliably and positively improves the dimensions of the pharyngeal airway in OSA patients. Meta-analyses of postoperative MMA data by Holty and Guilleminault [101] in 2010 and Zaghi et al. [121] in 2016 showed statistically significant reductions in AHI across their pooled studies [101, 121] and improvements in other measures, such as the respiratory disturbance index (RDI) [121], as well as lowest optimal values of nocturnal oxyhemoglobin (SpO_2 nadir) [101]. Another marker of surgical success recently proposed to determine stability of the lateral pharyngeal wall post-MMA formulated by a composite of AHI, oxygen-desaturation index (ODI), and Epworth Sleepiness Scale (ESS) [122]. MMA has been reported with high treatment success rates [101, 123]. For instance, Zaghi et al. demonstrated a success rate of 85.5% and a cure rate of 38.5% [121]; furthermore, this systematic review confirmed that predictors of surgical success were younger patients and had lower preoperative AHI and higher range of maxillary advancement.

- The 2018 systematic review and meta-analysis by John et al. analyzed 20 studies and 462 subjects (85.3% male and 14.7% female) published between 2000 and 2015. They reported that MMA surgical

success was 100% with respect to AHI (>50% change or <20 events per hour post-MMA) and RDI scores (<15 per hour and ≥50% reduction post-MMA). Furthermore, among the interventional studies included for analysis, the authors reported significant improvements in the outcome measures of AHI, RDI, Epworth Sleepiness Scale (ESS), and lowest oxygen saturation (LSAT). Mean changes between pre- and post-MMA were reported for AHI (12 studies; n = 251), RDI (6 studies; n = 163), ESS (7 studies; n = 118), LSAT (16 studies; n = 397), and BMI (11 studies; n = 217) were −44.76, −59.71, −8.02, +10.83%, and −1.02 kg/m^2, respectively (Table 28.2). Pooled forest plot analysis showed that MMA was favored at the meta-analysis level across all reported measures: AHI (P < 0.00001), RDI (P < 0.00001), ESS (P < 0.00001), LSAT (P < 0.00001), and BMI (P = 0.007). (See Figs. 28.2, 28.3, 28.4, 28.5, and 28.6) It is important to note the limitations of this meta-analysis. All included trials were reported after the year 2000, which represents some selection bias. Only one trial was randomized (Vicini et al. 2010 [124]). Furthermore, there was a high risk of information bias in the study by Dattilo and Drooger [125], an unclear risk of selection bias in Cohen-Levy et al. [126], Abramson et al. [127], and Li et al. [128], but an otherwise low risk of bias in all prospective cohorts. John et al. concluded that MMA is a successful treatment for OSA, confirming the same result in prior meta-analyses by Zaghi et al. in 2016 [121] and Holty and Guilleminault in 2010 [101].

28.2.1.1 Long-Term Follow-Up, Risk of Treatment Failure, and Complications

Evidence of MMA's longer-term safety and effectiveness for OSA was recently confirmed in 2015 by Boyd et al. in their prospective, two-center cohort study (n = 30) with a mean follow-up of 6.6 ± 2.8 years post-MMA, in which they reported their patients experienced adverse events (AEs) that mirrored shorter studies (e.g., malocclusion, minor bleeding, local infections successfully treated with antibiotics, frequent neurosensory changes of the inferior alveolar nerve, which commonly resolved by 12 months, and worsening of facial appearance noted in a low percentage of patients) [118]. In 2009, Blumen et al. also reported no major complications in their study of 50 patients post-MMA, citing that their most common complication was mental nerve sensory loss, which patients overwhelmingly deemed as secondary to the positive surgical outcome [136].

In 2017, Vigneron et al. reported long-term results of MMA for OSA with a minimum of 3-year follow-up and a mean follow-up of 13.8 years ±3.9, with 88 MMA patients treated between 1995 and 2009. The authors divided the patients into groups of either treatment success or treatment failure. AHI between treatment failure and success was 33.4 ± 18.7 vs. 4.7 ± 3.2, respectively (P < 0.004). Factors of long-term success included younger age (success rate was 100% for patients younger than 45 years of age), BMI <25, IAH <45, SNB <75%, narrow retrobasinlingual space (<8 mm), and patients who were treated with preoperative orthodontics. The risks of treatment failure are shown in Table 28.3.

Treatment success and the risk of complications may not be mutually exclusive. Many studies define treatment success as dependent upon meeting OSA research endpoints (e.g., AHI, blood pressure [BP]), sleepiness (ESS), QOL, etc.); however, this does not necessarily mean that complications were entirely averted. Although MMA has become an indispensable, permanent treatment for OSA with excellent outcomes, it must also be recognized for what it is: a major invasive surgical procedure for the correction of mandibular deformities, not unsusceptible to risk and the incidence of complications. In 2017, de Ruiter et al. [31] studied factors of treatment success and failure in 62 patients who underwent MMA for OSA. They confirmed that the AHI measure was useful to gauge treatment for both endpoints. They reported a 71% success rate (mean AHI reduction of 69%) and identified that the key predictors of treatment failures were age (58 vs. 53 years, respectively; P = 0.037) and a significantly larger neck girth (P = 0.008). Notably, the most common complications in MMA procedures were sensory impairment of the inferior alveolar nerve (60%) and malocclusion (24%) [31].

Generally, treatment failures may correlate with complications, but evidence to date is anecdotal. However, according to the expert opinion of the authors, there may indeed be a relationship between incidence of complications and treatment failure. This relationship may be impacted by several variables, including surgeon training and skill [144], longer procedure duration, infection susceptibility and wound contamination, psychologic resiliency of the patient, and the quality of postoperative care management.

28.3 Challenges in Balancing Airway Improvements with Facial Aesthetics

Subjects with OSA who have a normal facial profile pose a formidable challenge. MMA may achieve significant airway improvements, but advancement could also potentially disrupt facial aesthetics in patients who already have facial harmony. This can be a challenge to the MMA surgeon in balancing the potential improvement in the airway to achieve a good outcome for the patient's OSA while preserving or improving the patient's aesthetic profile.

Table 28.2 Mean (SD) values pre- and post-MMA of outcome measures from the 20 included studies of the meta-analysis by John et al. (2018) [129]

Study	AHI		RDI		ESS		LSAT		BMI	
	Pre-MMA	Post-MMA	Pre-MMA	Post-MMA	Pre-MMA	Post-MMA	Pre-MMA	Post-MMA	Pre-MMA	Post-MMA
Liu (2015) [122]	59.8 (25.6)	9.3 (7.1)	–	–	19.5 (2.9)	7.1 (2.6)	80.8 (7.6)	88.9 (3.4)	29.4 (5.1)	29.6 (4.1)
Butterfield (2015) [130]	45.54 (27.6)	7.71 (6)	–	–	13.15 (4.14)	6.14 (3.13)			30.33 (4.18)	30.05 (3.78)
Liao (2015) [131]	41.6 (19.2)	5.3 (4)	–	–	11.9 (7.3)	7 (3)	80.2 (9.7)	88.9 (5)	–	–
Schendel (2014) [132]	42.91 (21.17)	5.17 (8.34)	–	–	–	–	–	–	–	–
Cohen-Levy (2013) [126]	51.07 (15.21)	10.3 (7.24)	–	–	12.3 (5)	3.9 (2.8)	79.5 (13)	82.2 (5.4)	27.41 (3.5)	25 (2.6)
Boyd (2013) [133]	56.3 (22.6)	11.4 (9.8)	–	–	–	–	74.2 (13.8)	83.6 (10.5)	–	–
Serra (2012) [134]	–	–	–	–	–	–	85(6.8)	86(7)	–	–
Abramson (2011) [127]	–	–	–	–	–	–	80.5 (11.4)	90 (2.68)	–	–
Lin (2011) [135]	35.9 (17.95)	4.6 (4.03)	–	–	12.4 (4.5)	6 (2.3)	83 (7.2)	90.6 (3.6)	22.4 (2.73)	21.6 (2.35)
Vicini (2010) [124]	56.8 (16.5)	8.1 (7)	–	–	11.6 (2.8)	7.7 (1.3)			32.7 (5.8)	31.4 (6.5)
Blumen (2009) [136]	65.5 (26.7)	14.4 (14.5)	–	–	–	–	70.7 (19.2)	84.2 (7.4)	28.9 (4.6)	28.4 (4.2)
Fairburn (2007) [137]	69.22 (35.8)	18.57 (16.29)	–	–	–	–	80.45 (10.49)	87.8 (5.58)	33.85 (8.54)	34.65 (9.16)
Dattilo (2004) [125]	–		76.15 (45.71)	12.59 (12.11)	17.86 (3.76)	4.73 (2.6)	–	–	–	–
Goh (2003) [138]	70.7 (15.9)	11.4 (7.4)	–	–	–	–	58.6 (12.3)	83.9 (8.8)	29.4 (4.6)	27.2 (3.3)
Li 2002 [111]	–	–	75.3 (26.4)	10.4 (10.8)	–	–	74.2 (12)	86.9 (6.7)	33.5 (6.2)	32.3 (4.1)
Li (2001) [139]	–	–	61.6 (23.9)	9.2 (8)	–	–	75.9 (10.6)	87.5 (4.7)	–	–
Li (2001) [140]	–	–	60.3 (22.2)	10.8 (9.4)			75.8 (12.4)	86.7 (6.1)	–	–
Li 2000 [141]	–	–	60.3 (22.2)	10.8 (9.4)	–	–	73.3 (13.2)	88.1 (4.1)	–	–
Bettega (2000) [142]	59.3 (29)	11.1 (8.9)	–	–	–	–	82 (11)	90 (7)	26.9 (4.3)	25.4 (3.3)
Li (2000)	–	–	83 (30.1)	10.6 (10.8)	–	–	63.9 (17.7)	86 (7.9)	45 (5.4)	43 (4.3)
Mean change in pre- and post-MMA	**−44.76**		**−59.71**		**−8.02**		**+10.83%**		**+1.02 kg/m²**	

AHI apnea–hypopnea index (events/h), BMI body mass index (kg/m^2), *ESS* Epworth sleepiness scale, *LSAT* lowest oxygen saturation (%), *MMA* maxillomandibular advancement, *RDI* respiratory disturbance index (events/h), *SD* standard deviation

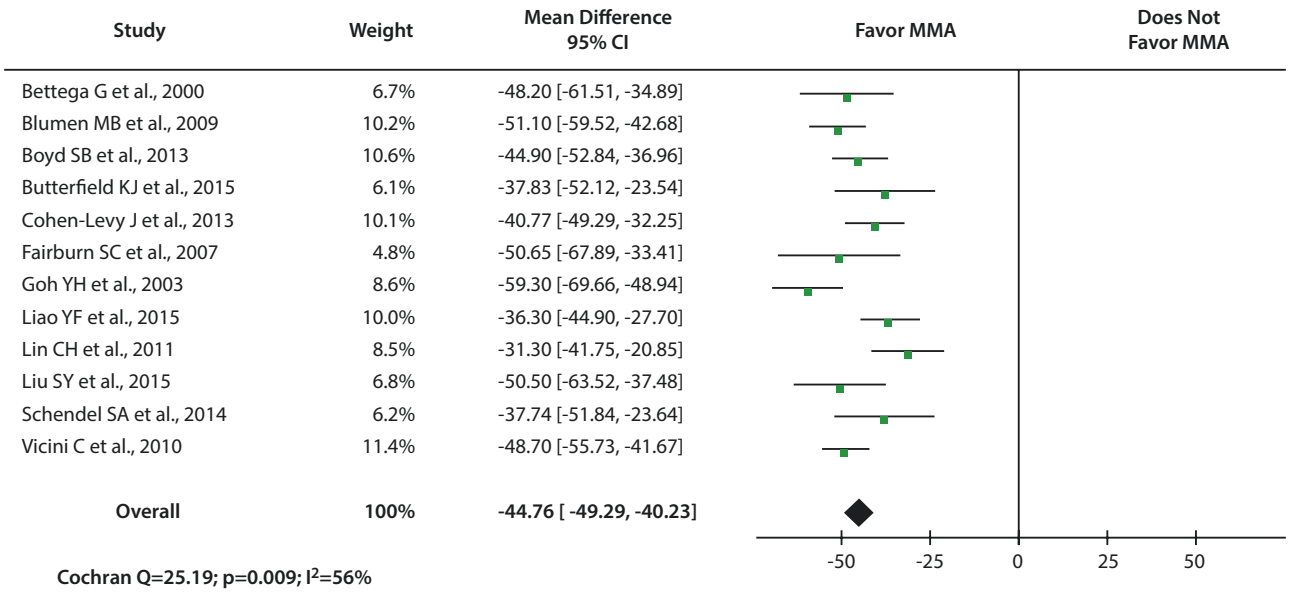

Fig. 28.2 Forest plot for AHI. (John et al. (2018) [129])

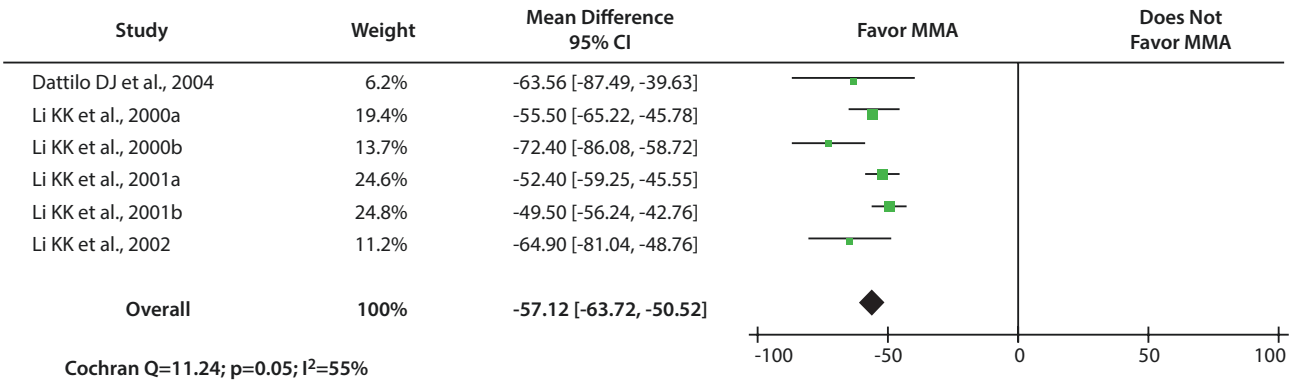

Fig. 28.3 Forest plot for RDI. (John et al. (2018) [129])

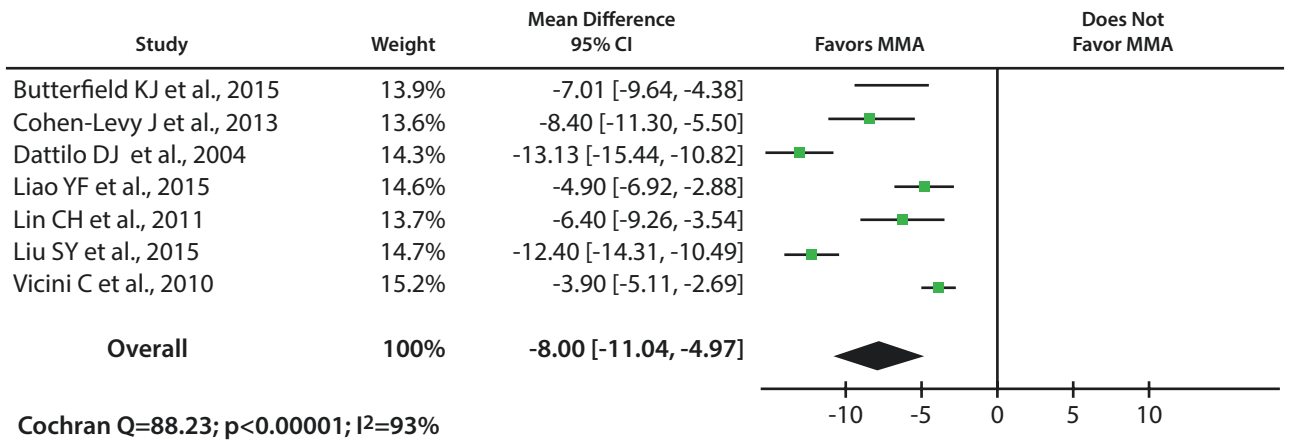

Fig. 28.4 Forest plot for Epworth Sleepiness Scale (ESS). (John et al. (2018) [129])

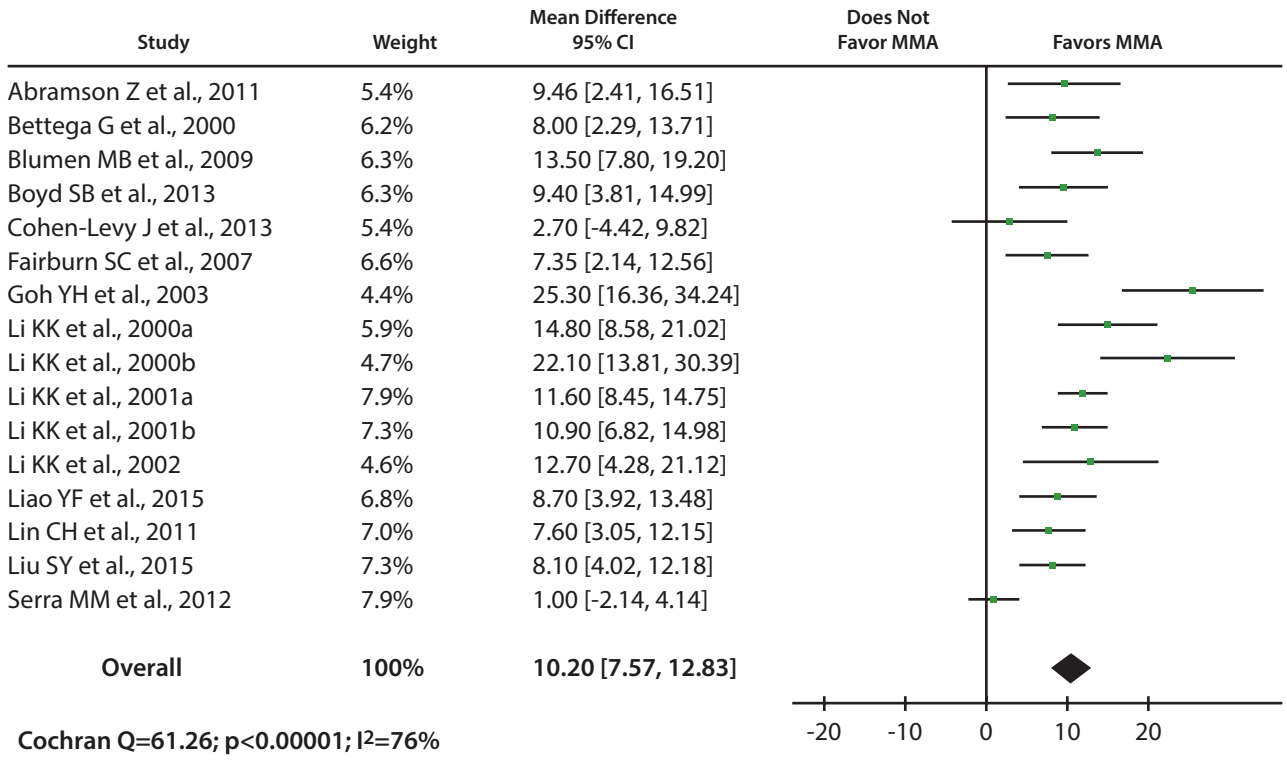

Fig. 28.5 Forest plot for lowest oxygen saturation (LSAT). (John et al. (2018) [129])

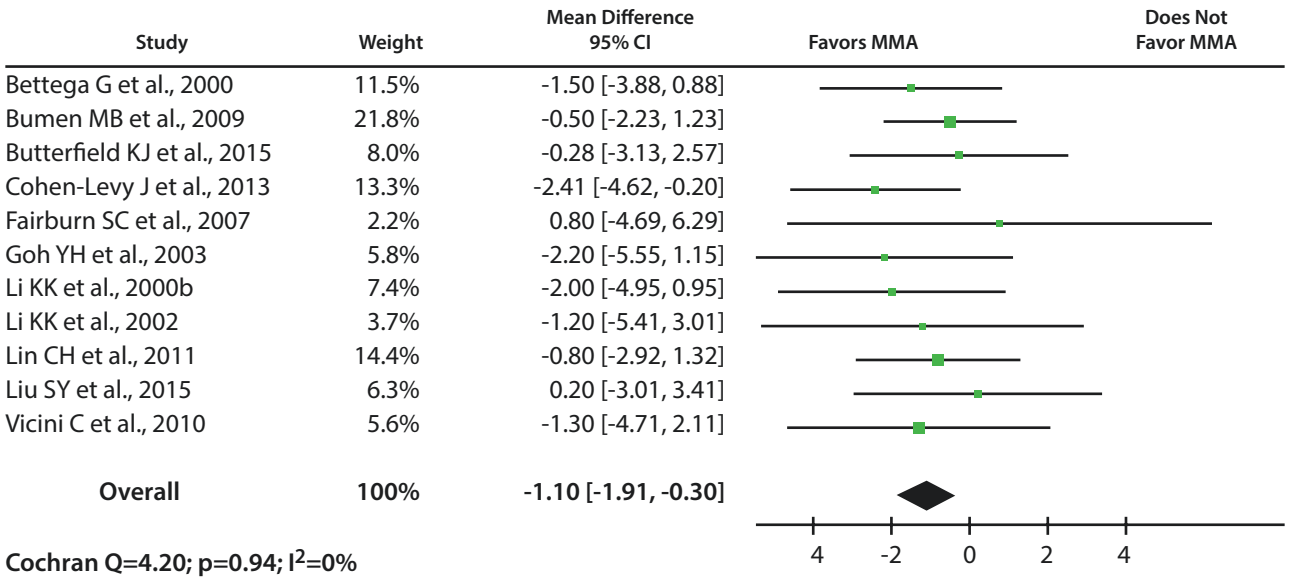

Fig. 28.6 Forest plot for body mass index (BMI). (John et al. (2018) [129])

28.3.1 Counterclockwise Rotation, Genioplasty, and Other Interventional Options

While various operative modifications have been proposed to minimize aesthetic disharmony post-MMA, counterclockwise rotation of the maxillomandibular complex and alteration to the occlusal plane is likely the best mitigating measure in this regard. It is always important to discuss the potential risks with patients in terms of potentially unfavorable aesthetic outcomes. Li et al. emphasized the importance of communication

Table 28.3 Risk of treatment failure (Vigneron et al., 2017 [143])

Risk parameter	Odds ratio	95% CI
Preoperative BMI >24.8 vs. <24.8 kg/m^2	14.00	1.43; 137.32
Preoperative age >45.01 vs. <45.01 years	14.00	1.43; 137.32
Gender Men vs. women	33.33	2.83; 392.60
Preoperative AHI >44.5 vs <44.5 events/hour	6.25	1.03; 38.08
Preoperative SNB >75% vs <75%	14.17	1.83; 109.86
Maxillary advancement >11 vs <11 mm	11.00	1.06; 114.09
Postoperative MRBL >8 vs <8 mm	6.25	1.03; 38.08

BMI body mass index, *AHI* apnea-hypopnea index, *SNB* Sella-nasion-sub chin point angle measured on a cephalogram, *MRBL* minimal retrobasilingual distance measured on a cephalogram

with all patients undergoing MMA, but were keen to note that there are patient subgroups who appear to be at greater risk of being dissatisfied with the aesthetic outcome: younger patients, patients with pre-existing bimaxillary protrusion, and nonobese patients [145]. However, employing counterclockwise rotation in lieu of a full-forward 10 mm movement has provided surgeons a viable tool to negotiate these hurdles, even in these patient populations.

By dropping the occlusal plane in a counterclockwise fashion to 8 degrees or less, a surgeon may not only achieve better airway, but also prevent a negative aesthetic outcome, such as upward tip of the nose and/or a highly protrusive looking upper and/or lower jaw [80, 146]. The aesthetic risk of a full, direct advancement of the upper and lower jaw may result in an excessively increased nasolabial angle and the patient's nares may become more visible in the frontal view and lips may appear more highly protrusive. In the profile view, excessive prognathism of the entire maxillomandibular complex could cause the nose to tip up and the jaws to be unaesthetically protrusive. The benefits of counterclockwise rotation are especially advantageous in some ethnic populations, such as Asians, who have more concave facial profiles [147].

To lessen or minimize advancement, other options could be explored in select cases, such as expanding the transverse dimensions of the dental arch to allow the tongue to extend superiorly toward the palate. Another option is to perform a concomitant transoral base-of-tongue resection to achieve the same degree of airway expansion, though it is important to note the caveats to this approach can potentially prolong recovery and the patient will need to remain intubated for up to 36 hours.

28.4 The Patient Evaluation Prior to MMA

28.4.1 Preoperative Medical Assessment

When a patient arrives to the office, a thorough comprehensive evaluation of the head and neck is conducted for these patients prior to planning of the surgery and its preparation. After the initial arrival of a patient to the office, a cone-beam computed tomography (CBCT) is performed. A nasal obstruction screener NOSE scale is filled out by the patient. From the CBCT scan, numerous radiographs are extracted, including the lateral cephalogram, assessment of the nasal sinuses, and volumetric analyses (including minimal cross-sectional evaluation, which is the most relevant 3D evaluation of the airway), and a panoramic radiograph should be performed to evaluate dentition. In addition, a nasal evaluation is performed to ascertain septal morphology, including turbinate morphology. Clinically, the patient's nares are visualized using a nasal speculum. A fiberoptic evaluation of nasal tissue, hypopharyngeal, and lateral pharyngeal wall collapse is performed during drug-induced sleep endoscopy (DISE). This will also allow assessment of the base of the tongue and correlate with CBCT evaluation. Sinuses are screened for any pathologic abnormalities. Intra-orally, the shape of the arch, crowding of the dentition, and width of the arch are noted and evaluated.

Clinical manipulation of the upper lip tissue is also performed to gain an understanding of maximal advancement that can be achieved and to determine the potential effects of this on the nasolabial angle. The patient's myofascial tissue is palpated due to the fact that excessive advancement in the maxilla could potentially have a negative effect on nasal structure and extensive lip protrusion. In case the situation is considered to be a problem for already-orthognathic patients, future need for rhinoplasty should be discussed with the patient. Any areas of tenderness are noted on a scale of 0–10. Presence of headaches/migraines, severity, and site are also noted. A very important anatomical landmark that is often ignored in MMA candidates is the temporomandibular joint (TMJ). TMJs are the foundation for jaw position, occlusion, facial balance, jaw function, growth and development, and airway function. TMJ pathology can adversely affect any of these factors.

Because MMA movements are significant, a thorough evaluation of TMJ stability is paramount to assure future stability. For a patient symptomatic in the TMJ region and having tinnitus and earache, a thorough evaluation of the bony and soft tissue structures of the TMJ are deemed necessary. MRI of the TMJ can allow visualization of the articular disc. It is important to note that for a subsection of patients diagnosed with weak and arthritic joints who require significant MMA/counterclockwise rotation, a combination surgery with total joint prosthesis is recommended. This will allow the patient to have a long-lasting and stable position of the mandible to maintain the airway open and prevent future relapse.

28.4.2 Guidelines and Indications for MMA

The first indication for MMA is an accurate diagnosis of OSA based on polysomnography and AHI ≥15 events per hour. Early guidelines for OSA surgery are presented below, as proposed by Prinsell in 2000 [148]:

1. **Surgical prerequisites.**
   (a) Clinically significant OSAS (AHI >15 or AI >5, LSAT <90%, and EDS)
   (b) Conservative treatments (e.g., CPAP) nonapplicable/unsuccessful/nontolerable
   (c) Medically/psychologically stable
   (d) Willing to proceed with surgery (i.e., informed consent)
2. **For specific site(s)/segmental area(s) that are distinctly identifiable**
   (a) Treat with appropriate procedures that address these specific sites
   (b) If a staged approach is recommended/desired, treat the most severe/critical site/area first
3. **For diffusely complex or multiple sites that are not readily distinguishable.**
   (a) Skeletal advancement procedures first to enlarge/stabilize the pharyngeal airway
      (i) Primary single-stage definitive treatment; or
      (ii) Minimize risk of postoperative edema-induced airway embarrassment associated with subsequent pharyngeal surgery
   (b) Pharyngeal soft tissue procedures second, if still necessary, for clinically significant residual OSAs

It should be noted that these guidelines do not apply to all patients, their expectations, quality of life, and their ability to adhere to CPAP therapy. For certain patients, CPAP is not an option because of claustrophobia and other reasons for nonadherence. Ultimately, for these patients, surgery is the ideal solution, but it must be tailored to each specific patient. Indications for MMA include patients who have severe OSA without significant pharyngeal tissue redundancy, patients with significant maxillomandibular deficiency, young patients who require long-term resolution of OSA, and patients who desire the most effective single-stage surgery [142]. Young patients who are in good clinical health and cardiovascular status are the best candidates for this procedure. Patients who have multiple comorbidities require proper optimization and need to undergo thorough risk assessment prior to surgery. However, it has been proposed that oxygen desaturation and morbid obesity are valid secondary factors that could indicate MMA intervention.

The risk of blood loss and need for hypotensive anesthesia place a significant amount of pressure on the body. Multiple referrals for systemic evaluation are necessary for these patients with comorbidities. Even though the risks and complications of these cases are not high, communication is of utmost importance between the surgeon, anesthesiologist, and primary and secondary care physicians, to tailor the best intraoperative procedure plan and optimal post-procedure care for the patients.

Intraoperatively, use of arterial lines for expedient evaluation of arterial blood oxygen and cardiac function also allows the anesthesiologist better ability to control the mean arterial pressure while also monitoring kidney function via urine output. Lowering mean arterial pressure allows the team to minimize blood loss during surgery. Cardine, propofol infusion, and nitroglycerin may be used, among other modalities.

28.4.3 Contraindications for MMA Surgery

MMA may not be the surgery of choice for certain subsets of patients who have numerous comorbidities (also known as multimorbidity) and are at high risk for having the procedure. While the complexity of the case varies from patient to patient, MMA can last anywhere between 4 and 7 hours. Procedure duration should be weighed against the patient's medical history and physiological reserve.

While many patients who suffer from OSA may already have compromised cardiac and multiorgan deficits due to OSA pathology and high levels of cortisol from poor sleep hygiene, these sequelae must be weighed and considered for each individual patient to determine recovery resilience. In elderly patients (older than 65 years of age), it must be noted that longer anesthesia times could cause postoperative amnesia that could last for 3–4 days. Again, a comprehensive anesthesia history can help predict many of these types of events and have a plan in place.

Patients taking anticoagulants are at high risk for excessive hemorrhage and, in the case of a LeFort I osteotomy, they are particularly at high risk for epistaxis, which could potentially compromise the airway. The airway of morbidly obese patients is at higher risk for complications and collapse, and in cases may be contraindicated. Patients with poor kidney function are typically not candidates for the procedure because of the higher risk of anesthetic complications and further renal damage. Heavy smokers are also at high risk for necrosis following LeFort I osteotomy, particularly if it is a segmented procedure, which can have a high risk of flap failure. Smokers are also at high risk for treatment failure in terms of risk of segment vascularity and healing, as well as to achieve successful intraoperative pulmonary function; therefore, preoperative smoking cessation is mandated. Diabetic patients with HBA1c >7 are at higher risk for myocardial infarction (MI), stroke, and postoperative infections. These patients would need to be maintained on antibiotics significantly longer; therefore, their procedures should be delayed until HBA1c is well controlled.

28.5 Procedure

Orthognathic surgery has been used for decades to correct dentofacial deformities. The number of orthognathic procedures had somewhat declined in the 1980s and 1990s, but its practice has been revitalized in the past two decades due its modification to MMA and its resultant surgical successes, particularly in OSA patients, but also in temporomandibular disorder (TMD) patients requiring temporomandibular joint reconstruction (TMJR), some of whom also have OSA.

28.5.1 Sagittal and LeFort I Osteotomies

28.5.1.1 Advancement Limitations of Sagittal Osteotomy

Originally pioneered by Trauner and Obwegeser in 1957 [149] (after its initial introduction by Schuchardt in 1942 [150]), their modified bilateral sagittal split osteotomy (BSSO) revolutionized maxillofacial surgery by introducing it as a safe and standardized procedure that is still performed globally with few modifications from their described approach. Their modification of Schuchardt's technique consisted of widening the gap between horizontal cuts to 25 mm to accommodate the inferior alveolar nerve (IAN) by connecting two horizontal cortical cuts on the lateral oblique ridge (avoiding the ramus's posterior border), and then the chiseling of the lateral cortex would lead to fracturing the ramus.

This procedure allows the surgeon to correct most asymmetries and allows jaw function immediately postoperatively. In addition, the rigid fixation achieved by securing plates would allow for healing. This is due to the heavy interface of bony contact of the proximal and distal segments. Before the application of plates for rigid fixation, positional screws were used to stabilize the proximal and distal segments. However, use of plates has allowed surgeons to bypass extra-oral stab incisions that are traditionally used to introduce bicortical screws. In our practice, we place screws and position plates intraorally.

Modifications of the sagittal split were then proposed by Dal Pont in 1961 and Hunsuck in 1968. In 1977, a seminal article by Bell and Schendel was published exploring the biologic basis for modifying the sagittal ramus split. This informed Epker's new modification, who published his paper later that same year. In 1987, Wolford, Bennett, and Rafferty [151] introduced important modifications of the sagittal split osteotomy of the mandibular ramus, which allowed for more controlled segment splits and greater control of the proximal segment. Later in 1990, Wolford and Davis [152] introduced the inferior border split for the mandible, which employed a new customized saw specially designed for inferior border cuts with the aim of achieving a low lingual split while also preserving the IAN. This was an important advancement because it reduced the likelihood that the IAN would be found in the proximal segment where it is more susceptible to procedural trauma.

For the maxillary step osteotomy, Bennett and Wolford [153] described their improvement in 1985. Traditional Le Fort I maxillary osteotomy is inclined anteroposteriorly. The maxillary step osteotomy's primary advantage allows for "pure" anteroposterior movement by obviating incline effects of the traditional technique.

28.5.1.2 Plates Vs. Screws

The two most common fixation devices used for BSSO are either a monocortical miniplate or bicortical titanium screws (See ◘ Fig. 28.7). A 2016 systematic review and meta-analysis by Al-Moraissi and Al-Hendi [154] found no significant difference in postoperative stability when comparing use of a monocortical plate ($n = 67$) or bicortical screws ($n = 60$) pooled from three studies for fixation post-BSSO. Bicortical screws may be useful for cases that require more than 6 mm of advancement to achieve postoperative stability and plate fixation is useful to stabilize fragments in cases where there is not enough bone overlap between the proximal and distal segments for bicortical screw placement. Use of mandibular locking plates can allow the head of the screws to be fully stabilized to the plate, which can prevent future loosening and infection.

These plates are bent to a passive fit of the bony structure. The combination of the passive fit and lock-

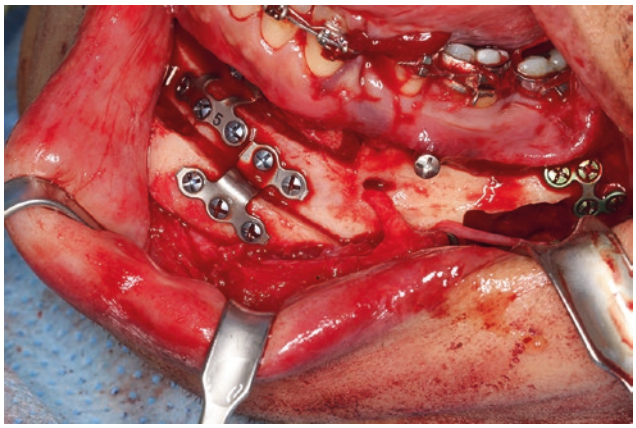

Fig. 28.7 Fixation achieved with implanted L-plates and Z-plates on a sagittal split

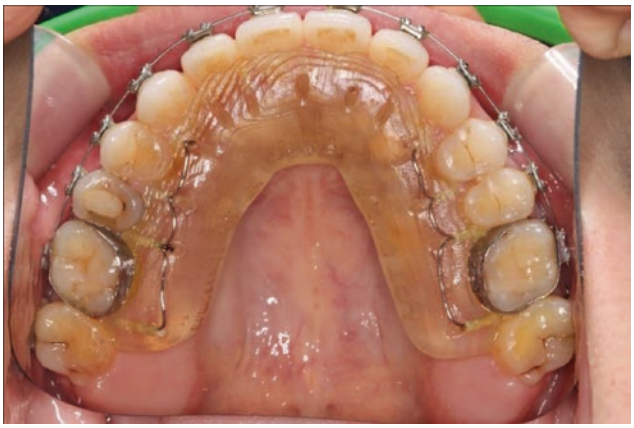

Fig. 28.8 After segmental osteotomy and maxillary expansion, a palatal splint is inserted and stabilized with interdental wire fixation. This splint will remain in the palate for 3–4 months post-surgery

ing screws prevents future hardware failure and infection. After fixation of the bilateral sagittal split and use of intermediate splints, incision lines are irrigated and incisions are closed. Next, a LeFort I osteotomy is completed, and segmental osteotomies are performed with a piezosurgery unit using prefabricated splints.

When necessary, it is important to employ maxillary expansion to achieve greater width and proper occlusion. This can be achieved by segmental osteotomy or, prior to MMA, with surgically assisted rapid palatal expansion (SARPE). Skeletal stability of SARPE for large expansions has been shown to be modest but stable, though operators should expect late loss of roughly one-third of the expansion [155, 156]. Based on clinician experience, either route may be taken, either SARPE performed prior to MMA or expansion during the advancement procedure, effectively consolidating both interventions in a single procedure. To ensure stability of the segmental osteotomy, a palatal splint is fixated to the maxilla with interdental wires to serve as a cast-like mechanism throughout bone healing and removed after 3–4 months (Fig. 28.8).

Throughout LeFort I osteotomy, the anesthesia team is notified to maintain the mean arterial pressure at 50 mm Hg. Any necessary modifications to the septum and turbinates are performed during this portion of the procedure with caution to not remove excess turbinate tissue and ensuring reduction of one-half to two-thirds of the turbinate at maximum [157].

In continuum, paramedian cuts are performed for segmental surgery. Grafting of the maxilla is important when rigidly fixating. The grafting in the anterior aspect of the maxilla will facilitate long-term stability of maxillary movement, which is particularly important in patients with extensive counterclockwise rotation of the occlusal plane. Lastly, if indicated, a genioglossal advancement with proper rigid fixation is performed. This advancement can be shaped to meet the aesthetic requirements of the case as the advanced bone can be modified by reduction.

28.6 Total Joint/TMJ Considerations in Advancing the Mandible

28.6.1 TMJ Pathology and Indications for Surgery

The TMJ is an anatomic centerpiece that determines jaw position and function, occlusion, growth and development, facial balance, and airway function. The pathology of the TMJ can adversely affect any of these factors. A range of TMJ pathology has been documented, including CT disorder and various forms of arthritis, disc displacement, ankylosis (or trauma resulting in ankylosis), symptomatic TMJ, facial asymmetry (which may be caused by a TMJ tumor), and agenesis, all of which may be indications for the procedure. The diagnosis and treatment planning for TMJ pathology and dentofacial deformities consist of the usual steps of a complete clinical evaluation consisting of a medical history, dental model analysis, and CBCT and MRI scans. For TMJ, MRI is a very important tool in diagnostic and treatment planning by evaluating both hard and soft tissues. It has been estimated that approximately 40%–60% of TMJ MRIs are improperly read. TMJ surgery may be needed for a majority of OSA patients with dentofacial deformities and should be performed only when necessary. In 2008, Goncalves et al. [158] described the relationship between joint pathology, disc displacement, and relapse.

In modern day practice, a combination of both TMJ surgery and orthognathic surgery has proven to be successful with long-term follow-up [159]. A more accurate

result has been made possible by the digital workflow that has been refined over the past decade. This signifies the importance of evaluating the joint prior to embarking upon MMA.

28.7 Post-MMA Follow-Up Care

The majority of patients are extubated after surgery when criteria are met. When extubated, the patient is transferred to the postanesthesia care unit (PACU). During this portion of the recovery, the patient has bilateral nasal trumpets in place to maintain a patent airway. When vital signs and pain control meet criteria, the patient is transferred to the intensive care unit (ICU) for one-on-one monitoring by a nurse. The head-of-bed will be maintained at 45 degrees and ice will be applied for the following 3 days postsurgery. A humidified oxygen tent is provided for the patient and pulse oximetry is continually monitored. The day after surgery, the Foley catheter and arterial lines are discontinued, and the patient is transferred to a transitional care unit. While in the transitional care unit, goals are set to meet ambulation needs, general and oral pain control, and fluid and protein intake. Nutrition and physical therapy consults are initiated. Oral hygiene is maintained to prevent infection. Intravenous antibiotics, steroids, and antiemetics are used as necessary. On average, patients are discharged 2–3 days post-MMA and followed in private practice 2–3 times a week for the first 2–3 weeks to help the patient change dental elastics as well as to monitor intraoral and extraoral wounds.

At week 3, physical therapy is initiated to allow the patient to achieve muscle release good incisal opening. Postsurgical orthodontics is initiated at 6 weeks after surgery. After the initial month, the patient is then seen at the 3- to 4-month mark to remove the palatal splint (if a segmental osteotomy is performed). These timelines may vary from patient to patient. Most patients are recommended to take 3–4 weeks off work for acceptable recovery, though this could be shortened or extended due to the patient's physiologic status and healing progress.

28.7.1 Relapse and Risks Leading to Reoperation

As noted above, a relapse of OSA could be attributable to postoperative weight gain and the natural history of other comorbidities and changes in medication [160]. However, various other problems could lead to instability, such as condylar resorption and its related skeletal instability, implant device failure, and postoperative trauma. However, relapse may occur and defy explanation. However, several structural factors have been associated with relapse, including changes attributable to any excess bone movement and/or rotation, changes of the teeth, and any unresolved malocclusion during preprocedure orthodontic treatment, any change in position of the condyles, and any significant changes in ramus inclination and the mandibular plane [161, 162].

28.7.2 Case 1: 38-Year-Old Female Intolerant of CPAP

A 38-year-old female referred by a pulmonologist presented with a history of OSA and a prior turbinectomy and was not taking any medications at the time. The patient was subsequently diagnosed with OSA and referred to my practice. The patient had been treated with CPAP since 2011 but reported it to be intolerable and became nonadherent. After thorough patient evaluation and discussion of risks and benefits, the patient opted for MMA and began presurgical orthodontics. MMA was performed with counterclockwise rotation. The patient began orthodontic treatment 5 weeks after surgery. AHI before (2013 sleep study) and after (2015 postoperative study) was 53 and 0.3, respectively. Her sleep efficiency improved to 91% with a lowest O_2 sat of 92%. Upon 4-year follow-up, the patient reported no significant evidence of OSA (See ◘ Figs. 28.9, 28.10, 28.11, and 28.12).

28.7.3 Case 2: 59-Year-Old Female with Scleroderma

A 59-year-old female presented to my practice with a history of scleroderma and multiple TMJ procedures since her first in the early 1990s (hypoplastic maxilla and mandible), as well as TMJ arthroscopy, discectomy, and placement of silicone spacers and bilateral rib grafts in the late 1990s. She had been on oxygen via nasal cannula since 1993 and had been reportedly adherent in her use of CPAP for sleep. CBCT or cephalometric imaging of TMJs revealed ankylosis of joints with floating bone particles in both TMJ fossae. Her maximal incisal opening was only 5 mm. A sleep study was performed in 2016 and revealed an AHI of 28 and confirmed the diagnosis of OSA. The patient opted for MMA with simultaneous TMJR with new TMJ prostheses. Presurgical preparation began with orthodontics. The 2016 procedure was a planned reconstruction with total joint prosthesis and MMA. The rib harvest that was previously completed in 1999 was removed and the maxilla was grafted at the time of LeFort I

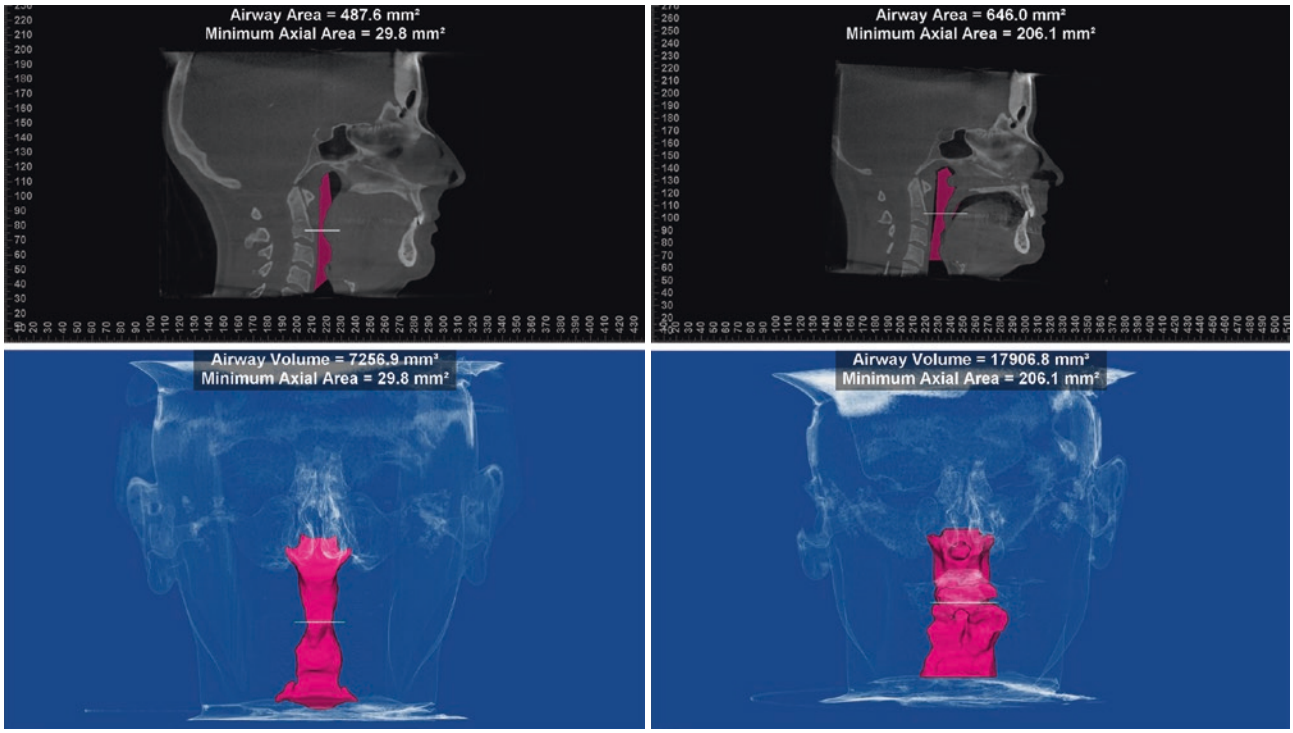

Fig. 28.9 Dolphin airway imaging showed minimum axial area improvement from 29.8 mm^3 preoperatively to 206.1 mm^3 postoperatively

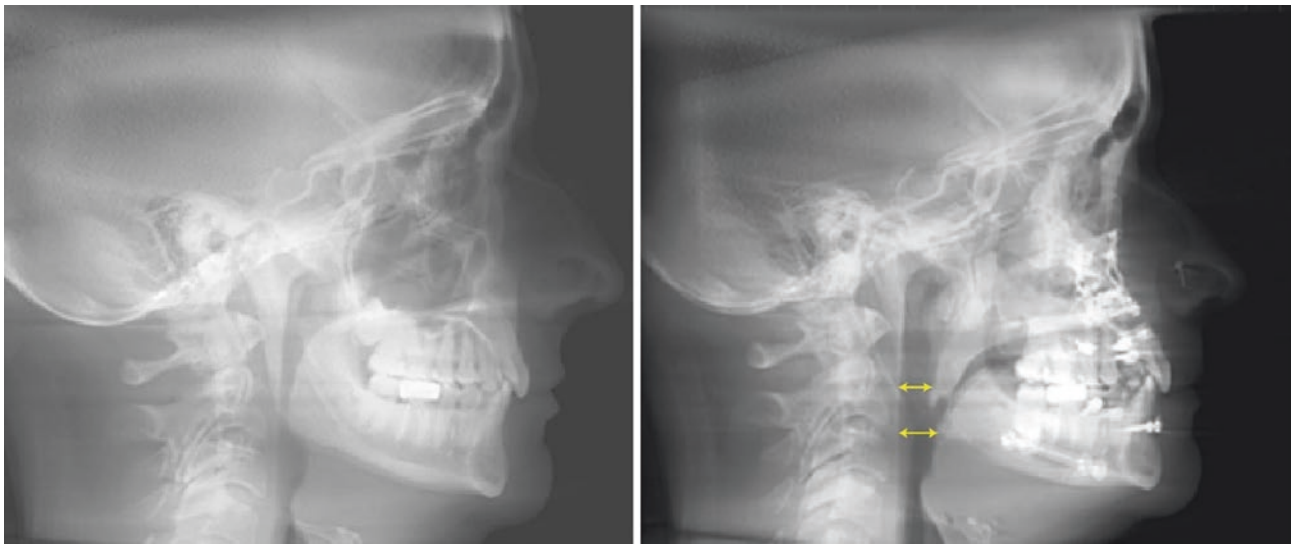

Fig. 28.10 Pre- and postoperative profile cephalometry comparison shows increased area of middle posterior airway space (MPAS) from 2 mm to 9 mm and an increased inferior posterior airway space (IPAS) from 3 mm to 11.5 mm

osteotomy in 2016. Postoperative follow-up was performed weekly, then bimonthly for 1 year. At 1-year follow-up, the airway minimum axial area improved from 88 mm^2 to 186 mm^2 (see Fig. 28.16 below) in the patient's pulmonary and renal function tests. The patient reported a decrease in polypharmacy, as medications decreased from 9 to 4. Postoperatively, the patient no longer required a nasal cannula or CPAP, and was evaluated to be ostensibly cured of OSA. The patient's function improved as her maximal incisal opening improved from 5 mm to 38 mm without pain (See Figs. 28.13, 28.14, 28.15, and 28.16).

Fig. 28.11 Pre- and postoperative profile photographs (profile and oblique) show improved craniofacial harmony and disappearance of the dorsal hump of the nose

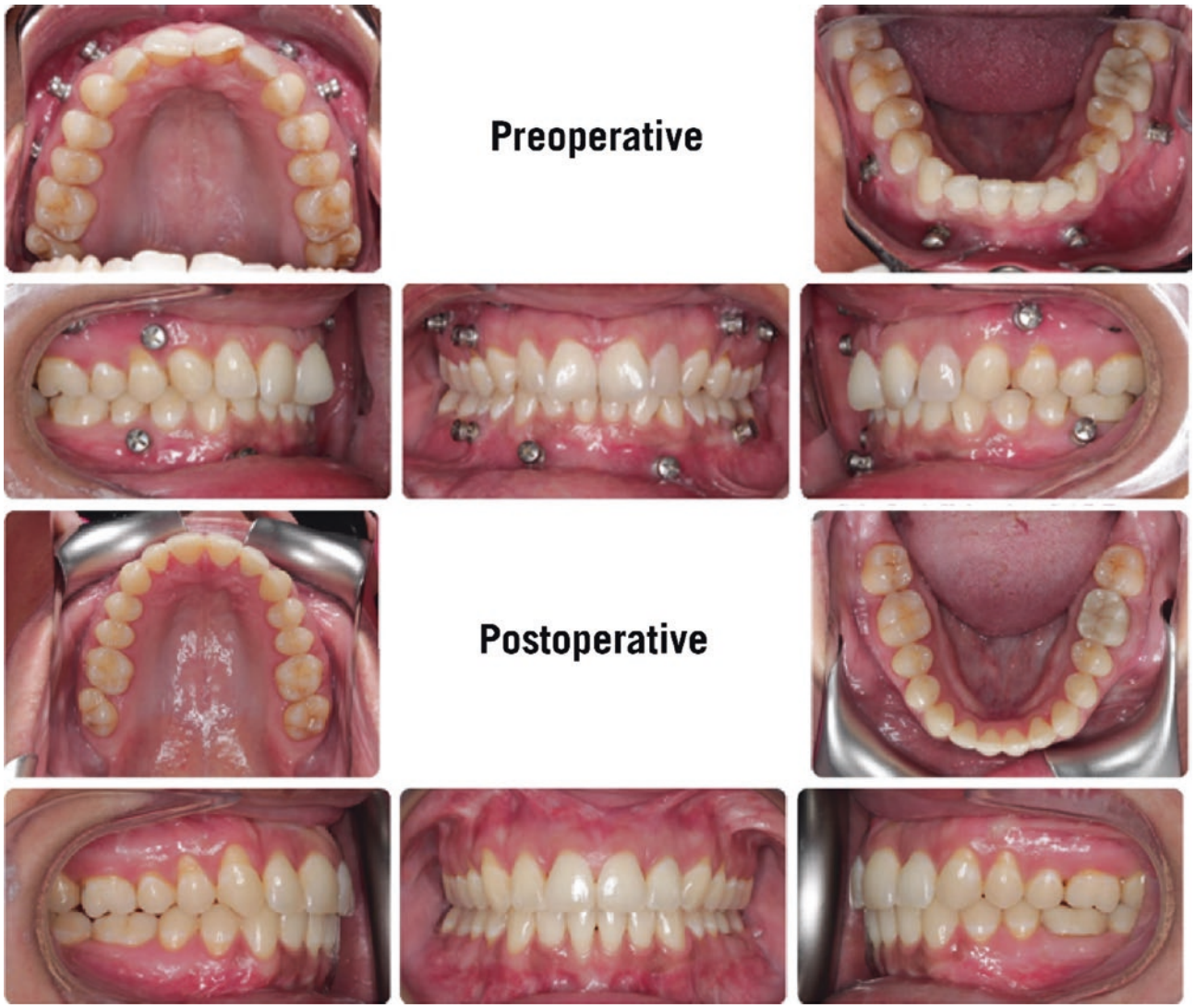

Fig. 28.12 Pre- and postoperative dental photographs show improved dental alignment, improved arch, preservation of occlusion, and satisfactory esthetic outcome

28.7.4 Case 3: Severe OSA and Pronounced Retrognathia

A 52-year-old male presented to our practice with severe OSA and pronounced retrognathia, hypertension, and polypharmacy. The patient was on CPAP, which was ineffective to manage his OSA. A prior sleep study showed an AHI of 57. After obtaining CBCT and airway analysis prior to DISE, it was appreciated that he had an above-average airway, which was likely collapsible. MMA was cautiously recommended as a solution since his airway did not radiographically appear constricted upon 3D analysis. The other challenge that was met during his case was the mechanics of MMA movement due to excessive proclination of his lower incisors. To solve this problem, subapico osteotomy and extraction of the first lower premolars was recommended to facilitate further mandibular advancement. The subapico osteotomy setback also allowed us to resolve the extensive of curve of spee. Following MMA, the patient's maxilla was advanced by 7 mm and pogonion by 22 mm. The maxilla was expanded by 12 mm to meet the demands of the uprighted posterior teeth. The pterygoid plates were advanced 11 mm on the left and 9 mm on the right. The patient's minimal cross-sectional area increased from 169 mm^2 to 308 mm^2. This volumetric increase impacted the fluid dynamics adequately to decrease the collapsibility of the soft tissue in his airway, subsequently curing him of OSA.

Fig. 28.13 Presurgical and postsurgical photographs

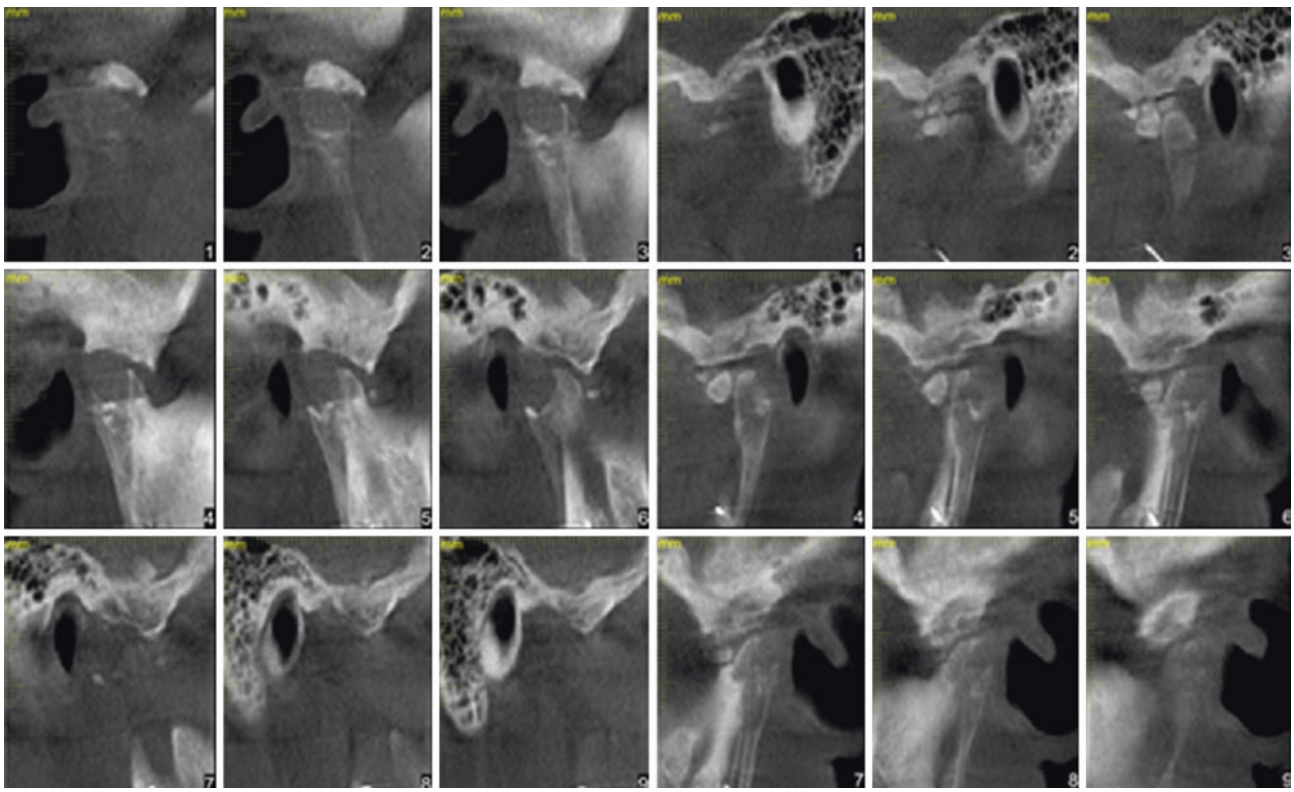

Fig. 28.14 Sagittal view of TMJs reveal ankylosis of joints with floating bone particles in the fossae. Maximal incisal opening was 5 mm

The case shows palatal expansion, which was performed to gain additional transverse width following presurgical uprighting of the lower teeth on the basal bone. (See Figs. 28.17, 28.18, 28.19, 28.20, and 28.21).

28.7.5 Case 4: High BMI and Severe OSA

A 54-year-old male with high BMI and severe OSA presented to our practice. The airway was diminished at the soft palate region. The patient was CPAP-intolerant and it was ineffective as a management modality. Because of the patient's high BMI, the patient was recommended to seek weight loss prior to surgery. This weight change did not decrease his AHI sufficiently due to the fact that the airway was still constricted at the soft palate region. No change in the minimal cross-sectional area was noticed.

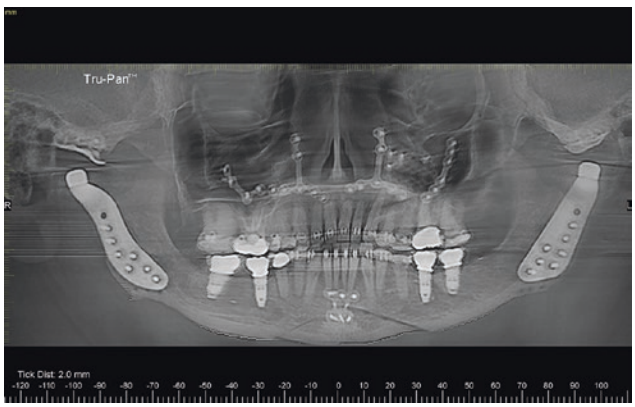

Fig. 28.15 Panoramic radiograph

After MMA, all signs and symptoms of his OSA was resolved and the patient was cured, and did not require further CPAP. The maxilla was advanced 10 mm and the pogonion was advanced 18 mm (while including the genioglossal advancement). The maxilla was expanded by 14 mm and the patient had his palatal splint removed at 4 months postoperatively. A counterclockwise rotation of 8 degrees was performed to increase the airway. The minimal cross-sectional area increased from 29 mm^2 to 340 mm^2. (See Figs. 28.22, 28.23, and 28.24).

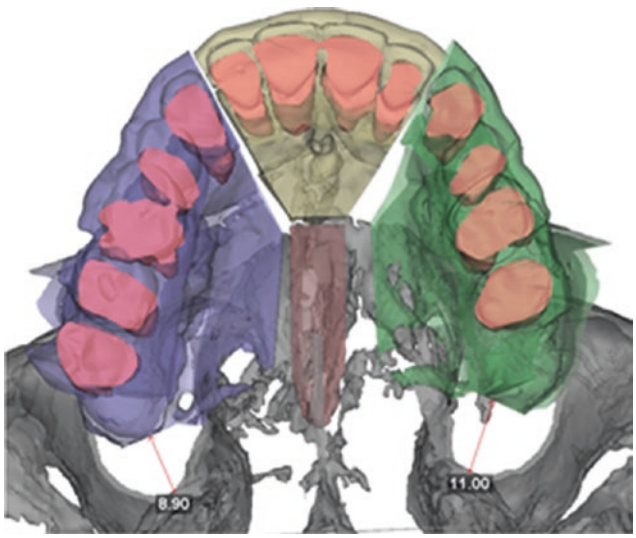

Fig. 28.17 Significant advancement was achieved at the pterygoid plates (11 mm on the left side and 9 mm on the right). A palatal splint was digitally fabricated and used to achieve stability of the transverse expansion

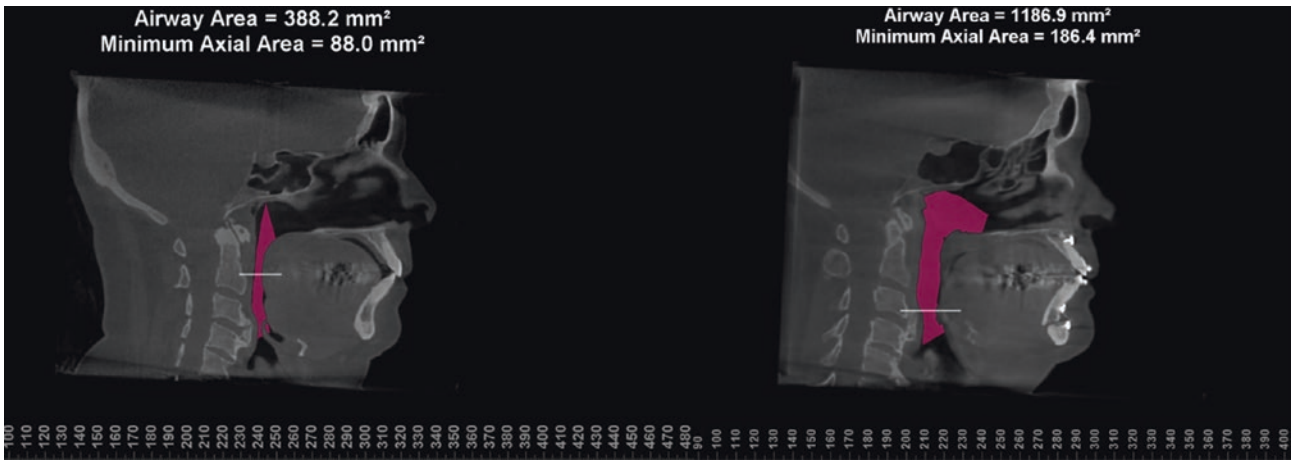

Fig. 28.16 Minimum axial area improved from 88 mm^2 to 186 mm^2

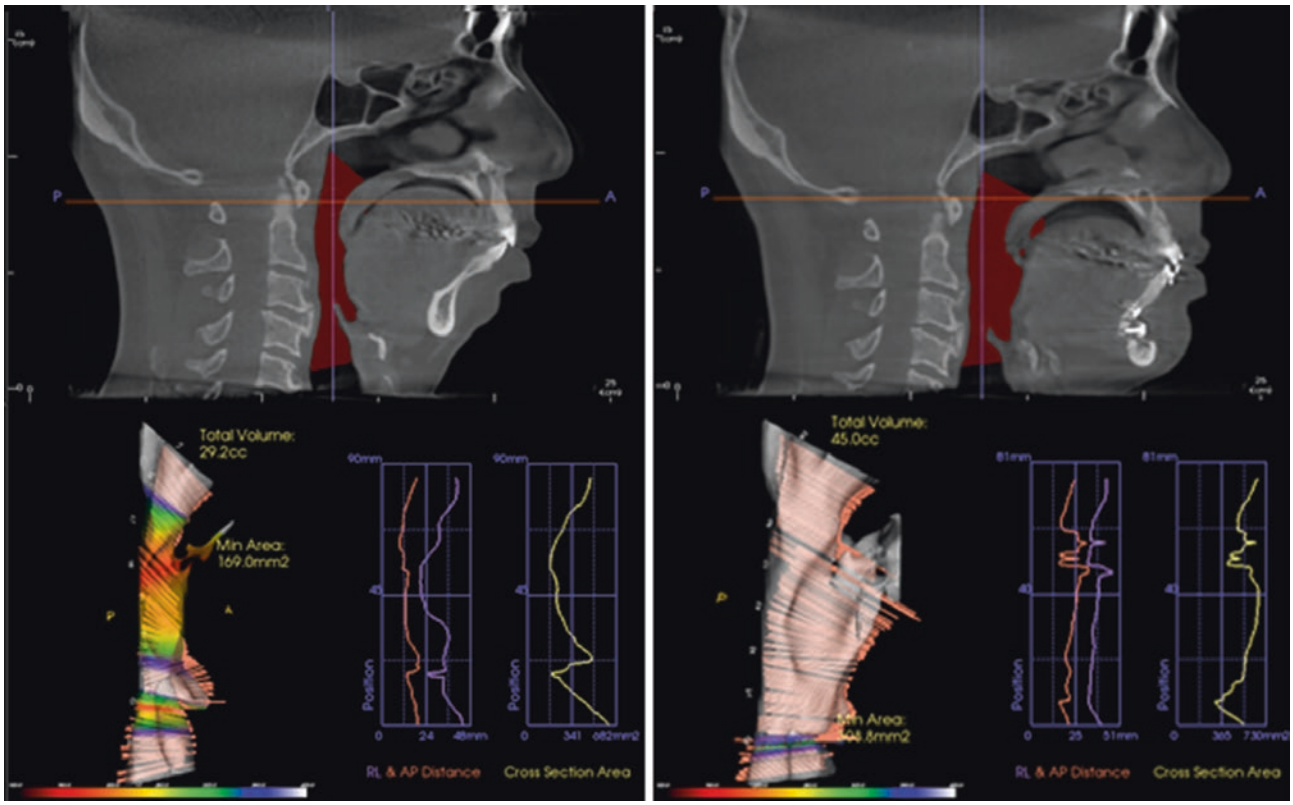

Fig. 28.18 Airway and volumetric improvement can be appreciated at all levels. MCA improved from 169 mm^2 to 308 mm^2

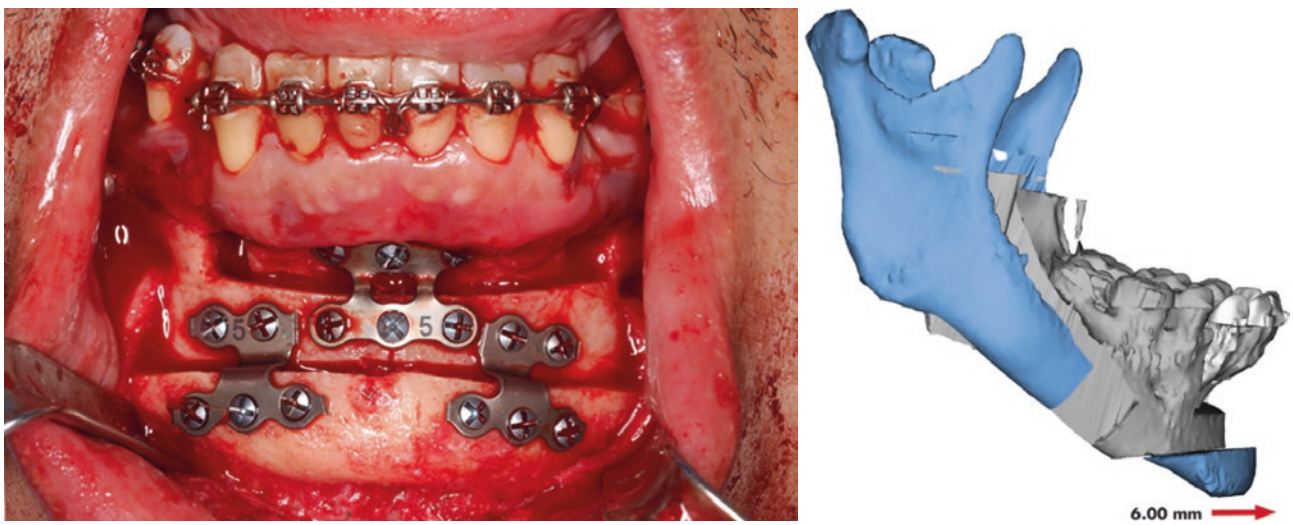

Fig. 28.19 Subapico osteotomy setback of the mandible was performed with rigid fixation with genioglossal advancement, which facilitated more forward advancement than was otherwise possible

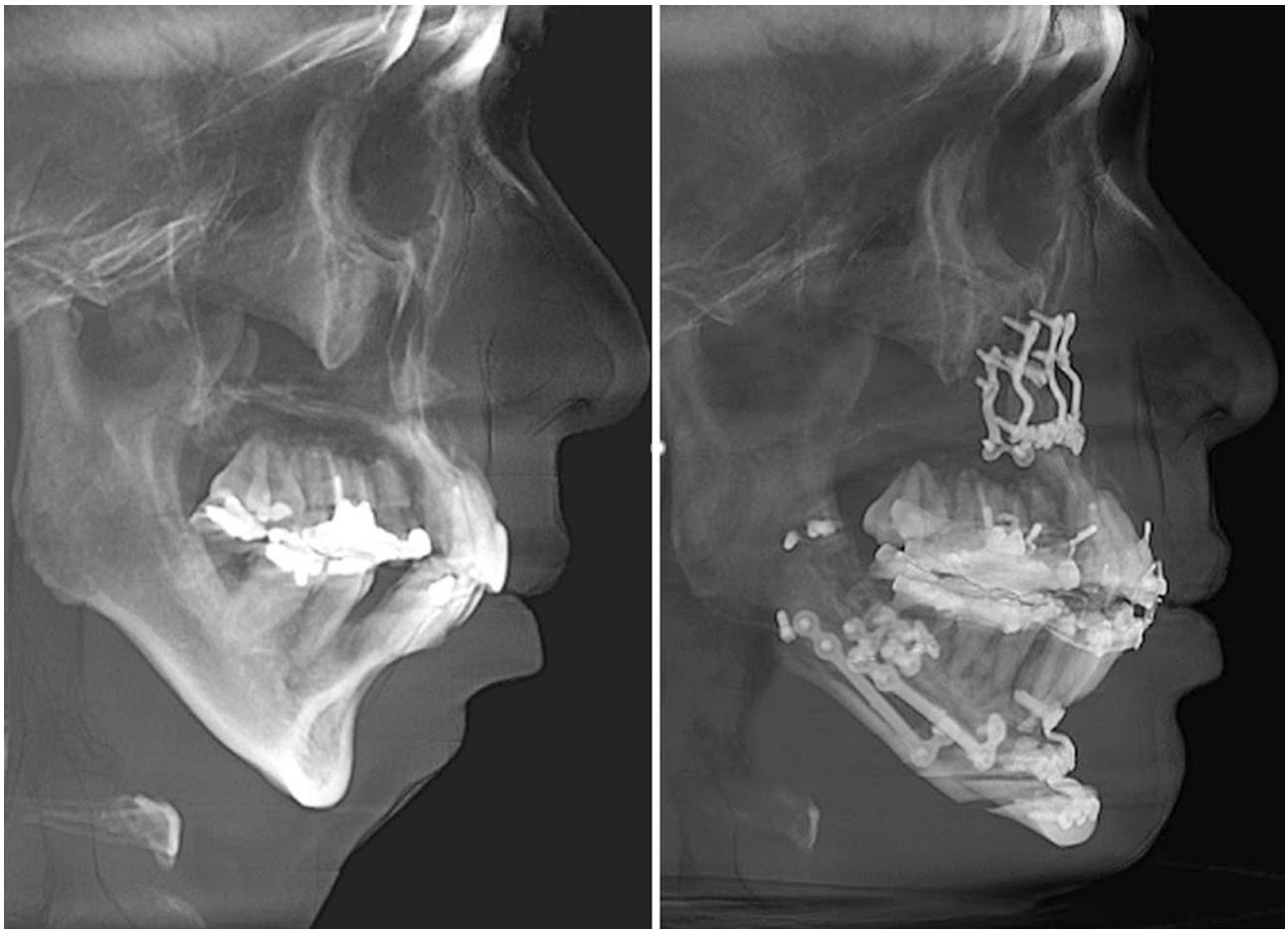

Fig. 28.20 Before and after lateral cepholemtric radiograph

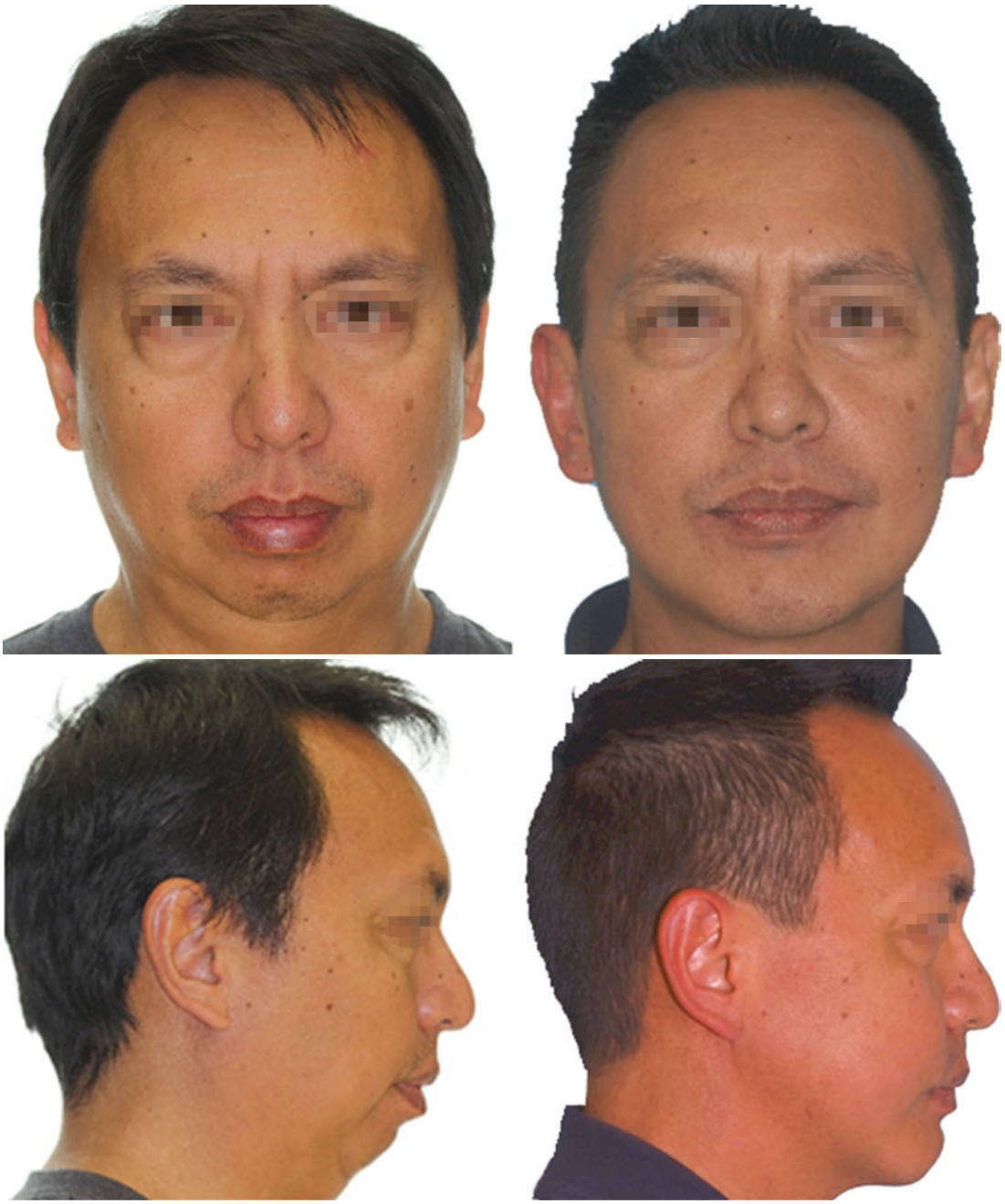

Fig. 28.21 Pre- and 1-year postoperative photographs show significantly improved facial harmony. The patient was cured of OSA

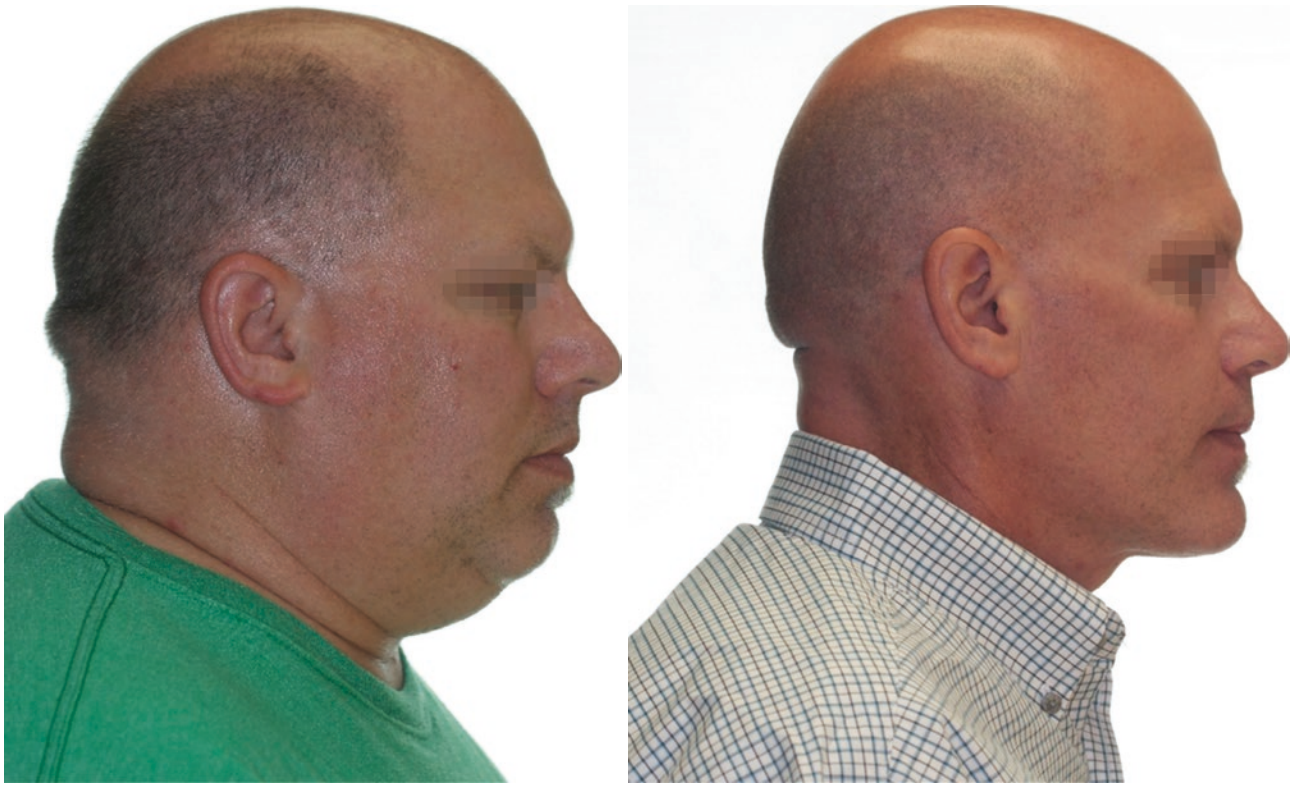

Fig. 28.22 Pre- and postoperative photographs

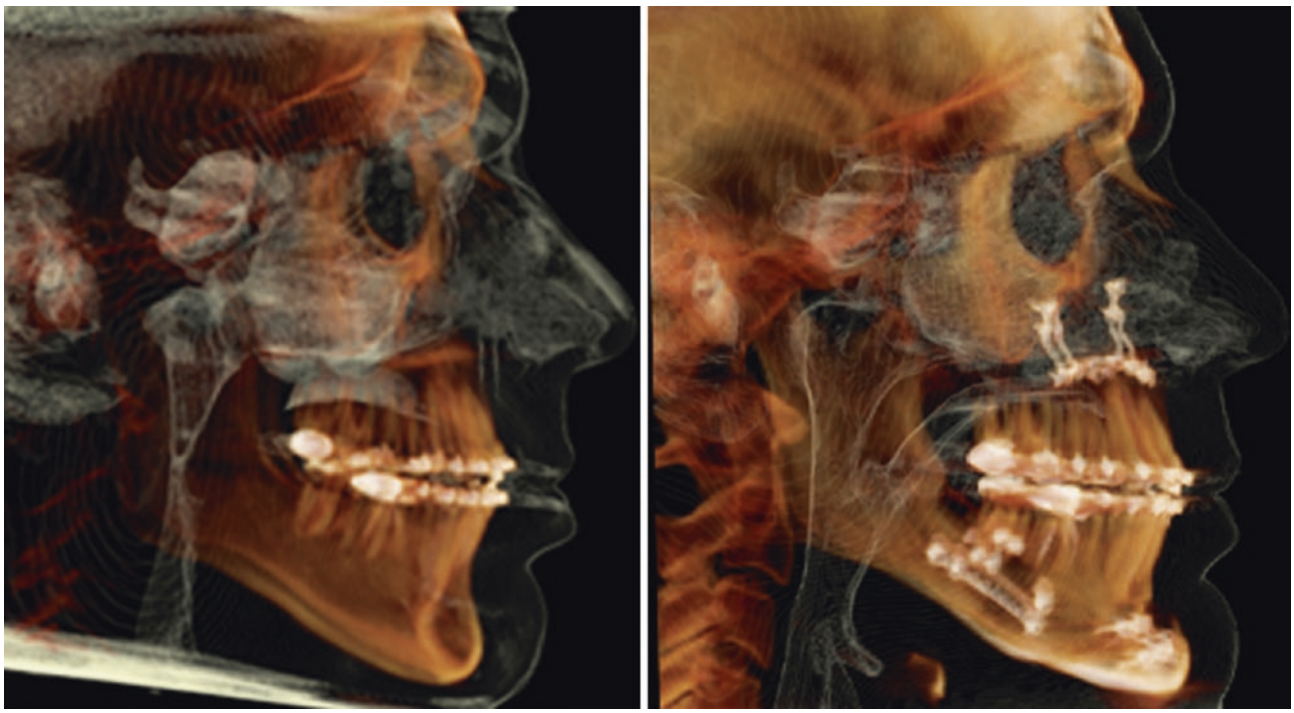

Fig. 28.23 Pre- and postoperative lateral cephalometric radiographs

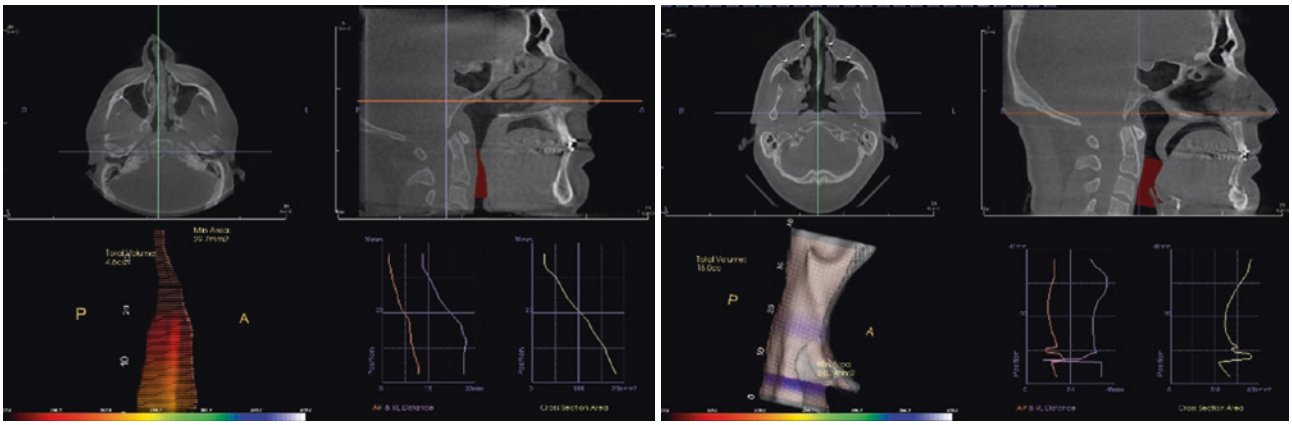

Fig. 28.24 Minimal cross-sectional area increased from 29 mm^2 to 340 mm^2

28.8 Conclusion

CPAP has long been considered the gold standard treatment for OSA and is often started first-line in concert with noninvasive lifestyle modifications, particularly for milder cases. The use of oral appliances has also increased since they are also relatively easy to use and noninvasive. However, surgical intervention may be the only effective way to treat OSA for many patients. Short of tracheostomy, MMA has become a definitive, permanent treatment for OSA. MMA as a definitive treatment for OSA is supported by an abundance of systematic reviews, meta-analyses, and cohort studies. However, more data are needed in the form of prospective randomized controlled trials and other research that can inform refinements of current practices and innovations. Further modifications of the MMA technique can optimize its employment, including streamlining of the virtual planning processes. Education and outreach of referring providers, potential candidate patients, former patients, and improving general awareness in communities will help render it a more acceptable and preferred treatment modality for OSA.

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Virtual Surgical Planning and Digital Workflow for Concomitant Temporomandibular Replacement and Maxillomandibular Advancement Surgery

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Contents

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Overview

Independently, both temporomandibular joint replacement (TMJR) and maxillomandibular advancement (MMA) are major surgeries. Many patients can benefit from both surgeries, but the choice as to whether both procedures are conducted simultaneously in a single surgical session (the one-stage approach) or in two separate stages in the phased approach can be challenging. Several factors must be weighed to determine the optimal approach for each individual patient, foremost of which is the joint patient–provider decision-making process, and surgeons must weigh several factors, including patient age, health status, resiliency, and the capabilities of tools in their surgical armamentarium.

The two-stage approach necessitates the patient to undergo two separate invasive surgeries with anesthesia, which can profoundly prolong recovery, introduces more elements of complication risk, and demands longer treatment, follow-up, and likely a significantly increased cost. A single-stage concomitant temporomandibular joint (TMJ) and orthognathic surgery (CTOS) requires a high degree of surgical expertise, skill, and proficiency to ensure optimal outcomes. The slightest malposition of hardware can impact the final clinical outcome. As such, these types of complex cases should only be performed by a surgeon experienced with orthognathic and TMJ procedures to achieve translational accuracy and a good treatment outcome.

However, the benefits to the patient from surgeon expertise and proficiency are wasted without maximizing the benefits of computer-assisted technology. Computer-aided tools can facilitate precise imaging measurement and provide opportunities for enhanced 2D and 3D visualization, including the use of simulation models, 3D-printed models, and adhering to a structured planning process. Success of CTOS not only hinges upon the robust, circumspect treatment planning offered by computer-aided surgical simulation (CASS) for CTOS but also depends on the surgeon's (and his or her staff's) operational familiarity, tech-savviness, and process mastery that such advanced technologies demand of their operators. This chapter reviews the literature supporting the use of virtual planning processes in orthognathic surgeries and presents a new treatment protocol for CASS application in CTOS cases that require reconstruction and specially fitted total joint prostheses. Also explored is how these processes compare with traditional protocols and reviews imaging and virtual planning accuracy facilitated by these state-of-the-art planning advancements.

29.1 Indications for TMJ, MMA, and CTOS: Traditional or Virtual Planning Approach?

TMJ disorders and dentofacial deformities commonly coexist. The TMJ disorders may be the causative factor of the jaw deformity and develop as a result of the jaw deformity, or the two entities may develop independently of each other. There are several common TMJ disorders that may adversely impact key orthognathic surgical outcomes and postoperative jaw position and occlusion. These disorders include (1) articular disc dislocation, (2) adolescent internal condylar resorption, (3) reactive arthritis, (4) condylar hyperplasia, (5) ankylosis, (6) congenital deformation or absence of the TMJ, (7) connective tissue and autoimmune diseases, (8) trauma, and (9) other end-stage TMJ disorders, all of which have been linked with dentofacial deformities, malocclusion, TMJ pain, headaches, myofascial pain, TMJ and jaw functional impairment, ear symptoms, and sleep apnea [1]. Patients with these conditions may be candidates for corrective surgery, including custom-fitted total joint prostheses for TMJ reconstruction TMJR, and orthognathic surgery, including MMA. The traditional model of CTOS treatment planning techniques, which has its own subset margin of error, may render the patient and their surgical outcome vulnerable to an undesirable result, and therefore, it requires substantial skill, experience, and expertise in delivering the most optimal clinical outcome. Traditional planning for bimaxillary surgery is not only time-consuming and laborious but has been criticized for being error-prone [2].

In the past 15 years, new 3D virtual planning tools have been adapted for use in orthognathic surgery with varying success. As with anything in health care, innovations must demonstrate improvements upon the tried and true traditional approaches of disease prevention, diagnosis of disease, and their treatments. New approaches for any condition should be welcomed if there are clear demonstrated benefits in terms of patient health outcomes and improved delivery of care. Any advancements over traditional approaches should be evaluated and considered carefully to determine not only safety and effectiveness but also cost, efficiency, and overall value.

Advances in the application of computer technologies have proven to be fruitful in countless disease states and interventions and are not only important in the domain of imaging alone. These advances have been instrumental in facilitating the entire process of high-quality interventional health care in the modern patient-centered medical system nearly universally across the gamut of medical pathologies and employed throughout the care continuum from diagnosis to treatment

to follow-up care. While the evolutions of enhanced surgical navigation processes, simulations, and customized digital manufacturing of splints and other devices are exciting advents in the delivery of care for orthognathic surgery (and CTOS in particular), they must also be carefully considered and evaluated as equivalent or better than the traditional approach.

29.2 The Value of Computer-Aided Surgical Simulation (CASS): Adoption, Accuracy, and Cost

29.2.1 CASS Adoption Widespread

Computer-aided surgical simulation (CASS) has been consistently used by many different specialties for the diagnosis, planning, and treatment in numerous conditions over the past several decades. CASS technology has been integrated into many maxillofacial surgical applications [3, 4], including the pediatric craniofacial population [5], correction of dentofacial deformities (such as prognathism and asymmetry [6] and mandibular contour osteoplasty [7]), congenital deformities, defects after tumor ablation, posttraumatic defects [8], reconstruction of cranial defects [9], and reconstruction of the TMJ [10] and as a novel means of condylar repositioning for 3D-printed models [11]. Virtual planning has become routine in complex osteotomies, such as three-part osteotomies [12], mandibular angle, and angle splitting osteotomies [13], a tool to evaluate different sagittal split ramus osteotomy fixation techniques [14], and has shown utility to osteotomy and repositioning guides [15]. CASS technology has demonstrated improvements in surgical accuracy, provides the basis of intermediate and final surgical splint fabrication, and decreases surgeons' time input for presurgical preparation compared to traditional methods of case preparation [16, 17].

Over time, it may be argued that CASS may arm less-experienced surgeons with virtual surgical planning (VSP) tools to begin considering more complex procedures, particularly CTOS. That is not to say that technology itself replaces some measure of surgeon skill and experience, but rather may allow complex surgeries to be more approachable to operators who have confidence in the accuracy and reliability of CASS equipment operation to consider riskier procedures. Nonetheless, it must be emphasized that experience and skill remain paramount factors in achieving CTOS stability regardless of treatment approach (traditional or CASS). Treatment success is contingent upon postoperative stability and relies upon precise positional accuracy, which, if miscalculated, may lead to reoperation. While obvious, it must be stated that an additional reintervention procedure (or procedures) will result in more patient pain, downtime, less productivity, and lost wages, as well as other problems associated with decreased quality of life during the healing period. Furthermore, reoperations subject the patient to an increased risk of complications and a lower likelihood of a completely satisfactory outcome for both the patient and surgeon.

CASS may presumably be more amenable and appealing to surgeons who are relatively comfortable with technologic applications (innovators, early adopters, or early majority [18, 19]) in their own healthcare settings with the goal of ultimately improving the chances for procedural success and a good outcome for the patient. However, there are currently no data to elucidate differences in readiness to adopt CASS between surgeon specialties and other social and professional demographics that encourage the diffusion of innovations for CTOS, a topic that should be explored in future research.

To date, however, it appears that the adoption of CASS processes (including VSP and three-dimensional [3D] printing of splints) has become so widespread that they are now the standard of care for orthognathic surgery [20]. Insomuch as CASS protocols are properly transferred to surgery, CASS has been called "a paradigm shift" in orthognathic surgery [21]. CASS planning has been consistently demonstrated to be an efficient and reliably cost-effective alternative to traditional orthognathic surgery planning across several published studies, demonstrating equivalent or better clinical outcomes compared to the traditional standard two-dimensional planning approach with conventional splint fabrication [22].

29.2.2 Overall CASS Accuracy

Recently, a 2018 study from Belgium by Shaheen et al. [23] used a validation protocol employing inter- and intraobserver reliability measures using intraclass correlation coefficient (ICC) in 15 patients undergoing bimaxillary surgery. Their CASS protocol planning used the PROPLAN software (Materialise, Leuven, Belgium). The virtual planning process itself used a stepwise protocol (Step 1: import DICOM images; Step 2: cranial base registration; Step 3: registration of the maxillary segments; Step 4: calculation of 3D translational and rotational displacements; Step 5: data export). Reliability was reported to be excellent (ICC range: 0.94–0.98; mean variability <0.4 mm and <0.7 degrees for translational and rotational movements).

In 2016, Zhang et al. evaluated the accuracy of CASS in planning in two-jaw orthognathic surgery of 30 patients and compared the preoperative plan with actual postoperative skull models. They used digital

imaging and communications in medicine (DICOM) data files from spiral CT and stereolithic model (STL) data obtained from scans of the dental arch surface. The overall linear mean difference was 0.81 mm (0.71 mm for the maxilla; 0.91 mm for the mandible), and the overall angular mean difference was 0.95 degrees, thus representing a clinically accurate repositioning of the bony segments in two-jaw orthognathic surgery.

In 2013, Hsu et al. [24] performed a prospective study to determine the accuracy of a CASS protocol for orthognathic surgery in 65 consecutive patients at three centers. They reported that the protocol can indeed be accurately and reliably transferred to surgery to not only properly position the maxilla and mandible but for an accurate genioplasty as well. Interestingly, the computer-generated chin template for the genioplasty group at one site was found to provide greater measurement accuracy in the chin subgroup compared to intraoperative measurements [24], suggesting that virtual chin models can also be incorporated into virtual planning with confidence.

Also in 2013, Zinser et al. [21] compared the predictive accuracy between the application of computer-aided design/computer-aided manufacturing (CAD/CAM) splints only (group A; $n = 8$), surgical "waferless" navigation only (group B; $n = 10$), and classic intermaxillary occlusal splints only (group C; $n = 12$) in maxillary planning, and they found that the highest accuracy for the maxillary planning transfer was observed with CAD/CAM splints (<0.23 mm; $P < 0.05$) followed by surgical "waferless" navigation (<0.61 mm, $P < 0.05$) and classic intermaxillary occlusal splints (<1.1 mm; $P < 0.05$). Interestingly, the authors reported that only group A (CAD/CAM splints) preserved the condyles centrally in the TMJ. While maxillary accuracy was found to be improved in the CAD/CAM group, the authors noted that precise prediction of the mandible and soft tissue is much more difficult [21].

In 2013, Sun et al. evaluated accuracy of a CASS protocol with intermediate splint fabrication for 15 bimaxillary surgery patients. They found no significant differences between the virtually planned and the actual movement in three dimensions: sagittal ($P = 0.10$), vertical ($P = 0.69$), and horizontal ($P = 0.83$). The next year, Sun et al. reported on the accuracy of an intraoperative image-guided navigation technology in 17 bimaxillary patients and found that it was a useful and promising tool to guide maxillary positioning in the sagittal ($P = 0.82$), vertical ($P = 0.85$), and mediolateral ($P = 0.81$) directions.

29.2.2.1 Soft-Tissue Prediction Simulators

During the planning process, many patients nervously anticipate coping with what may be potentially major changes in their craniofacial morphology. Therefore, predictive software simulation models have been developed that are useful for this purpose. Integrating such predictive virtual models can not only engage patients in the process of their own care by exploring treatment options but also help manage their expectations and potentially avoid dissatisfaction after the procedure. The accuracy of such models has often come in question. In 2015, Ullah et al. evaluated the accuracy of a 3D soft-tissue predictive model for Le Fort I advancement osteotomies in 13 patients using cone-beam computed tomography (CBCT). They found that the gap between the predictive and actual postoperative differences in soft tissues of the chin and upper lip was statistically nonsignificant (<3 mm for segmented anatomical areas; $P < 0.001$), ranging from 0.65 mm differences in the chin to 1.17 mm in the upper lip. Notably, however, the model needed better accuracy in predicting nasal and paranasal positions.

Liebregts et al. [25] reported on the accuracy of 3D soft-tissue simulation in 60 patients who underwent bimaxillary osteotomies. They used a mass tensor model (MTM) to predict soft-tissue changes and found it to be accurate for clinical use. The mean absolute error between the predictive simulation and actual postoperative profile was 0.81 ± 0.22 mm. Subregional accuracy for the upper lip lower lip, and chin subregins was reported to be 93%, 90%, and 95%, respectively (mean absolute error of < 2 mm for the whole face and upper lip) [25].

In a recent 2018 study, Holzinger et al. [26] used their newly developed computer-assisted Sotirios planning software to determine its accuracy in predicting soft-tissue changes in 16 orthognathic surgery-first patients 6 months postoperatively and reported a mean error measurements of 1.46 mm ± 1.53 mm, which represented clinically suitable accuracy to predict soft-tissue outcome.

29.2.2.2 CASS Guides Harvesting, Molding, and Placement of Free Fibular Flaps

Metzler et al. [27] reported that their 3D CASS protocol for 10 patients who underwent mandibular reconstruction with a free fibular flap was clinically acceptable and reproducible in precision and accuracy. In 2015, Rustemeyer et al. [28] reported that their CAD/CAM technique actually reduced ischemia time of fibula flaps. However, they found that there was no impact on the flap survival or on expediting the total duration of the reconstruction procedure. One 2016 study by Wang et al. [29] compared virtual planning surgery and conventional surgery in mandibular reconstructions with a vascularized fibula flap. The study reported that a CASS approach facilitated the use of prefabricated cutting plates and guides that more easily render fibula flap molding and placement, as well as reducing operating

time, ultimately contributing to a more accurate reconstruction compared to conventional surgery that may positively impact clinical outcomes [29].

29.2.2.3 CASS Planning vs. Conventional Planning

A recent 2018 review by Lin et al. confirmed that the use of 3D printing methods for orthognathic surgery has become widely adopted and generally provides benefits over traditional approaches in optimizing both functional and aesthetic outcomes, improved patient satisfaction, and a more accurate translation of treatment planning [30]. One notable 2018 study by Steinhuber et al. [22] compared office-based VSP to conventional surgical planning for orthognathic surgery. All 40 single-jaw surgery patients (n = 18) and double-jaw surgery patients (n = 22) were planned using both CASS (with computer-aided design/computer-aided manufacturing splints [CAD/CAM]) and conventional planning (with manual splint fabrication). The mean time to fully plan for a single-jaw surgery was 109.3 ± 10.8 minutes for CASS and 145.5 ± 11.5 minutes for the conventional planning method; double-jaw surgery took a mean 149.6 ± 15.3 minutes for VSP and 224.1 ± 11.2 minutes for the conventional planning method, demonstrating that VSP significantly shortened the planning time required in both single-jaw ($P < 0.001$) and double-jaw surgeries ($P < 0.001$). Another study by Kwon et al. in 2014 [31] compared the surgical accuracy of compared VSP to conventional articulator model surgery (AMS) in 42 patients who underwent bimaxillary surgery. They reported that surgical accuracy in maxillary positioning between the two planning methods was comparable and concluded that VSP was a suitable alternative.

29.2.2.4 CASS Accuracy in Surgery-First Patients

In 2018, Tran et al. reported on the accuracy of CASS in 15 surgery-first patients who underwent bimaxillary surgery with 3D-printed surgical splints. They found that the overall linear mean difference between planning and surgical outcome was 0.88 mm (maxilla = 0.79 mm for the maxilla; 1 mm for the mandible), and the overall angular mean difference was 1.16 degrees. They concluded that CASS with the use of 3D-printed splints facilitated the diagnosis and treatment planning, ultimately offering an accurate outcome in surgery-first orthognathic surgical patients. As cited earlier, the 2018 Holzinger et al. [26] study examined their soft-tissue predictor software in 16 orthognathic *surgery-first* patients, which was reported to perform with excellent clinical accuracy.

29.2.2.5 Accuracy of Upper Airway Imaging Programs and CT Scanners

One of the important features of CASS is its greater accuracy in determining key craniofacial measurements prior to CTOS. When obstructive sleep apnea (OSA) is considered, accurate and reliable measurement of the airway is a high priority and mismeasurement could potentially lead to unwanted outcomes. In 2012, Weissheimer et al. [32] compared the precision and accuracy of six imaging software packages for measuring the volume of the upper airway using cone-beam computed tomography (CBCT) in 33 growing patients. They used an oropharynx acrylic phantom and used an i-CAT scanner (Imaging Sciences International, Hatfield, PA). The programs compared were Mimics (Materialise, Leuven, Belgium), ITK-Snap (► www.itksnap.org), OsiriX (Pixmeo, Geneva, Switzerland), Dolphin3D (Dolphin Imaging & Management Solutions, Chatsworth, CA), InVivo Dental (Anatomage, San Jose, CA), and Ondemand3D (CyberMed, Seoul, Republic of Korea). For reliability, intraclass correlation coefficient was tested and found that all six software packages were reliable, though the rate of volumetric errors notably differed between some programs. They reported that the accuracy of Mimics, Dolphin3D, ITK-Snap, and OsiriX (all <2% volumetric errors vs. gold standard) was statistically different from InVivo Dental and OnDemand3D (>5% volumetric errors vs. gold standard) for upper airway assessment ($P < 0.05$, with <2% volumetric rate of error) [32]. Since 2012, incremental improvements in reliability and accuracy have been made, as suggested by the more recent 2017 evaluation by Chen et al. [33] of the reliability and accuracy of three different software packages. The authors compared Amira (Visage Imaging Inc., Carlsbad, CA), 3Diagnosys (3diemme, Cantu, Italy), and OnDemand3D (CyberMed, Seoul, Republic of Korea). They found that all three packages demonstrated excellent reliability in intra- and interobserver measurements of the upper airway (intraclass correlation coefficient ≥ 0.75), with excellent agreement between all three in measurements of volume, length, and minimum cross-sectional area. Furthermore, all three packages were found to underestimate upper airway volume by −8.8% to −12.3%, length by −1.6% to −2.9%, and minimum cross-sectional area by −6.2% to −14.6%.

Accuracy of MDCT and CBCT scanning is also an important factor in determining oropharynx volume and related morphology. In 2018, Chen et al. also reported on the accuracy of two multidetector row computed tomography (MDCT) scanners (GE Discovery CT750 HD, Siemens Somatom Sensation) and three different CBCT scanners (NewTom 5G, 3D Accuitomo

170, Vatech PaX Zenith 3D). Of all scanners evaluated, the Siemens MDCT (accuracy of 98.4%; 14.3 cm^3) and the Vatech CBCT (accuracy of 98.9%; 14.4 cm^3) demonstrated the best performance in volumetric accuracy.

29.2.3 Cost

The actual cost–benefit, cost utility, or cost-effectiveness of CASS compared to traditional CTOS protocols has not yet been performed from with any methodologically robust cost studies. Questions remain as to the utility of CASS as an "expensive toy or useful tool" [34], though the purchase cost of even basic CASS technology has decreased along as it has been adopted with nearly universal acceptance in the global orthognathic surgical community. As to whether the cost of CASS is justified, we have little data, but we surmise that the increased accuracy of CASS in its multiple iterations and differing protocols over the years may likely impart cost–benefits from avoidance of complications and other adverse surgical outcomes (such as reoperations) that may be precipitated, at least in part, because of errors in the planning process.

CASS may impart a cost–benefit in time and labor savings (although this may be somewhat offset by the long learning curve in training not only surgeons but also their staff, as discussed below). One 2006 cost study that evaluated a CASS protocol reported that, compared to traditional complex craniomaxillofacial surgeries, CASS cost is less in surgeon planning and procedure time (5.25 hours for CASS vs. 9.75 hours for traditional methods), patient time, and material costs, ultimately concluding that CASS is faster and less costly than traditional methods [35]. Xia et al. noted that any new innovative surgical design candidate or new process should be justified by a positive answer to the question of at least two of these three benefits over the traditional method: (1) is it faster, (2) is it cheaper, and/or (3) does it yield better outcomes?

Similarly, the cost of 3D printers and the 3D printing process has steadily decreased, as this technology is used more and more in general usage by the public, but it is also being adopted by orthognathic surgeons as a key visualization tool for orienting and educating patients as to their own surgical process, and hence the surgeon can more accurately measure for anchoring splints and other devices critical to surgical success. Even low-cost fused deposition modeling 3D printer (also known as a rapid prototyping 3D printer) has been shown to be an excellent fast-printing option that has a similar relative error margin vs. more costly technologies [36]. Even a paper-based 3D printer demonstrated that its average error margin of printing a human mandible was no greater when compared to other types of 3D printers [37].

29.2.4 CASS Learning Curve and Related Costs

While there is little research on the topic of learning curves in orthognathic surgery and related surgeon training, learning curve is likely a factor in the adoption by established practitioners. As such, it is important to consider the learning curve of CASS and time commitment to reach competency and mastery, which was recently pointed out in a 2017 systematic review evaluating surgical navigation technology applications in oral and maxillofacial surgeries. The authors of the systematic review found that although computer-guided processes may be time-consuming, it is inevitable foreign technologies and processes will, at some point, become familiar and routinely efficient. Overall planning and procedure times will ultimately benefit once CASS techniques and process are "sufficiently mastered," citing the experience of surgical-guided navigation in plastic surgeries [38, 39]. Furthermore, the surgeon's entire team must also dedicate time to become familiar with CASS processes, lengthening not only the learning curve and associated costs of training until competence is demonstrated.

29.2.5 Surgical Simulation and Training to Improve Clinical Outcomes

Impressive progress has been made in the development of virtual reality (VR) simulators in just the past 5 years. VR simulators promise to be a cost-effective and efficient alternative to traditional medical training and planning. The development of controllers that allow haptic feedback in conjunction with virtually immersive head-mounted devices (HMDs) has been instrumental in placing surgeons in the driver's seat before they get into the surgical suite. Many simulators feature multimodal (visual, touch, sound, smell, etc.) output/feedback, which are designed immerse the learner in interactivity and achieve training objectives to closer emulate competence that can later lead to mastery.

Sofronia et al. in 2013 [40] reported on their development of a bilateral sagittal split osteotomy (BSSO) virtual reality-based simulator for training surgeons specifically tuned to provide feedback, so trainees can recognize and constructively learn from failures of sawing and splitting, two major causes of surgical errors and complications. More recently, a 2018 paper by Arikatla et al. [41] describes that their development of a high fidelity haptic feedback simulator is suitable for real-time virtual surgery. They claim to be able to simulate bone drilling, treating the drill burr, or oscillating saw as a virtual coupling object with realistic physics-coded software meant to approximate contact forces on the

bone. The haptic feedback device (Geomagic Touch™) can provide maximum force feedback of 3.4 N, which can also synchronize virtual position, including tool orientation, to render forces along 3 Cartesian coordinates. Their hardware consists of Oculus Rift HMD with an organic light emitting diode (OLED) panel for each eye at a pixel resolution of 1080 × 1200 [41].

29.3 Protocol for Traditional CTOS

Traditional (or conventional) CTOS planning relies on clinical evaluation, 2D prediction tracing based on cephalometric analysis, photography, as well as dental cast models mounted in an articulator with facebow transfer, and model surgery is performed to simulate jaw movement using fabricated surgical splint. All these steps provide the basis for the surgeon to move the upper and lower jaws to establish optimal treatment outcome in function, facial harmony, occlusion, and oropharyngeal airway dimensions [22]. If total joint prostheses are needed, a CT scan of the maxillofacial region is performed encompassing the TMJs, maxilla, and mandible with 1 mm overlapping cuts. An STL model is then fabricated using these CT imaging data with the mandible as a separate piece. Using the original cephalometric tracing and prediction tracing (Fig. 29.1a), the mandible on the STL model is placed into its future predetermined position using the planned measurements for correction of mandibular anteroposterior and vertical positions, pitch, yaw, and roll.

The mandible is stabilized to the maxilla with quick-cure acrylic. Many patients with temporomandibular disorders (TMDs) requiring concomitant orthognathic surgery are also candidates for counterclockwise rotation of the maxillomandibular complex, which requires the development of posterior open bites on the model (Fig. 29.1b). Because the mandibular position on the STL models is established using hands-on measurements, the operator's manual dexterity and 3D perspective are critical in properly positioning the mandible. However, it must be pointed out that this step is inherently risky as it is accompanied by a certain margin of error.

The next step requires the preparation of the lateral aspect of the rami and fossae (Fig. 29.2a, b) for fabrication of the patient-fitted total joint prostheses. The goal of this step is to recontour the lateral ramus to a flat surface in the area where the mandibular component will be placed. The fossa requires recontouring only if heterotopic bone or unusual anatomy is present. The recontouring areas are marked in red for duplication of bone removal at surgery. Because most patients with TMJ problems requiring CTOS can benefit from counterclockwise rotation of the maxillomandibular complex, the STL model is likely to be set with posterior open bites, because the maxilla is maintained in its original position.

Once the STL model is finalized, it is sent to TMJ Concepts (Ventura, CA) to perform the design, blueprint, and wax-up of the custom-fitted total joint prostheses (Fig. 29.2c), with the design and wax-up sent to the surgeon for approval before manufacture of the prostheses. The period from CT acquisition to the manufacturer's completion of the custom-fitted prostheses is approximately 8 weeks. The surgical procedures are then performed on articulator-mounted dental models. The mandible is repositioned on the articulator, duplicating the movements performed on the STL model, and the intermediate splint is constructed. The maxillary model is repositioned, segmented if indicated, and placed into

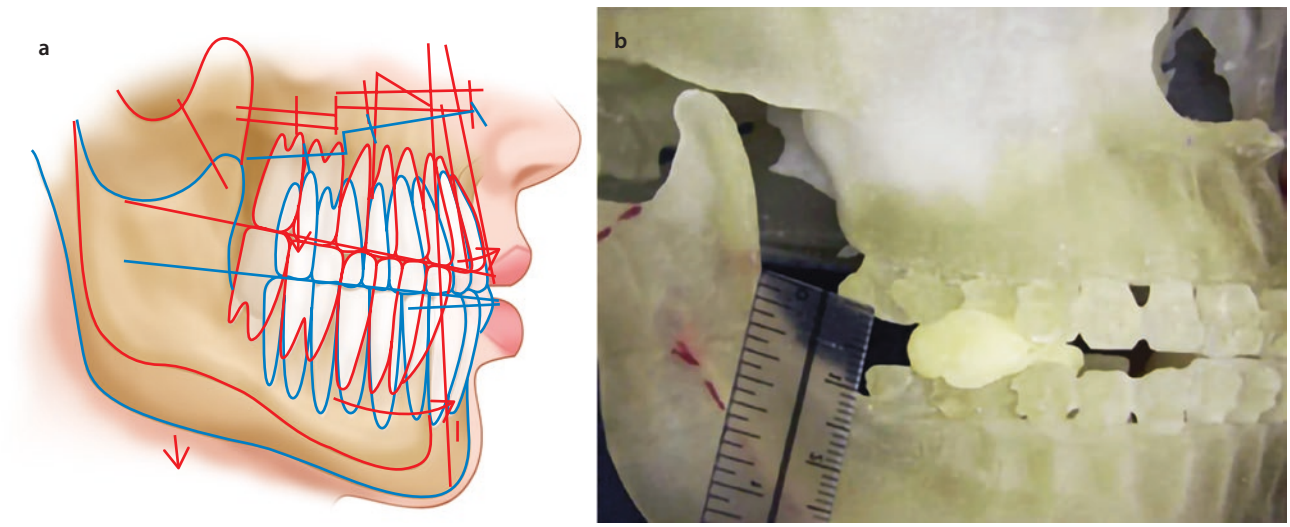

Fig. 29.1 **a** Measurement of the cephalometric prediction tracing for open bite produced at the second molar after counterclockwise rotation of the mandible into its final position. **b** Duplication of the measurement obtained from the prediction tracing to the final mandibular position on the STL model and fixating the mandible to the maxilla with methyl methacrylate. (From Movahed et al. [16]; with permission)

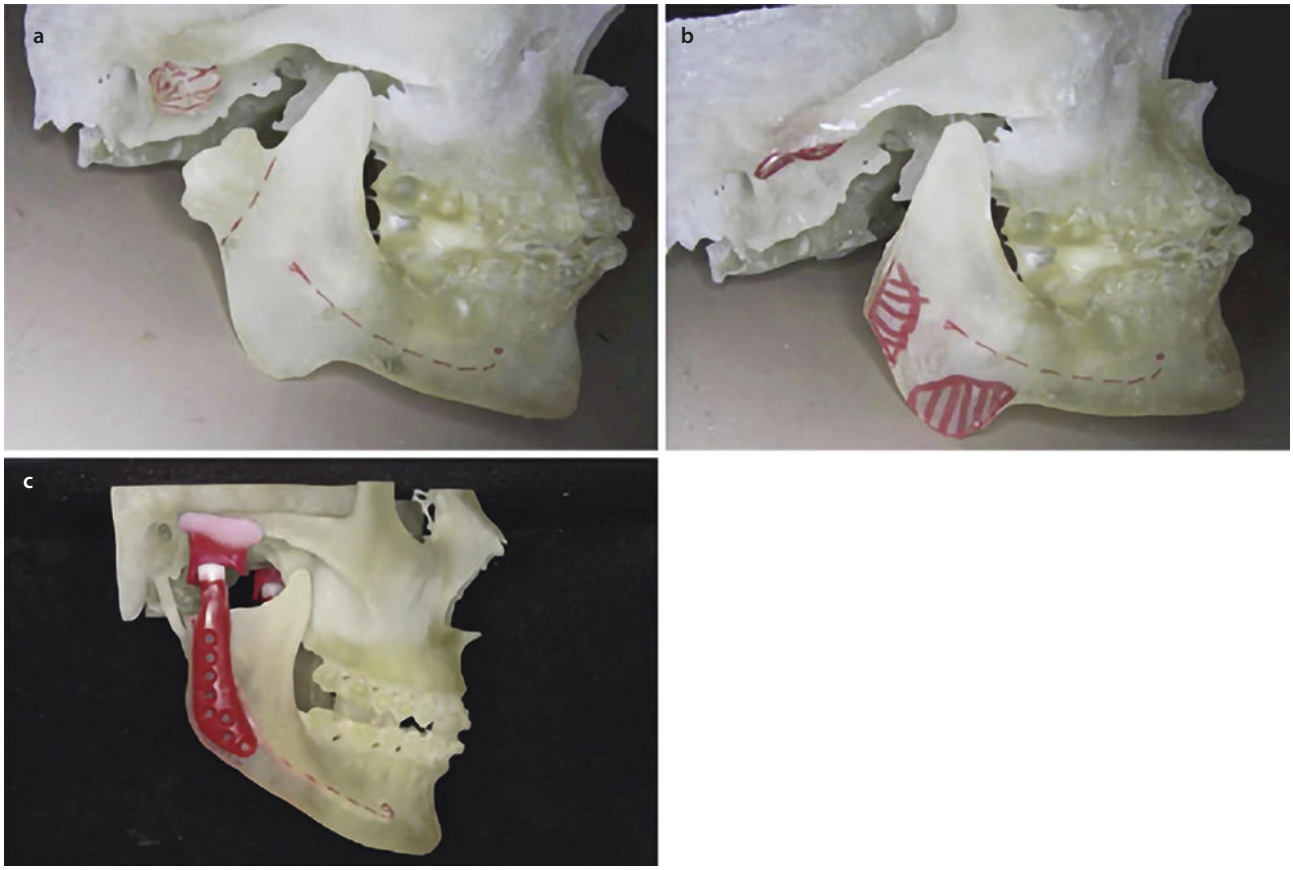

Fig. 29.2 **a** Marking the condylectomy osteotomy and the irregularities of the fossa. **b** The STL model after condylectomy and recontouring of the fossae and rami (marked in red). **c** This STL model demonstrates a prosthesis wax-up for approval by the surgeon. (From Movahed et al. [16]; with permission)

the maximal occlusal fit. Then, the palatal splint is constructed.

CASS technology is used to move the maxilla and mandible into their final position in a computer-simulated environment. Using the computer simulation, the anteroposterior and vertical positions, pitch, yaw, and roll are accurately finalized for the maxilla and mandible based on clinical evaluation, dental models, prediction tracing, and computer-simulation analysis. Using Digital Imaging and Communications in Medicine (DICOM) data, the STL model is produced with the maxilla and mandible in the final position and provided to the surgeon for removal of the condyles and recontouring of the lateral rami and fossae if indicated. The STL model is sent to TMJ Concepts for the design, blueprint, and wax-up of the prostheses. Using the Internet, the design is sent to the surgeon for approval. The custom-fitted total joint prostheses are then manufactured. It takes approximately 8 weeks to manufacture the total joint custom-fitted prostheses.

29.4 New Protocol for Concomitant TMJR and MMA Using CASS

Using CASS technology for CTOS cases eliminates the "traditional" steps requiring the surgeon to manually set the mandible into its new final position on the STL model, thus saving time and improving surgical accuracy. Although dental model surgery is necessary only if the maxilla requires segmentation, models in the CASS process do not require mounting on an articulator, ultimately saving considerable time by eliminating the time required to mount the models and prepare the model bases for model surgery, mandible repositioning, constructing the intermediate occlusal splint and the final palatal splint. With CASS technology, the splints are manufactured by 3D Systems (Rock Hill, SC) and total joint prostheses (if needed) by TMJ Concepts. The new CASS protocol process is detailed below.

29.4.1 Overall CASS Process

29.4.1.1 Step 1: Patient Referral

The patient may be referred to the orthognathic surgeon by any source, but some typical referring sources are rheumatologists, orthodontists/dentists, otolaryngologist, sleep medicine physician, craniofacial surgeon, oncologist, primary care physician, patient self-referral, or referral from a former patient, friend, or family member.

29.4.1.2 Step 2: Initial Outpatient Evaluation In-Office

At the initial outpatient office visit, the patient is welcomed and complete administrative releases and consent for care is established. This is followed by an initial CBCT scan. Baseline photographs of the patient are taken (anterior and profile), as well as intraoral photographs. The patient is then interviewed, and a complete clinical workup and history is performed. The patient's medical and dental history is discovered (including any chronic diseases or traumatic events), and their medication history and current medications, any pertinent family history, and any prior referrals (such as orthodontic care) or prior procedures are documented. Imaging data are evaluated to determine any soft-tissue deficiency of skeletal and soft-tissue markers. Any symptoms are discussed (such as TMJ pain and discomfort at rest and/or while eating). The clinical exam consists of palpation manipulation to assess the amount of necessary advancement, and an assessment of pitch, yaw, and roll of occlusal bite is performed, as well as a periodontal examination of dentition. The surgeon will initiate a discussion of medical assessment findings, present potential treatment options, and elicit patient buy-in and engagement throughout the treatment process. If the patient decides to move forward, the surgeon will discuss of potential complications of surgery in general, as well as specifically focusing on any risk factors discovered in the patient's medical history and initial discussion. The surgeon will ascertain the patient's immediate caregiver support (spouse, siblings, children, or other family, and/or other caregiver support). A recommendation will then be made for the patient to engage in other important medical consults (e.g., cardiac assessment, pulmonary function test; if revision case, assess quality of tissue and bony structure). The next follow-up will be at preoperative visit postorthodontic care. The next preoperative visit will establish perioperative telemedicine check-ins with the patient and caregivers for all patients regardless of geographic location.

29.4.1.3 Step 3: Referral to Orthodontist to Initiate Presurgical Orthodontic Care

Orthodontic care is begun with braces or Invisalign selected for level alignment of teeth on the alveolus. In addition, there is a need for divergence of teeth and opening spaces in the distal-lateral spaces of the maxilla, which allows for safer segmental osteotomy and achieving a more predictable alignment of a reproducible bite during surgery.

29.4.1.4 Step 4: CT Scan Data Sent to 3D Systems for Splint Fabrication

The CT scan data are uploaded to the cloud database at 3D Systems for virtual surgical planning of the fabrication of the CAD/CAM splints and any other surgical models that needed fabricating (order form available at ► https://www.3dsystems.com/sites/default/files/2017-05/MM-163%20Rev%20H_VSP%20Orthognathics_0.pdf). In multidisciplinary conference, the surgeon and orthodontist agree upon preplanned skeletal movements implemented as part of the virtual surgical planning process. Once all parties agree upon the plan and it is finalized, a physical STL 3D model is fabricated and sent to TMJ Concepts for splint fabrication. The TMJ Concepts protocol is initiated to make the total joint when the patient is deemed ready by the orthodontist and surgeon to proceed with surgery (protocol available at ► https://tmjconcepts.com/tmj/files/CT_Scan_Protocol_F071-H.pdf) [42].

29.4.1.5 Step 5: TMJ Concepts Begins Fabrication of Total Joint Prostheses

TMJ Concepts takes on fabrication of the total joint prosthesis. TMJ Concepts will immediately report any abnormalities to the surgeon and any corrective action is directed, obviating the time-consuming need for the model to be shipped, manually altered by the surgeon, and shipped back. The average duration of prosthesis fabrication is approximately 2–4 months, depending on the company's workload.

29.4.1.6 Step 6: STL Scan Obtained from Patient 3 Weeks Prior to Surgery for Validation

To validate accuracy, the STL scan is obtained from the patient's dentition 3 weeks prior to surgery for fabrication of the total joint prostheses. During this time, the patient's dentition has been held into position by orthodontia. The STL scan is then sent to 3D systems and aligned with the follow-up CT scan that has already been performed. Dental segment positioning is rechecked

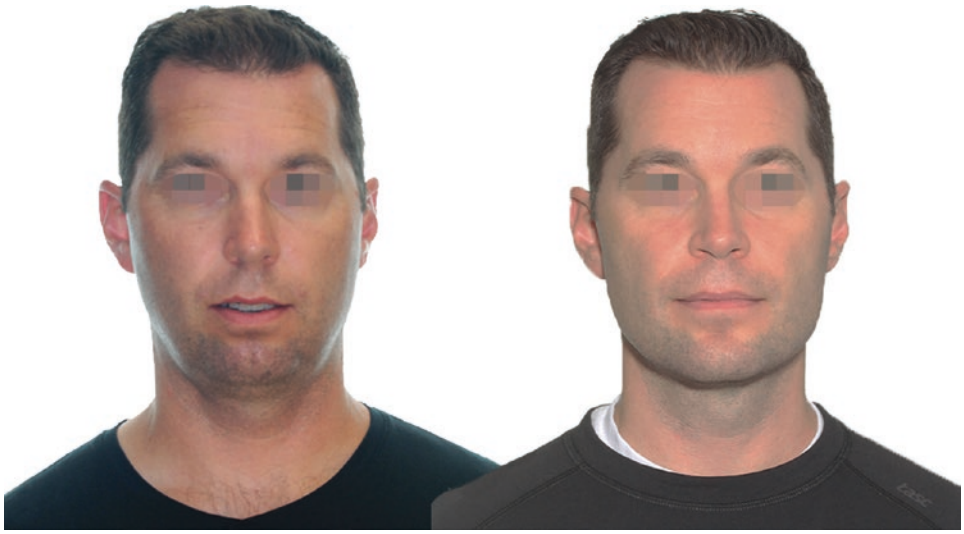

Fig. 29.3 Preoperative photography shows retrognathia

and verified by the multidisciplinary team consisting of engineer, orthodontist, and surgeon. Once agreed upon by all parties, the intermediate, palatal, segmental, and final splints are printed from the virtual file and readied for shipment. The 3D prints are then shipped and received by the surgeon days prior to surgery and checked for defects.

29.4.1.7 Step 7: Procedure

On the day of surgery, surgeon is provided with splints provided by 3D systems and prostheses provided by TMJ Concepts. The fabricated splints are rechecked and cleared for use in the procedure operating theater. CTOS (TMJR and/or MMA) is then performed, without any necessity of lateral ramal shave.

29.4.1.8 Step 7: Follow-Up: Perioperative Care, Transition to Office Visits, and Follow-Up Imaging

Following CTOS, the patient is moved to the intensive care for 48–72 hours and then cleared for discharge if there are no extenuating complications. Telemedicine follow-ups are scheduled at regular intervals beginning upon discharge until the first office visit.

29.4.2 Case 1: Symptomatic Idiopathic Condylar Resorption

A male patient was followed from age 13 to 18 and referred by orthodontist in 2014 at the age of 15 for evaluation of TMJ pain, clicking, and popping accompanied with headaches. At the time of the first visit, the patient was in full orthodontics and a conservative treatment of arthroscopy/arthrocentesis is completed. The patient continued follow-up with some increases in pain and progressing retrognathia noticeable during this pubertal period (Fig. 29.3). Initial orthodontic treatment was completed within a year (completed at age of 15) with little symptom alleviation over that period. Throughout the patient's growth period, pain was managed with dental splints and nonsteroidal anti-inflammatories (NSAIDs). From 2014 to 2017, multiple arthroscopy/arthrocentesis procedures were performed to alleviate discomfort from additional dislocations, jaw locks, and related pain. TMJ surgery was discussed as an option with the patient and family in annual follow-ups in 2015 and 2016. Because of the patient's pain severity and progressing retrognathia of the skeletal morphology throughout puberty, the decision was made to proceed with CTOS with the objectives of achieving a functional airway and to restore the patient's occlusion with optimal aesthetic harmonious positioning. Surgery planning was initiated when the patient was 17 years old (Fig. 29.4). Surgical risks were discussed and presurgical treatment education discussed requirements for perioperative care. The patient was once again treated with orthodontics to regain level alignment prior to surgery (Table 29.1). The virtual planning protocol was initiated to prepare for surgery as previously detailed (Fig. 29.5). In 2017, the patient underwent surgery, which included reconstruction of custom-fitted prosthesis, maxilla LeFort I counterclockwise rota-

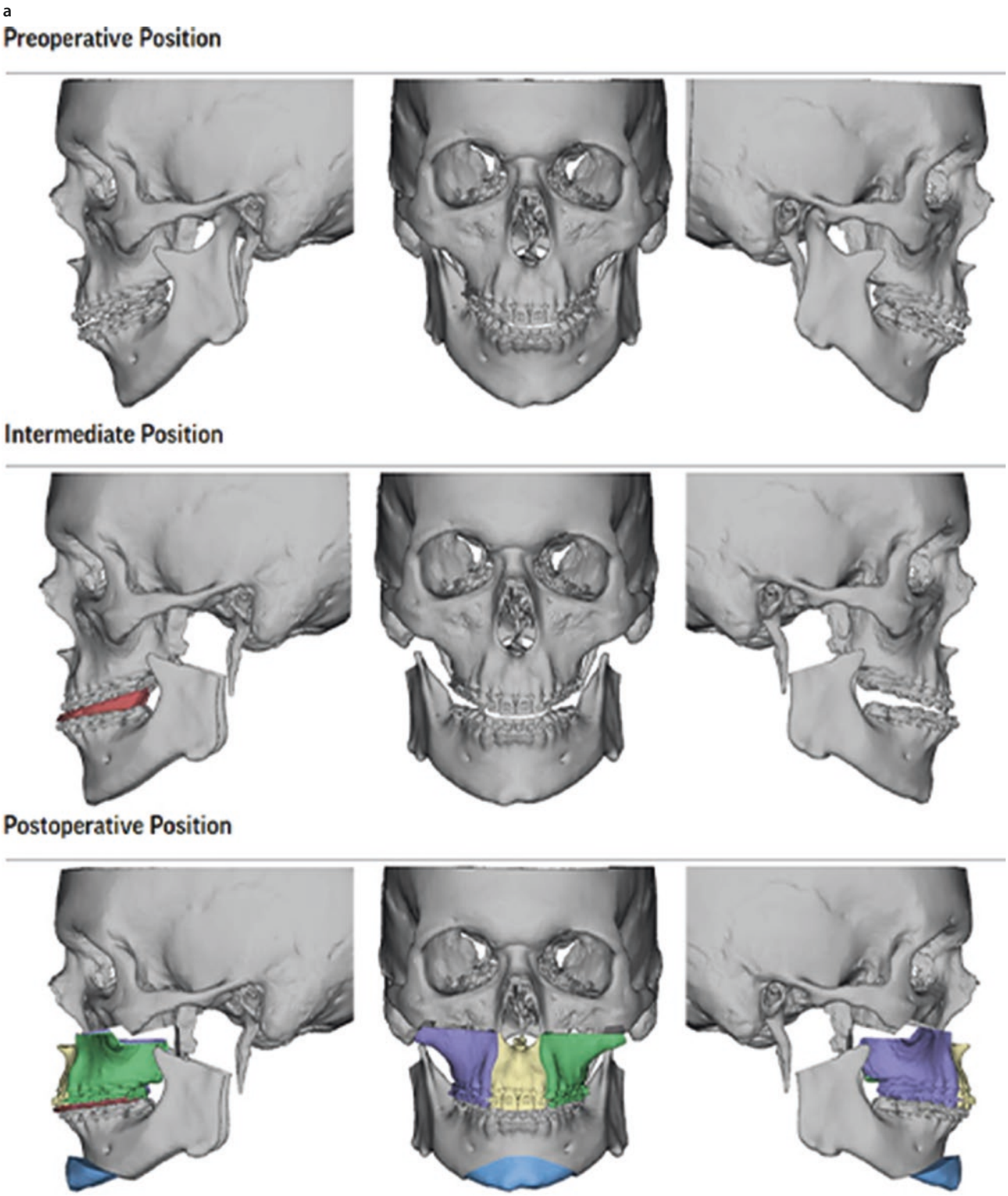

Fig. 29.4 Presurgical planning

b

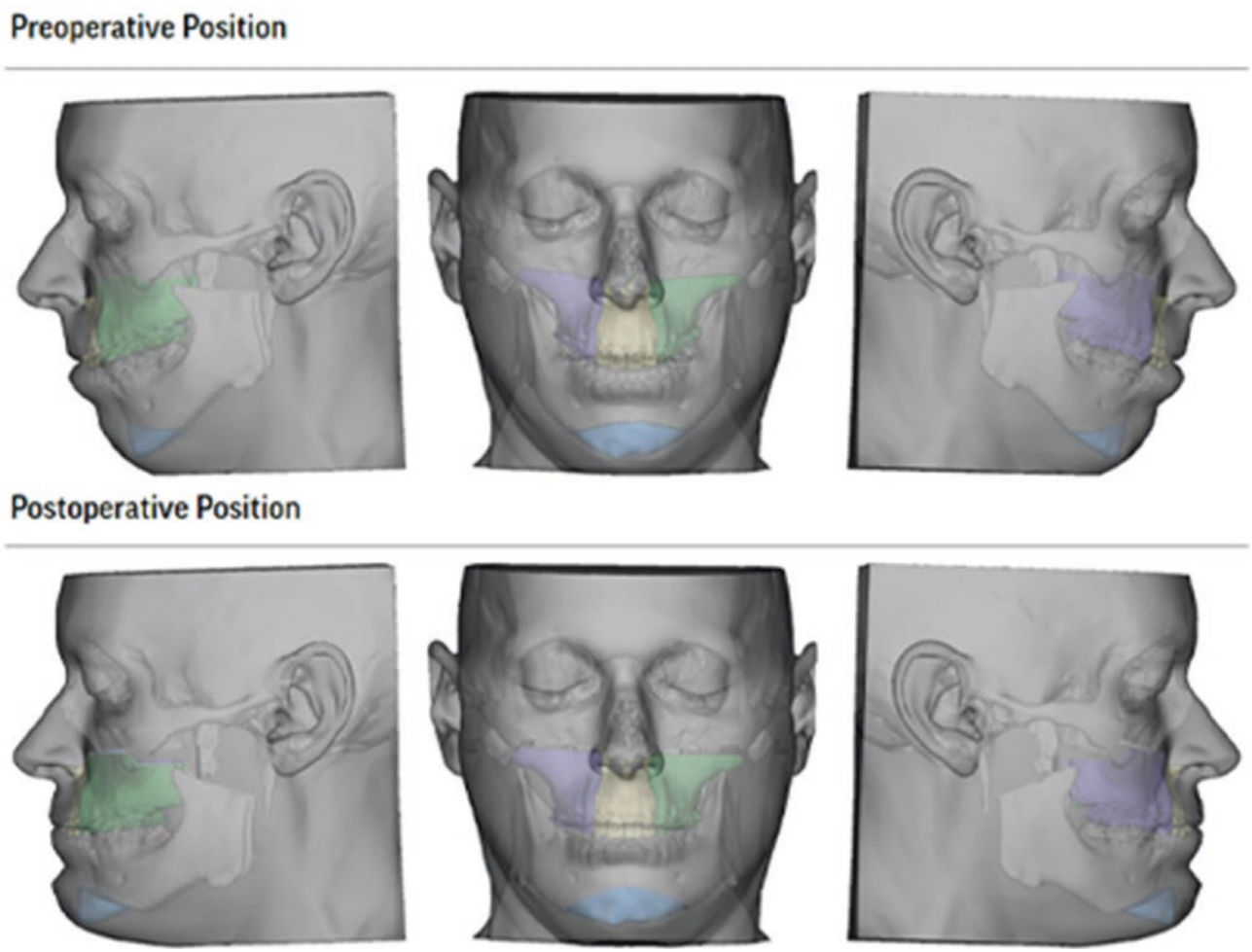

Fig. 29.4 (continued)

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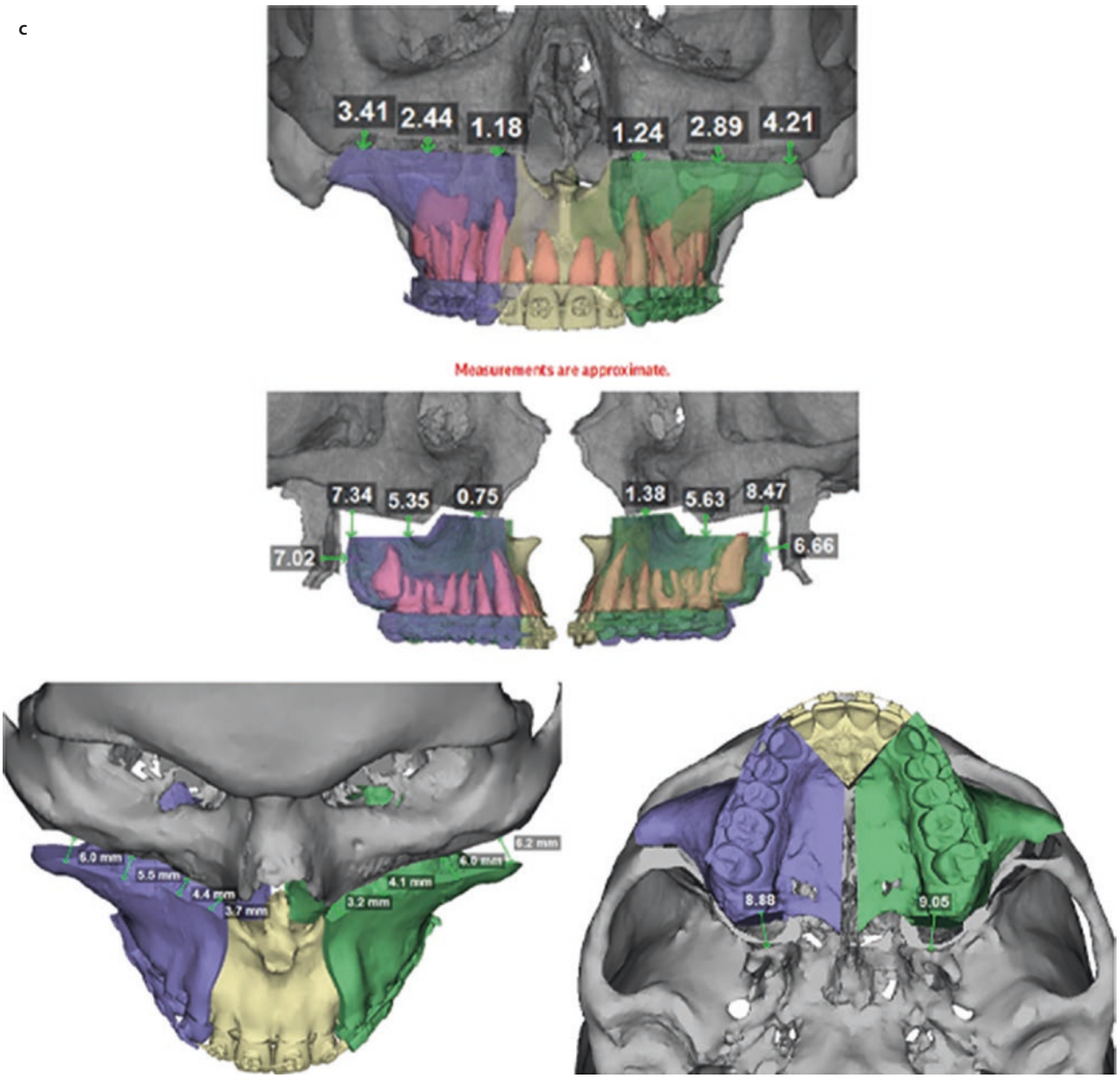

Fig. 29.4 (continued)

Table 29.1 Measurements

Point	Name	Anterior/Posterior	Left/Right	Up/Down
ANS	Anterior Nasal Spine	2.36 mm Anterior	2.80 mm Left	3.57 mm Up
A	A Point	4.74 mm Anterior	1.67 mm Left	1.72 mm Up
ISU1	Midline of Upper Incisor	9.50 mm Anterior	0.25 mm Left	2.00 mm Up
U3L	Upper Left Canine	8.37 mm Anterior	0.02 mm Right	0.93 mm Down
U6L	Upper Left Anterior Molar (mesiobuccal cusp)	7.00 mm Anterior	0.67 mm Right	5.96 mm Down
U3R	Upper Right Canine	10.24 mm Anterior	0.11 mm Right	1.36 mm Up
U6R	Upper Right Anterior Molar (mesiobuccal cusp)	9.54 mm Anterior	0.75 mm Right	2.63 mm Down
ISL1	Midline of Lower Incisor	9.81 mm Anterior	0.10 mm Left	1.64 mm Up
L6L	Lower Left Anterior Molar (mesiobuccal cusp)	8.30 mm Anterior	0.59 mm Right	5.16 mm Down
L6R	Lower Right Anterior Molar (mesiobuccal cusp)	10.01 mm Anterior	0.63 mm Right	2.25 mm Down
B	B Point	14.04 mm Anterior	1.67 mm Right	0.94 mm Down
Pog.	Pogonion	25.38 mm Anterior	4.75 mm Right	0.28 mm Up

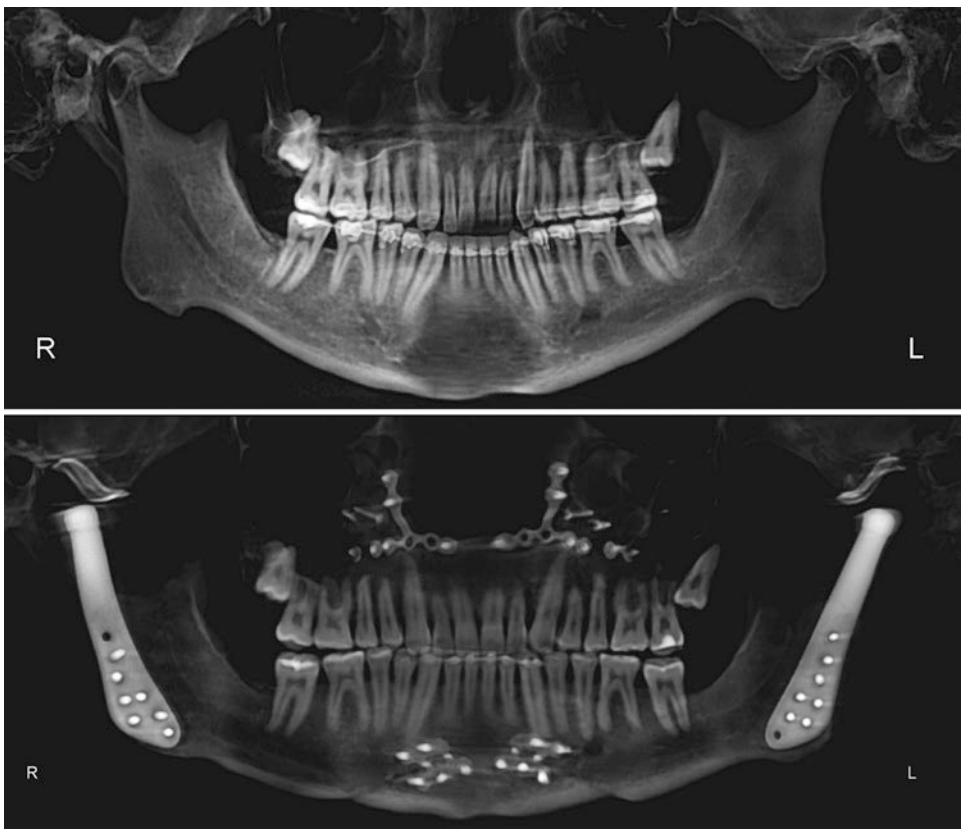

Fig. 29.5 Virtual planning workflow

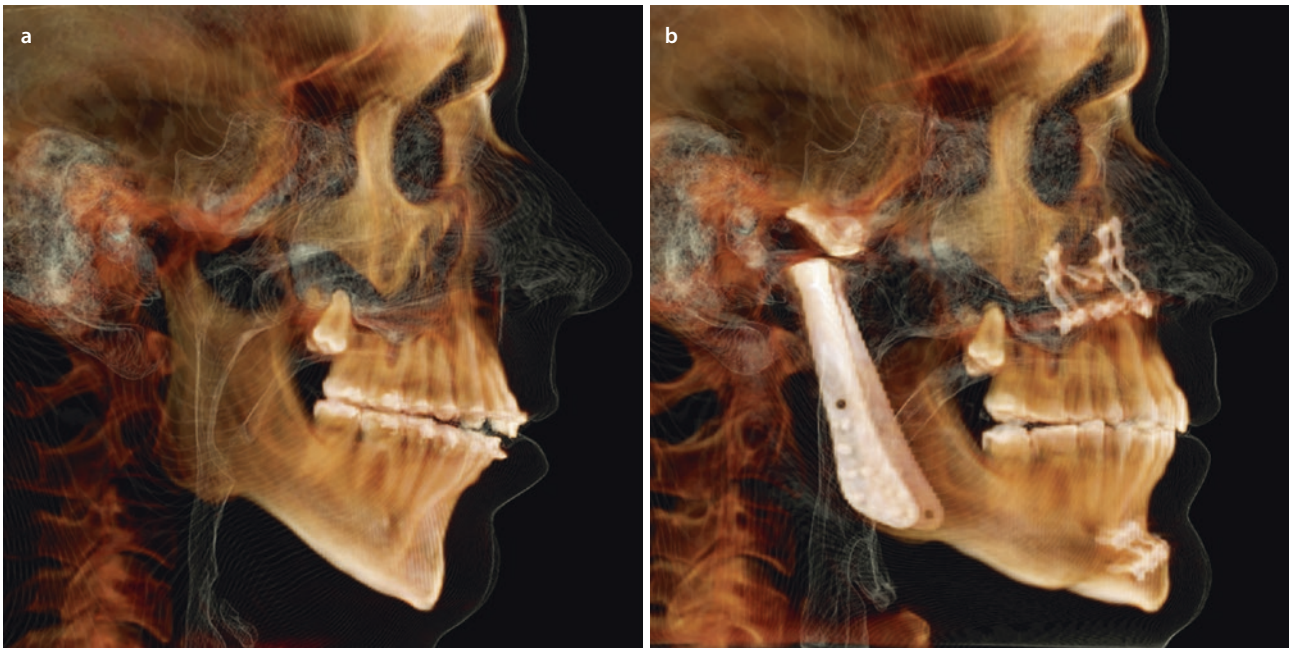

Fig. 29.6 Pre- and postsurgical CBCT images obtained and 1-year post-index procedure showing a minimal axial area improvement of 94 mm^2 preoperatively and increased to 151 mm^2 at 1-year post-index procedure

tion with skeletal fixation, counterclockwise rotation performed concomitant with total joint replacement (TJR) (abdominal harvest of fat graft to TMJ), and genioplasty. Outcomes are shown in the postsurgical photographs and figures (Figs. 29.6 and 29.7). It is evident that the patient no longer has lip incompetence and function has been restored without any pain with a maximal incisal opening of 44 mm, with facial harmony regained. Upon follow-up, the patient reported that pain had subsided and headaches ceased.

29.4.3 Case 2: TMJ Pain and Condylar Resorption

A 25-year-old woman with chronic TMJ pain was referred by the patient's orthodontist after second orthodontic relapse to the practice of Movahed OMS for evaluation. The patient had been diagnosed with TMD and idiopathic condylar resorption (ICR) at the age of 23. The patient had undergone multiple orthodontic treatments since the age of 14. The patient had a history of eating difficulty and discomfort, and often required NSAID pharmacotherapy prior to eating (no significant NSAID side effects reported). After referral to Movahed OMS after second relapse for evaluation of TMJ pain and potential surgical correction, full orthodontics were once again initiated to level and align to prepare for surgery. The patient was diagnosed with ICR as the normal anatomy of the condyles was no longer recognizable. Preoperative photos show lip incompetence was evidence attributable to retrognathia of maxilla and mandible. Virtual surgical planning was performed in tandem with the patient to reinforce shared decision-making process, keeping the patient aware of all steps in the process. The patient underwent TMJR, MMA with counterclockwise rotation, and genioplasty approximately 1 year after orthodontia at the age of 26. Maximal incisal opening gain was 41 mm in postoperative physical therapy. Patient healed rapidly. Upon follow-up, joint function completely restored with no more lip incompetence. Facial balance and harmony achieved with good patient satisfaction. Although the patient required orthodontics 1 year postoperatively, NSAIDs were no longer needed prior to meals (Figs. 29.8, 29.9, 29.10, 29.10, and 29.11).

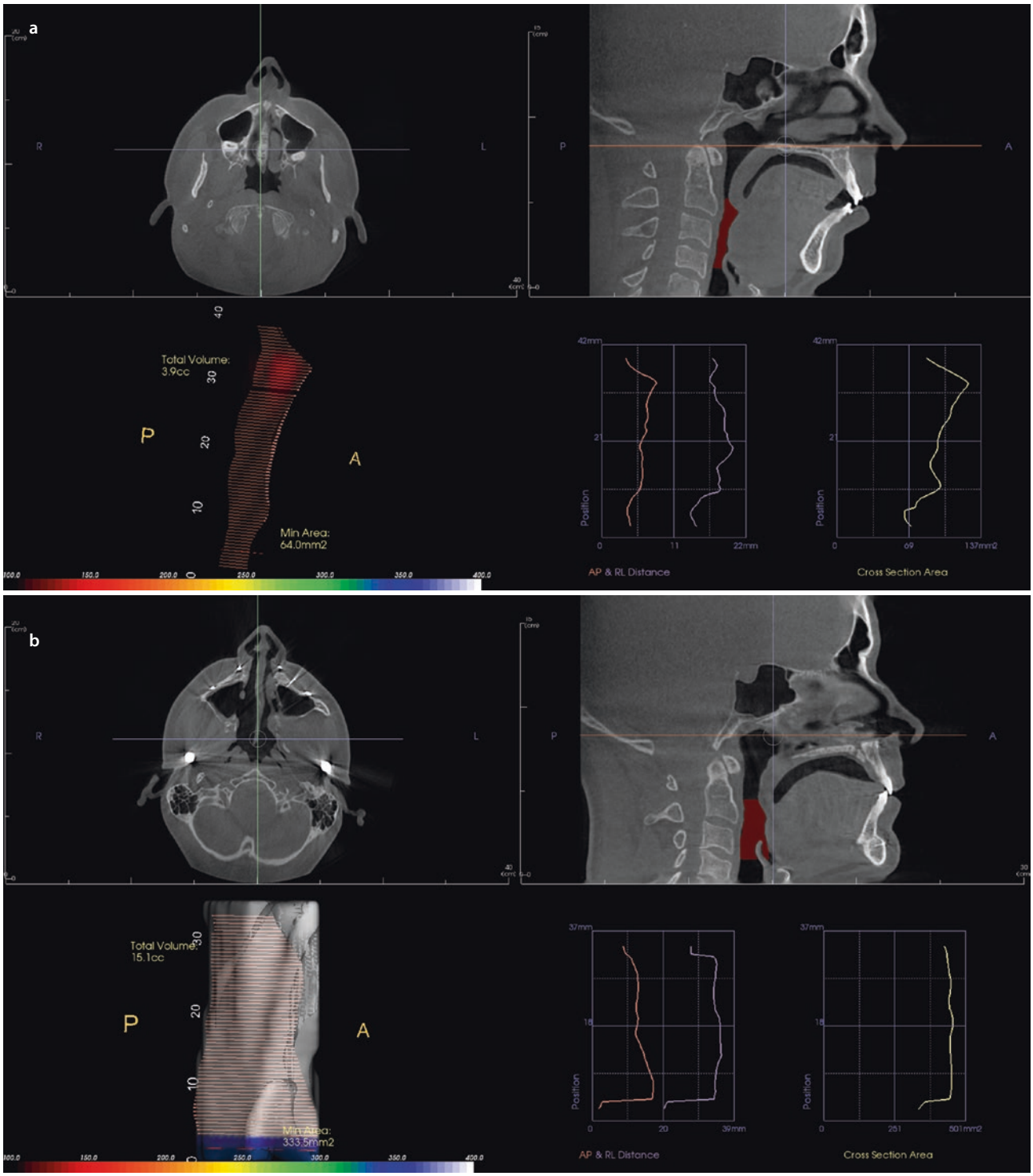

Fig. 29.7 Preoperative (4 weeks prior) and postsurgical (2 months postoperatively) photographies

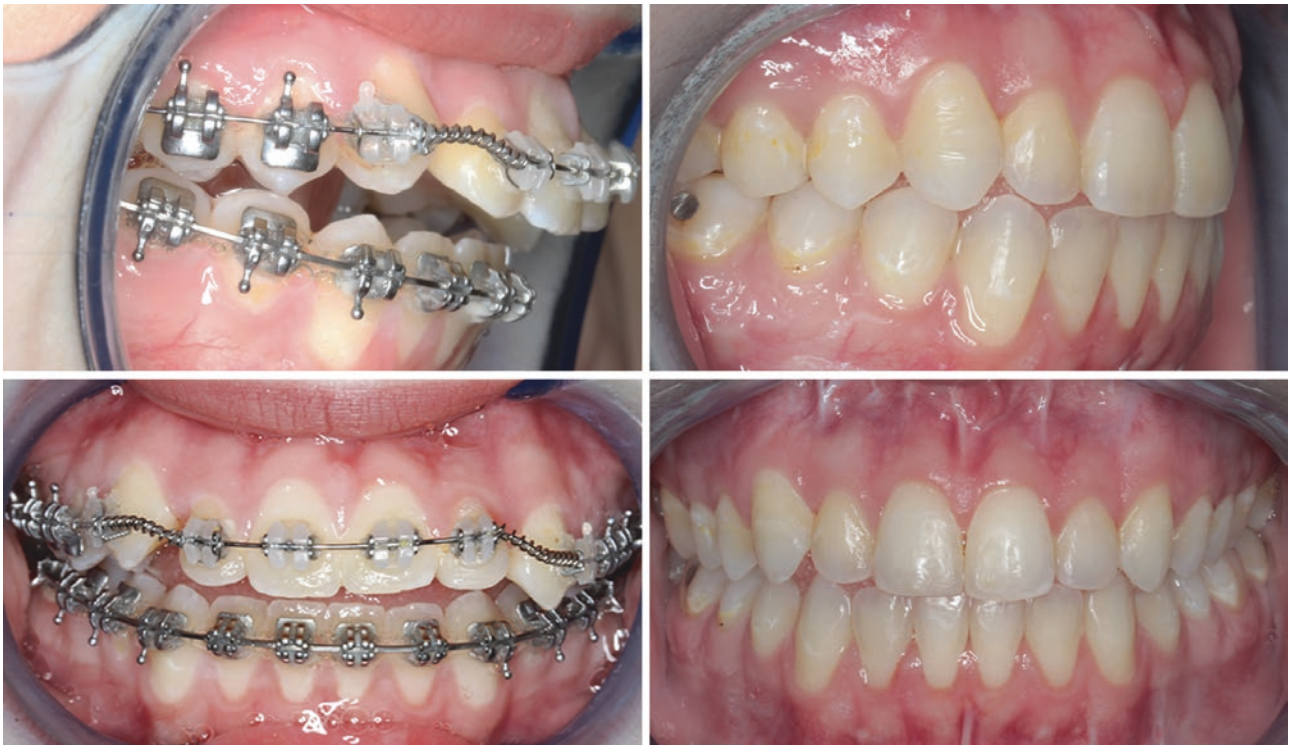

Fig. 29.8 Pre- and postoperative profiles

29.4.4 Case 3

29.4.4.1 Case: Patient with Juvenile Idiopathic Arthritis

A 15-year-old girl was referred to Movahed OMS for evaluation and management of TMJ pain and shortness of breath from an oral surgeon, who noted that the patient's airway was compromised. Notably, the patient also had a medical history of juvenile idiopathic arthritis (JIA) diagnosed at age 7. TMJ symptoms and airway difficulties were suspected to be caused by absolute destruction of normal joint anatomy and resultant severe retrognathia impeding the airway (Figs. 29.12 and 29.13). Patient had an apnea-hypopnea index (AHI) of 21 and was diagnosed with OSA. After evaluation and imaging, multiple treatment options were discussed. Patient and family opted for TMJR to restore joint function and restore facial harmony to reverse sequelae of her pathology. Virtual surgical planning protocol was begun to prepare for the procedure (Table 29.2). A LeFort I advancement was performed with counterclockwise rotation and segmental osteotomy of the maxilla with uprighting of the anterior segment and expansion of the palate. TMJR was achieved with total joint prostheses from TMJ Concepts with abdominal fat harvested from abdomen and applied to the TMJs. Mandibular advancement was also performed in a counterclockwise fashion, as well as genioglossal advancement (Figs. 29.14, 29.15, and 29.16).

Fig. 29.9 Preoperative lateral cephalometry

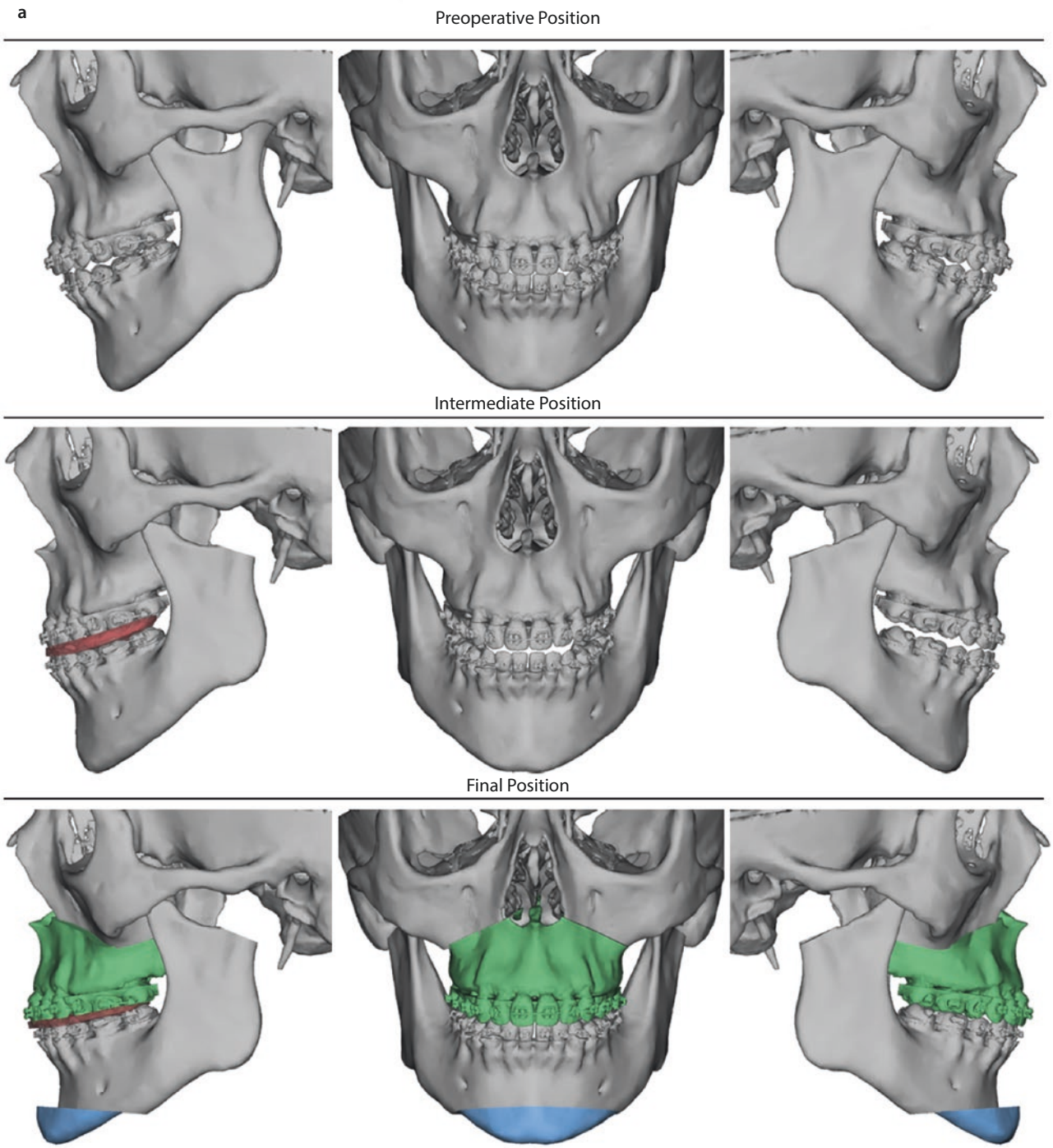

Fig. 29.10 Postsurgical profile photographs

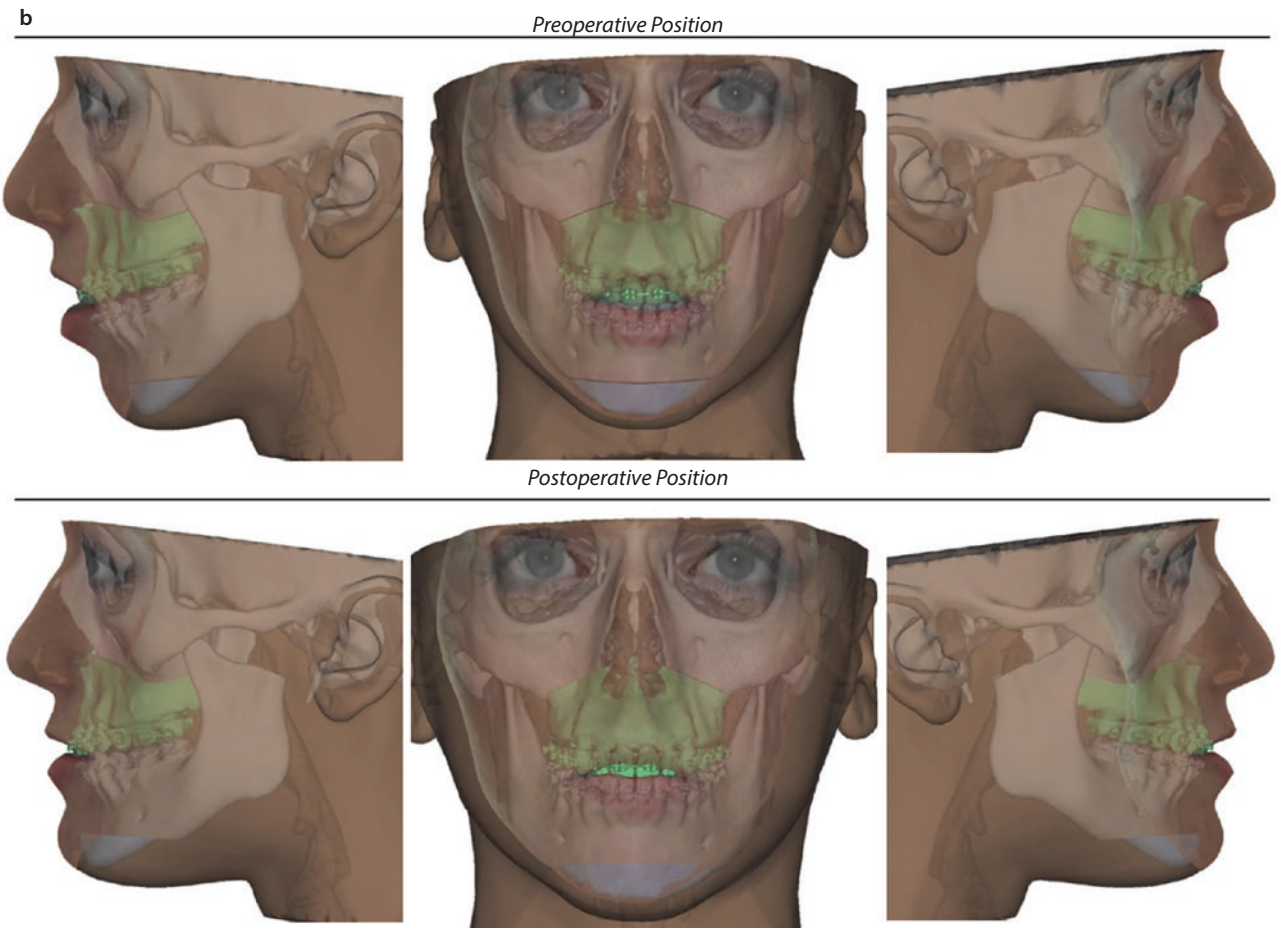

Fig. 29.10 (continued)

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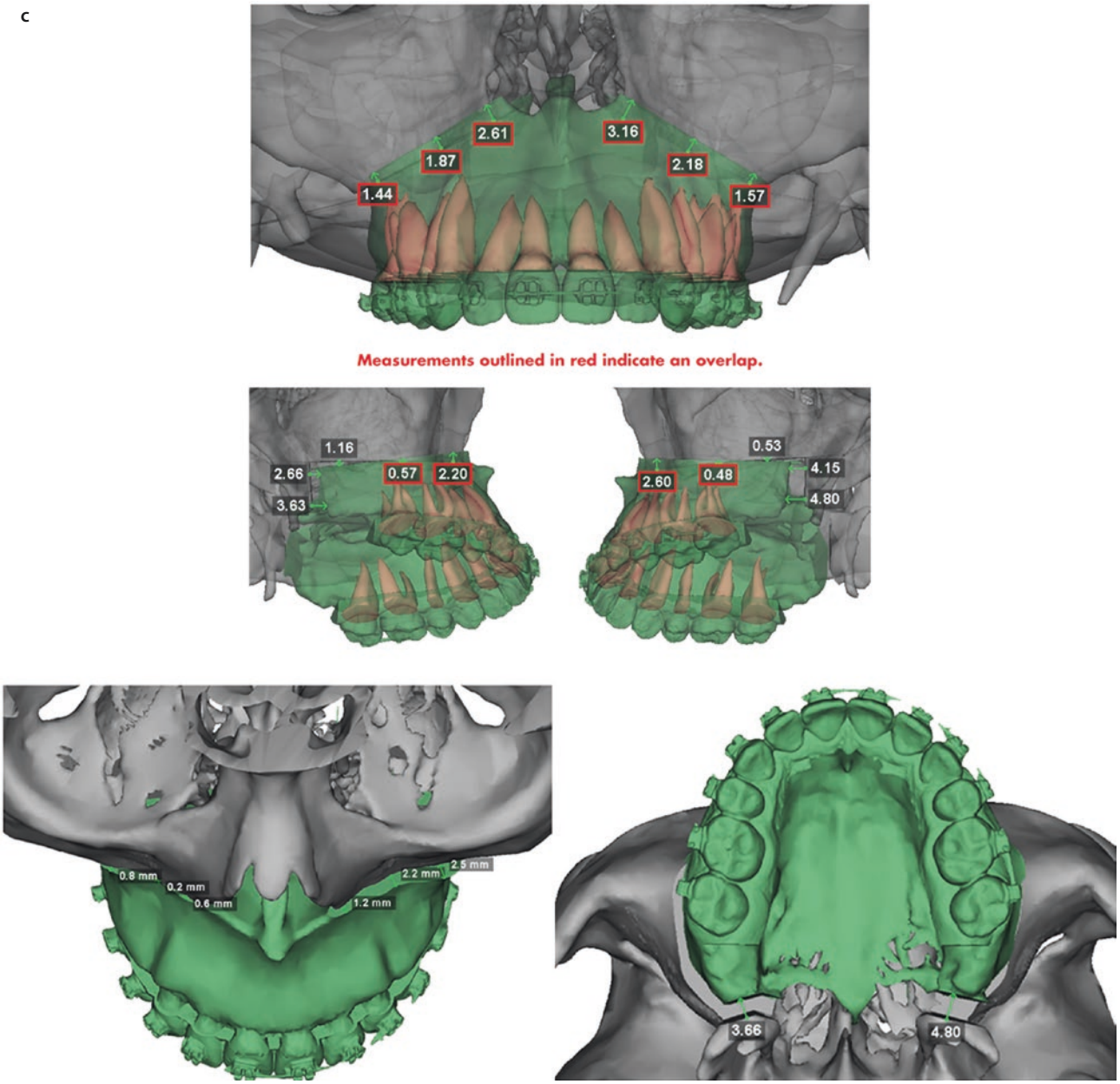

Fig. 29.10 (continued)

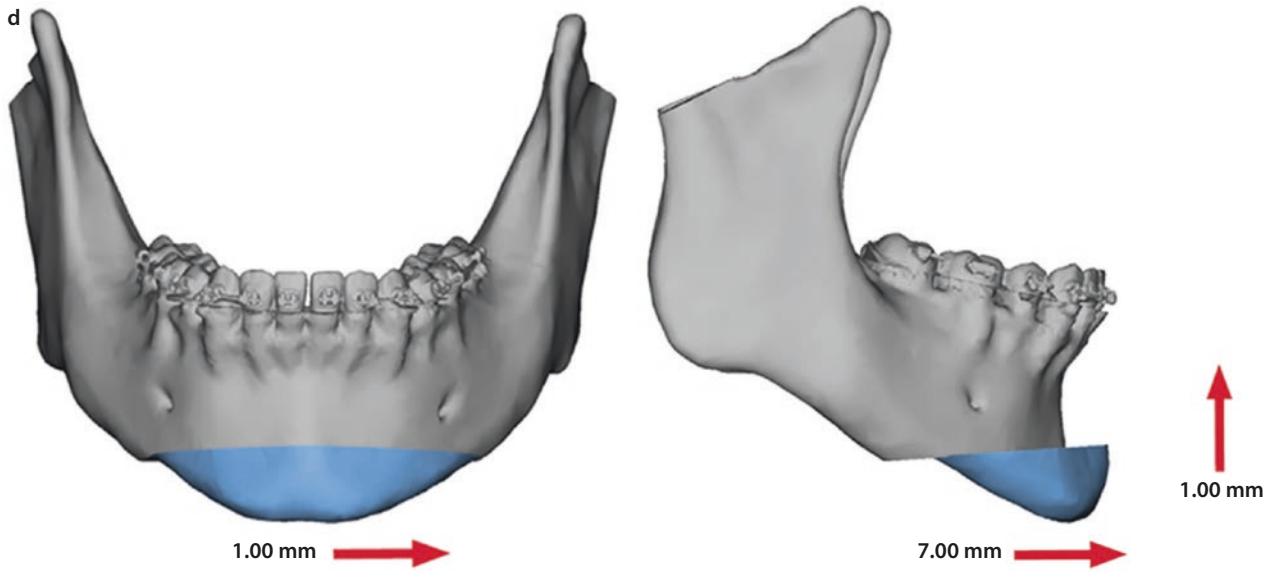

Fig. 29.10 (continued)

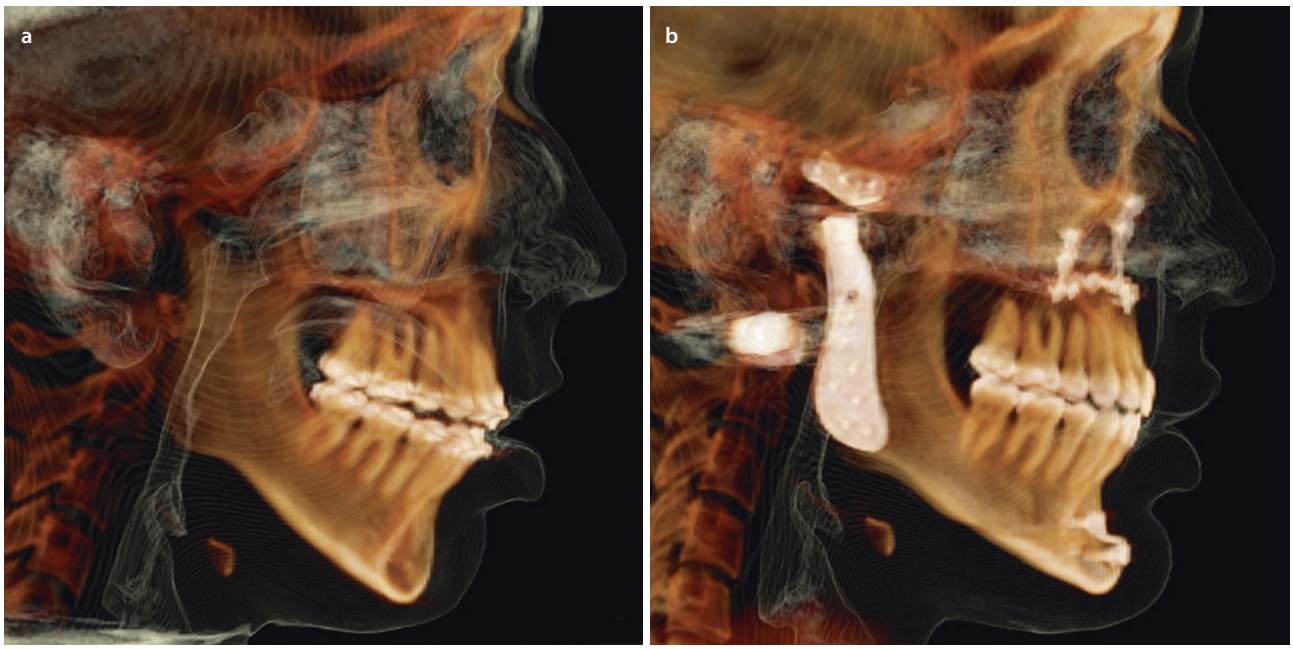

Fig. 29.11 Postoperative lateral cephalometric

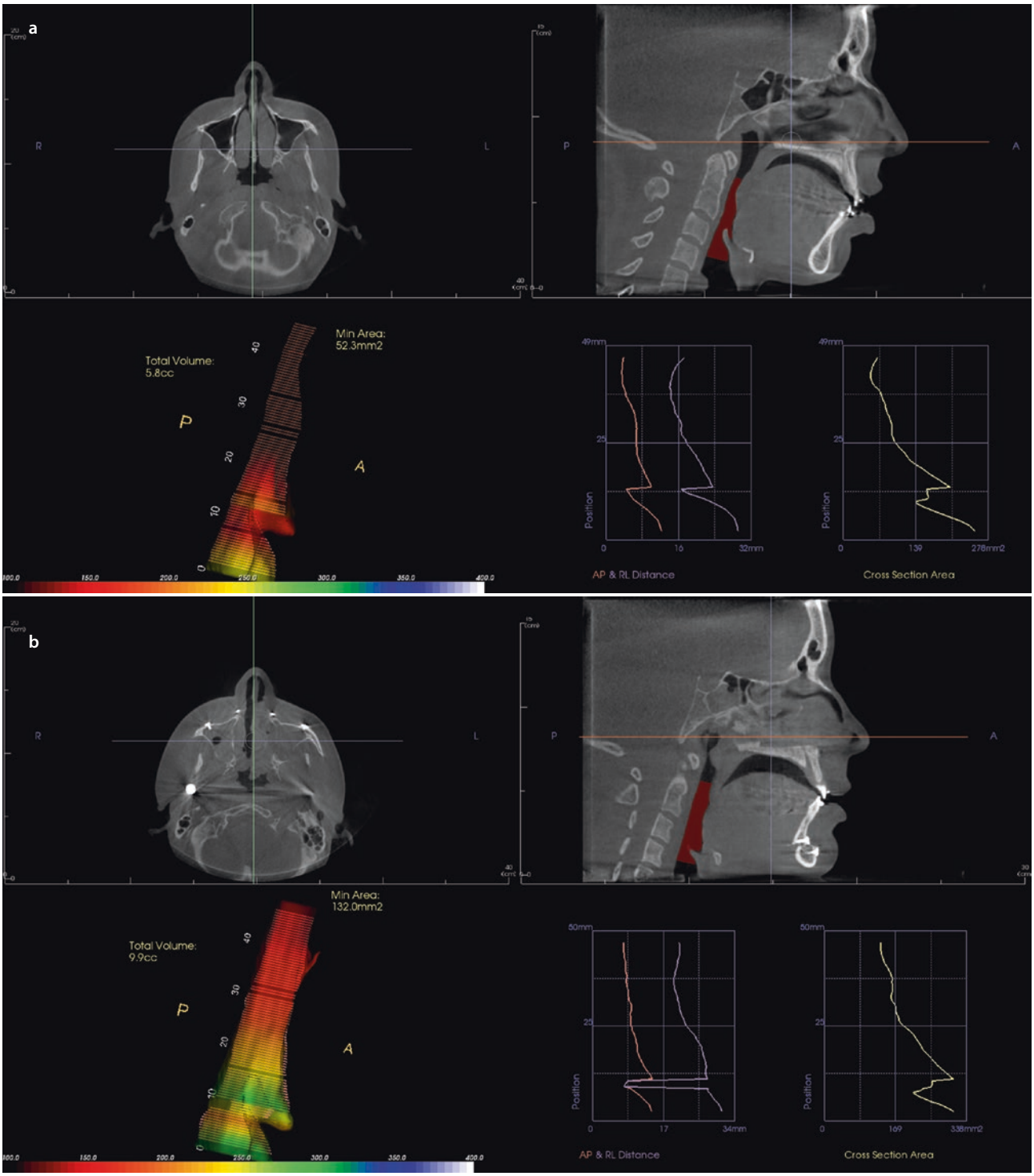

Fig. 29.12 Preoperative photography

Fig. 29.13 In panoramic view, it is evident that the inflammatory pannus caused by the JIA has resulted in complete destruction of the TMJs

Table 29.2 Virtual surgical planning was engineered to gain proper facial balance. Decision was made to move 32 mm at the pogonion in addition to 7 mm movement at the midline of the upper incisors

Point	Name	Anterior/Posterior	Left/Right	Up/Down
ANS	Anterior Nasal Spine	0.46 mm Posterior	2.00 mm Left	2.61 mm Up
A	A Point	0.98 mm Anterior	2.00 mm Left	2.00 mm Up
ISU1	Midline of Upper Incisor	7.00 mm Anterior	2.00 mm Left	3.00 mm Up
U3L	Upper Left Canine	6.77 mm Anterior	2.00 mm Left	1.46 mm Up
U6L	Upper Left Anterior Molar (mesiobuccal cusp)	4.87 mm Anterior	2.65 mm Left	4.63 mm Down
U3R	Upper Right Canine	6.08 mm Anterior	2.46 mm Left	1.15 mm Up
U6R	Upper Right Anterior Molar (mesiobuccal cusp)	4.90 mm Anterior	1.85 mm Left	4.59 mm Down
ISL1	Midline of Lower Incisor	9.56 mm Anterior	2.00 mm Left	4.00 mm Up
L6L	Lower Left Anterior Molar (mesiobuccal cusp)	6.56 mm Anterior	2.00 mm Left	5.84 mm Down
L6R	Lower Right Anterior Molar (mesiobuccal cusp)	6.55 mm Anterior	2.00 mm Left	6.36 mm Down
B	B Point	18.34 mm Anterior	2.00 mm Left	2.00 mm Down
Pog.	Pogonion	31.70 mm Anterior	2.00 mm Left	2.31 mm Down

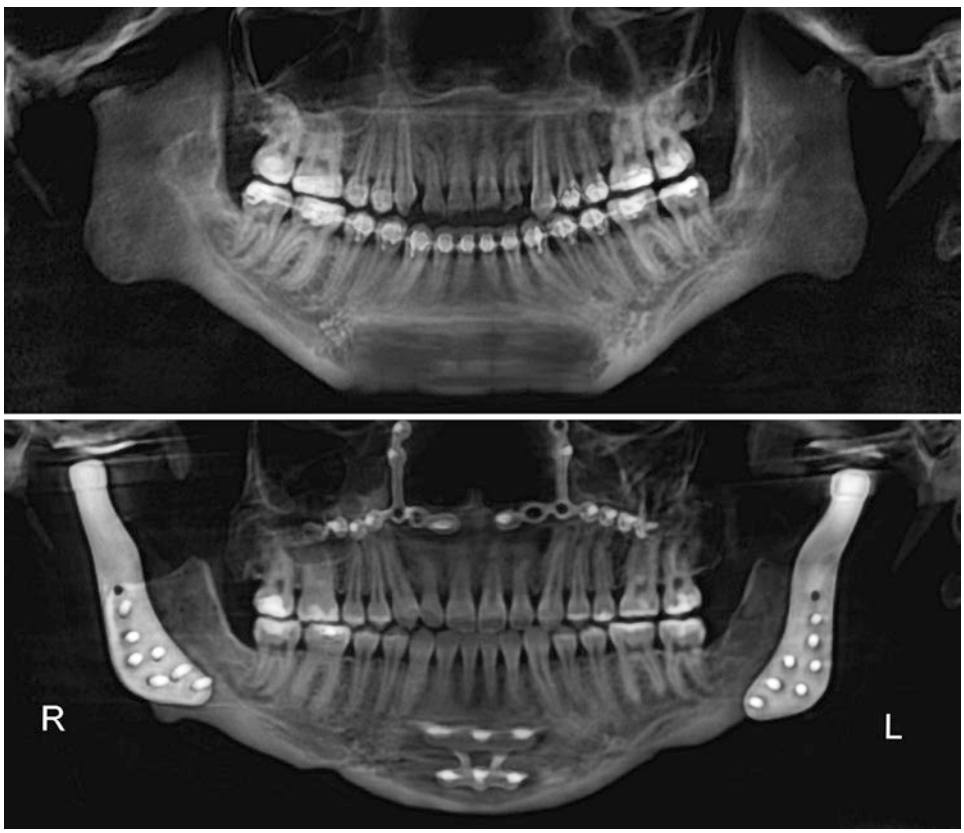

Fig. 29.14 3D rendering of preoperative and postoperative position

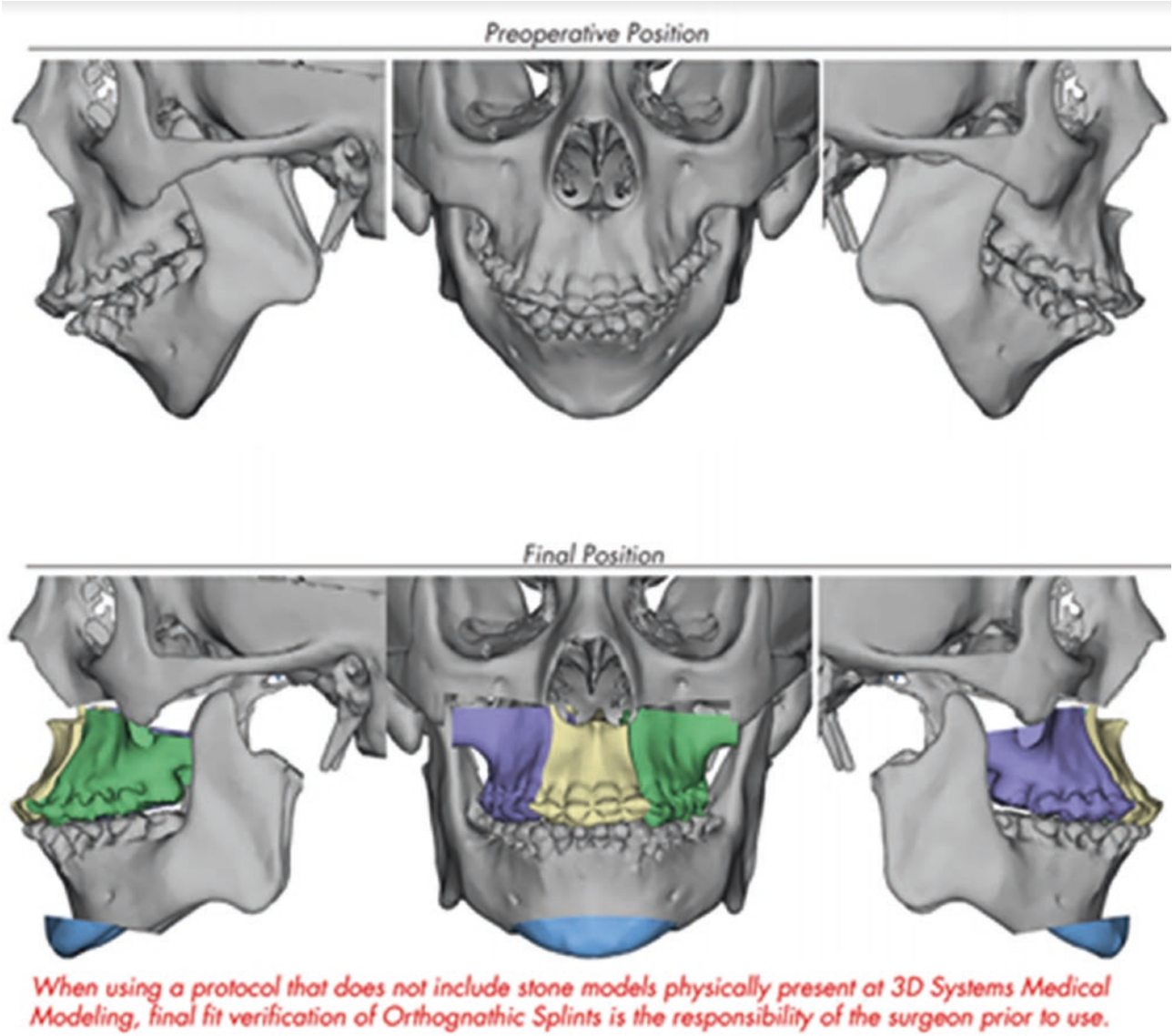

Fig. 29.15 Pre- and postoperative CBCT take with i-CAT FLX V series. Airway imaging analysis by TX STUDIO for Anatomage imaging software

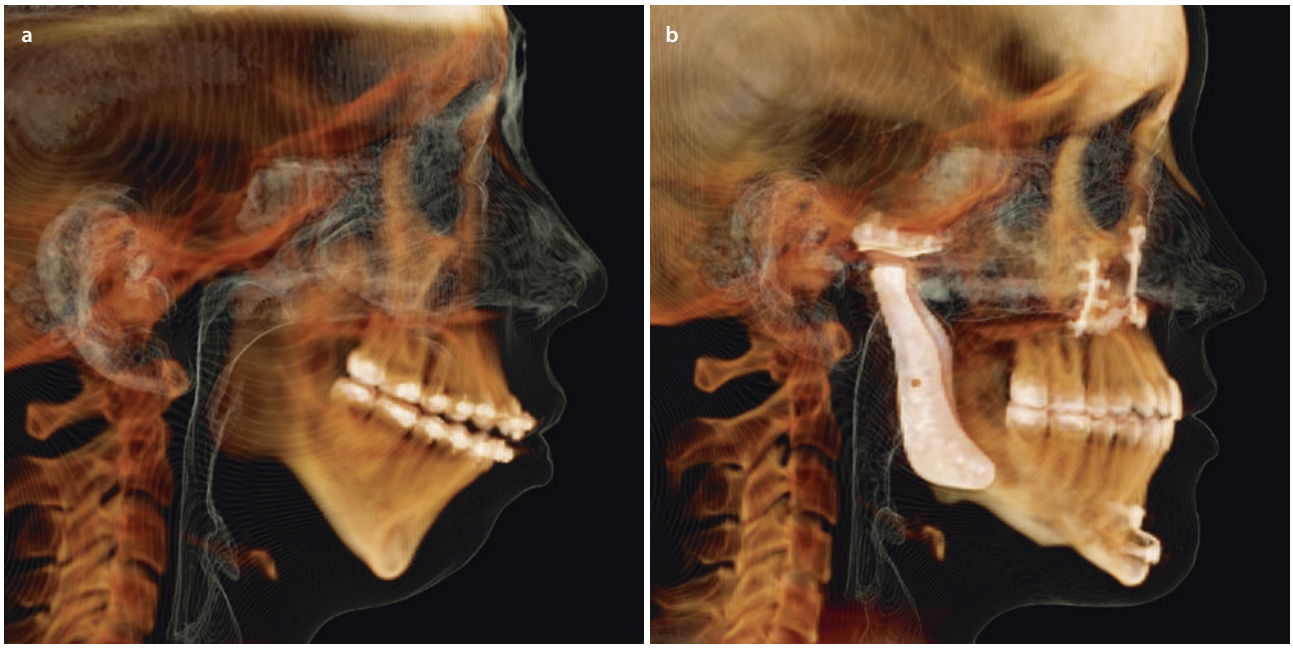

Fig. 29.16 Pre- (left) and postoperative (right) airway analyses showed minimal axial area improved from 29.8 mm^2 to 204 mm^2

29.5 Conclusion

Using CASS technology for CTOS cases, the surgeon superimposes the orthognathic computer-simulated surgery into the production of the STL model, hence decreasing the margin of error that can occur with hands-on positioning of the mandible on the STL model. Furthermore, this technique decreases the time taken by the surgeon in the laboratory by 3D Systems for splint fabrication and by TMJ Concepts for the fabrication of prostheses, and for setting the STL model with increased accuracy in the process (Table 29.3).

The remaining areas in which improvement can be made in CASS technology include performing recontouring of the rami and fossae in the simulated environment in an accurate fashion, eliminating the requirement for the acquisition of dental models by using laser scanning technology, and performing accurate maxillary segmentation and equilibration using CASS technology. Further research is necessary to achieve these goals and to move the workflow directly from the CASS environment to the fabrication of custom-fitted prostheses, without requiring the surgeon to have "hands-on" involvement in the process (Fig. 29.17).

Table 29.3 Protocol comparison

New protocol for CTOS using CASS	Traditional protocol for CTOS preparation	Traditional protocol for CTOS intermediate and palatal splint fabrication
CT scan of entire mandible, maxilla, and TMJs (1-mm overlapping cuts) Processing of DICOM data to create a computer model in the CASS environment Correction of dentofacial deformity, including final positioning of the maxilla and mandible, with computer-simulated surgery STL model constructed with jaws in final position and sent to surgeon for condylectomy and rami and fossae recontouring if indicated Model sent to TMJ Concepts for prostheses design, blueprint, and wax-up Surgeon evaluation and approval using the Internet TMJ prostheses manufactured and sent to hospital for surgical implantation Acquisition of final dental models 2 weeks prior to surgery (2 maxillary, 1 or 2 mandibular models if dental, equilibrations are required); 1 maxillary model is segmented, and models equilibrated if indicated to maximize the occlusal fit; models sent to medical modeling Models incorporated into computer-simulated surgery for construction of intermediate and final palatal splints Surgeon receives models, splints, and printouts for computer-simulated surgery	CT scan, including the entire mandible, maxilla, and TMJs Fabrication of STL model with the mandible separated Surgeon positions the mandible in its final position and fixates it Removal of condyles and recontouring the lateral aspect of the rami and fossae if indicated Model sent to TMJ Concepts for prostheses design, blueprint, and wax-up Approval of total joint prostheses blueprint and wax-up by the surgeon Manufacture of custom-fitted total joint prostheses Prostheses sent to hospital for surgical implantation	Acquisition of dental models Mounting maxillary and mandibular dental models on an articulator Repositioning the mandibular dental model, duplicating the positional changes acquired on the STL model Fabrication of intermediate splint Repositioning maxillary dental models with segmentation if indicated Construction of palatal splint Ready for surgery

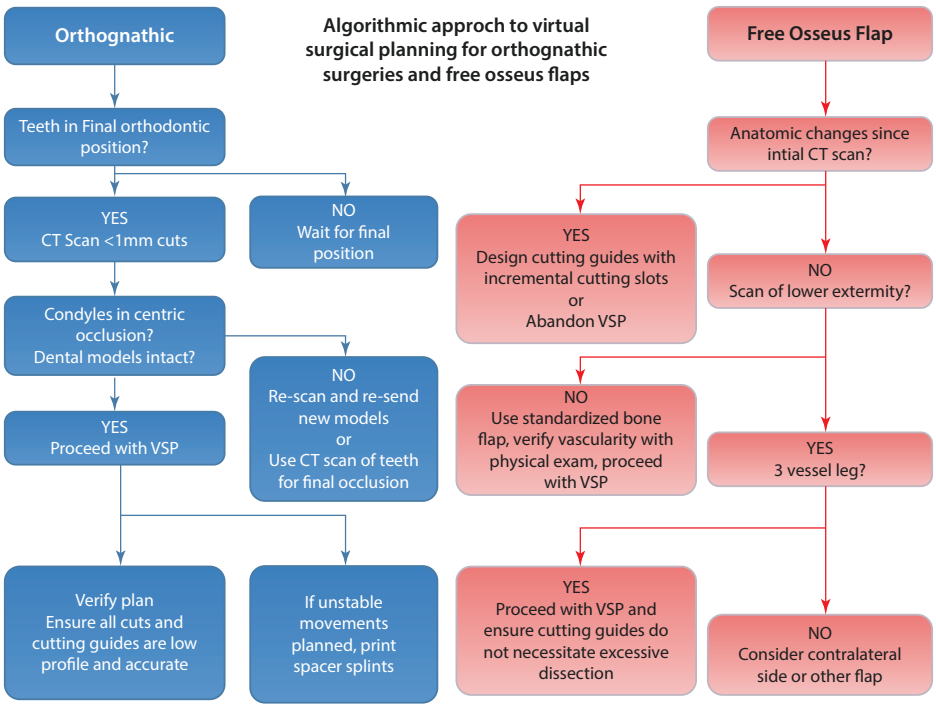

Fig. 29.17 One algorithmic approach to virtual surgical planning (VSP) by Efanov et al. (2018) [43] illustrating a stepwise approach for the planning of orthognathic cases (left) and an approach to free osseous flaps (right).

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Postoperative Management of the Maxillomandibular Advancement Patient

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Contents

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30.1 Introduction

Maxillomandibular advancement (MMA) is among the most successful surgical treatments for obstructive sleep apnea (OSA) [1–5]. Composed of a bilateral sagittal split osteotomy and a Le Fort I osteotomy, this procedure increases the resting tension of the pharynx by advancing the patient's skeletal framework, thereby decreasing nocturnal airway collapse and disruption of sleep [6–9]. Obstructive sleep apnea has multiple associated comorbidities that complicate the patient's preoperative, perioperative, and postoperative management [10–15].

Patient demographic for those undergoing maxillomandibular advancement for OSA differs greatly from that of the typical orthognathic patient. Patients are older, have a larger number of comorbidities, have a higher American Society of Anesthesiologists (ASA) classification, and have increased odds of postoperative complications [16]. The postoperative management of these complicated patients is outlined in this chapter to encompass strategies that reduce the risk associated with their chronic disease processes.

30.2 Preoperative Considerations

30.2.1 Surgical Facility

Hospital costs and reduced insurance reimbursement for surgical procedures have led to both a decrease in some hospital-based surgical interventions and a drive to increase outpatient/ambulatory surgery [17, 18]. Maxillomandibular advancement is among those surgeries that have declined in frequency due to financial constraints. Many surgeons currently perform outpatient orthognathic surgery for dentofacial deformities, and there has been little data supporting the need for inpatient surgery and postoperative hospitalization for most orthognathic surgery patients. Unfortunately, patients undergoing maxillomandibular advancement for obstructive sleep apnea are not among the patient population that can safely have outpatient surgery.

Patients with obstructive sleep apnea have a myriad of comorbidities that complicate their perioperative management. These comorbidities predispose patients to a higher rate of postoperative complications that may need to be managed in the immediate postoperative setting. Respiratory depression, metabolic syndrome, cardiac disease, blood pressure control, and difficulty in pain management are just a few of the considerations that require immediate and decisive management. Patients with moderate-to-severe sleep apnea undergoing MMA should be treated in a surgical facility with the appropriate personnel and equipment required to manage their possible complications [19]. It is recommended that all patients undergoing MMA should have at least an overnight observational period after surgery.

30.2.2 Medical Clearance

Patients with OSA often have a complicated medical history with many comorbidities either associated with or directly caused by their OSA. Patients may have refractory hypertension, pulmonary hypertension, coronary artery disease, cerebrovascular disease, previous myocardial infarction, metabolic syndrome, obesity, diabetes mellitus, endocrine disease, and/or kidney disease. Consultation with a primary care physician, preoperative anesthesia clinic, or appropriate specialists is warranted when severe or complicated comorbid conditions are present. Optimized control of comorbidities prior to surgery is paramount to reduce the risk of postoperative complications.

30.2.3 Anesthesia Considerations

The majority of closed claims associated with difficult airway management and extubation are caused by patients with either a difficult induction/intubation, obesity, or obstructive sleep apnea [20]. Patients requiring maxillomandibular advancement for obstructive sleep apnea are at high risk for having a difficult airway for intubation and maintaining a patent airway after extubation. A thorough preoperative oropharyngeal physical examination and screening should precede a candid preoperative conversation with the anesthesiologist in order to reduce the risk of postoperative desaturations, apneas, or anoxic injury.

Patients with obstructive sleep apnea and obesity will predictably have reduced functional residual capacity, redundant oropharyngeal tissue, and baseline hypercapnia that make oxygenation maintenance throughout the procedure more difficult. Techniques implemented during anesthesia to reduce the risk of postoperative complications may include intubation and extubation in a 25° head-up position, awake fiberoptic intubation, reduction in opiates intraoperatively, and postoperative oxygen delivery via nasal cannula [21–23]. Intraoperative administration of long-acting local analgesics can help the anesthesiologist reduce the opiate requirements of the patient postoperatively [24–27]. Discussion with the anesthesiologist prior to surgery can solidify preoperative plans for patient safety and prevent surprises for either the surgeon or the patient on the day of surgery.

Preoperative CPAP has been reported to reduce the risk of significant postoperative complications in patients undergoing hip or knee replacement [28]. Patients with OSA that received prophylactic CPAP prior to surgery had lower rates of intermittent desaturation, severe hypercapnia, witnessed apneas, unplanned ICU admissions, or severe cardiopulmonary complications. CPAP compliance prior to surgery may possibly confer protection in the postoperative setting. Postoperative CPAP is absolutely contraindicated in patients undergoing maxillomandibular advancement for OSA.

30.3 Inpatient Postoperative Management

30.3.1 Immediate Postoperative Course

After maxillomandibular advancement and any other planned surgical procedures, it is imperative that the surgeon maintains a presence in the operating room for extubation. Deep extubation of the OSA patient is contraindicated. Once the patient has shown evidence of purposeful movement, sustained a head lift ≥5 seconds, had full reversal of neuromuscular blockade with train-of-four assessment, and is breathing with adequate voluntary tidal volume, the patient is safe for extubation. Additional equipment for reintubation or emergent tracheostomy should be readily available. After the patient has demonstrated adequate oxygenation and sustained oxygen saturation, the patient may transfer to the post-anesthesia care unit.

Maxillomandibular surgery and other surgical treatments of the upper airway increase oropharyngeal edema and worsen airway obstruction in the first 24–48 hours [29]. This compounded with an approximately 2.5-fold increased risk of developing postoperative respiratory failure compared to controls makes postoperative monitoring of patients with OSA a high priority [30]. Continuous pulse oximetry should be used throughout the MMA patient's hospital stay. Admission to a floor with continuous pulse oximetry monitoring has been shown to decrease the number of rescue events in hospitalized patients and should be implemented [31].

There is no consensus as to whether a patient should be admitted to an Intensive Care Unit (ICU), step-down unit, or the general floor after MMA for OSA. General consensus with the anesthesiology department after extubation and the patient's ability to maintain oxygen saturation on minimal oxygen support should determine whether a patient should be admitted to a higher level of care. The typical patient with OSA treated by maxillomandibular advancement does not need an ICU level of care; however, the ultimate determination of the level of care a patient receives is dependent on the surgeon, the anesthesiologist, and the patient's immediate postoperative course.

30.3.2 Acute Pain Management

Pain management in both the immediate inpatient setting and in the outpatient setting should focus on decreasing the opiate requirements for adequate pain control. It is crucial to utilize a multimodal pain regimen that takes into account the patient's preoperative/postoperative pain levels, drug tolerance, and the need for the preservation of a strong respiratory drive. Often, patients with OSA have polypharmacy, obesity, and preoperative pain that make it difficult to address patient analgesia requirements while preserving respiratory function and drive. The use of multiple pharmacologic agents has become a necessity for safe pain management in this population.

Patients requiring maxillomandibular advancement for treatment of obstructive sleep apnea have a higher analgesic requirement when compared to patients requiring orthognathic surgery for dentofacial deformities [16]. Due to the significant pain relief opiate medications can provide, they are certainly appropriate for postoperative pain management when required; however, clinical judgement should be used as to the extent of their role (◘ Table 30.1). The combination of significant respiratory depression, a congested postoperative airway, and obesity increases the risk for severe hypoxia or apnea. Postoperative monitoring using continuous pulse oximetry, telemetry, and an attentive nursing staff is recommended until adequate pain management on non-opiate medications is achieved [32]. Opiate medications should primarily be used for "break-through" pain and weaned during the postoperative period. Nursing staff should be aware of the signs and symptoms of opiate overdose with particular attention to respiratory drive in patients with obstructive sleep apnea. In the event that a patient has a severe desaturation or apnea, nursing staff should be knowledgeable about the immediate reversal of opiate medications while always addressing the basic life support algorithm (◘ Table 30.2).

Opiate dependence has emerged as a severe debilitating disease over the past few decades. Shorter hospitalizations require the patient to manage their moderate-to-severe postoperative pain after the transition home. Although it is not unreasonable for prescribers to utilize opiate medications as part of a multimodal analgesia regimen, patients are at risk for long-term opiate dependence, respiratory depression, and driving impairment [33]. Physicians should utilize all modalities to decrease long-term opiate usage after MMA.

Table 30.1 Opiate analgesics for adult perioperative pain

Medication	Route(s) of administration	Dose (mg)	Onset of action (hr)	Half-life (hr)
Morphine	Intravenous Intramuscular Oral	2.5–15 10–15 30–60	0.25 0.3 0.5–1	2–3.5 3 3
Hydromorphone	Intravenous Intramuscular Oral	0.2–1.0 1–4 1–4	0.2–0.25 0.3–0.5 0.5–1	2–3 2–3 2–3
Fentanyl	Intravenous Transmucosal Transdermal	20–50 (μg) 200–1600 (μg) 12.5–100 (μg)	5–10 (min) 0.1–0.25 12–24	0.5–1 2–12 20–27
Oxymorphone	Intravenous Intramuscular Oral Subcutaneous	5–10 0.5–1.0 1–1.5 1–1.5	0.5 0.15 0.15 0.15	3.3–4.5 3–5 3–5 3–5
Codeine	Oral	15–60	0.25–1	4
Hydrocodone	Oral	5–7.5	0.5	2–3
Oxycodone	Oral	5	0.5	3–5
Tramadol[a]	Intravenous Oral	50–100 (400 Max/D) 50–100 (400 Max/D)	0.5–1 0.5–1	4–6 5–6

[a]Tramdol is not classified as an opiate by the FDA

Table 30.2 Reversal medications and how to administer them effectively

Indication	Medication	Route of administration	Dose and frequency	Max dose	Duration of action
Opiate overdose	Naloxone	Intravenous	0.1 mg every 2–3 min	0.8 mg	30–60 min
Benzodiazepine overdose	Flumazenil	Intravenous	0.1–0.2 mg every 2–3 min	1 mg	45 min (dose dependent)

There is a wide variety of non-opiate medications available for long-term pain control [34] (Table 30.3). Acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDS), beta-blockers, local anesthetic, and CNS neurotransmitter modulators are among the most commonly used for long-term treatment of pain without significant abuse potential [35, 36]. Decreasing the use of opiate medications helps minimize the risk of postoperative nausea/vomiting (PONV), respiratory depression, constipation, and urinary retention caused by their use. Preoperative gabapentin [37] or Cox-2 inhibitors [38] and intraoperative administration of ketamine, ketorolac [39], beta-blockers [40], and liposomal local anesthetics [24–27] help to diminish postoperative opiate requirements and increase long-term pain control.

In the Post-Anesthesia Care Unit (PACU), a combination of ketorolac, intravenous acetaminophen (Ofirmev), and judicious opiate administration (morphine, fentanyl, hydromorphone) can be used when the patient cannot tolerate oral medications. PACU nurses should be educated on decreased use of opioid analgesia in lieu of multimodal pain regimens. After the patient can tolerate oral medications, oral acetaminophen, NSAIDs, gabapentin, and any preoperative pain regimens the patient found useful prior to surgery can be utilized while opiate medications are weaned. Drug combinations can be used to increase effectiveness. In particular, acetaminophen and ibuprofen administered concomitantly have been reported to have synergistic advantage over either drug alone [41].

Unless contraindicated, multimodal anesthesia and analgesia should be the standard in OSA patients after MMA. An established analgesic routine will help to

Table 30.3 Non-opiate analgesics for adult perioperative pain

Medication	Route(s) of administration	Dose (mg)	Dose frequency (hr)	Max daily dose (mg)	Onset of action (hr)	Duration of action (hr)
Acetaminophen	Oral Suppository Intravenous	500–1000 650 650–1000[a]	4–6 4–6 4–6	4000 4000 4000	1–2 1–2 5–10 (min)	4–6 3–4 4–6
Ibuprofen	Oral Intravenous	400–600 400–800	4–6 4–6	2400 3200	0.5–1 <2	4–6 4–6
Naproxen	Oral	500 + 250[c]	6–8	1500	0.5–1	8–12
Ketoprofen	Oral	25–50	6–8	300	<0.5	<6
Ketorolac[d]	Oral Intramuscular Intravenous	20 + 10 30–60 30 + 15–30	4–6 6 4–6	40 150 Day 1, 120 Day 2–5	0.5–1 5–10 (min) 5–10 (min)	6–8 6–8 6–8
Diclofenac	Oral Intravenous	50 37.5	8–12 6	150 150	0.5–1 5–10 (min)	6–8 Unk
Meloxicam	Oral	7.5–12	24	15	0.5–1	24
Celecoxib	Oral	400 + 100–200[b]	12–24	400	1–2	8–12
Gabapentin	Oral	1200–3600	8	3600	2–4	8

[a]≥50 kg: 650 mg IV every 4 hours; 1000 mg IV every 6 hours. Maximum single dose: 1000 mg IV
[b]400 mg PO loading dose followed by 200 mg PO on day 1. 100–200 mg PO twice a day for maintenance
[c]500 mg PO loading dose followed by 250 mg PO every 6–8 hours OR 500 mg PO every 12 hours
[d]Ketorolac maximum dosage for the first day is 150 mg IV or IM. Maximum dosage for the next 5 days is 120 mg IV or IM. Maximum number of days for PO/IM/IV ketorolac is 5 days due to GI adverse effects. PO 20 mg loading dose followed by 10 mg PO every 6–8 hours. IV 30 mg loading dose followed by 15–30 mg IV q4–6 hours. Only one route of administration should be utilized at any given time

decrease length of stay, postoperative complications, and increase patient satisfaction.

30.3.3 Postoperative Nausea and Vomiting

Postoperative nausea and vomiting (PONV) is a significant concern for patients after maxillomandibular advancement for obstructive sleep apnea. It can increase length of hospitalization, increase duration of PACU admission, cause significant morbidity through aspiration, and increase healthcare costs. After any surgical procedure, the incidence of postoperative nausea is about 50% and the incidence of postoperative vomiting is about 30% [42, 43]. Data on PONV following orthognathic surgery report similar rates with the incidence of postoperative nausea at 67% and postoperative vomiting at 27% [44].

Numerous independent risk factors have been indicated as significant predictors for PONV (▶ Box 30.1).

Box 30.1 Risk factors for PONV in adults
Female
Nonsmoker
History of PONV, motion sickness, or PONV in relatives
Opiate administration during or after surgery
Prolonged surgery >60 minutes
Surgery on the maxilla

There are conflicting data on whether a maxillary osteotomy will increase the risk of PONV compared to mandibular osteotomy alone. *Silva et al* reported an incidence of 40% PONV for orthognathic surgery but with an increase in PONV to 57% with a maxillary osteotomy [45]. Phillips et al reported no significant difference with the addition of the Le Fort osteotomy; however, there was a slight increase in PONV in those

patients with a Le Fort I osteotomy with or without a mandibular osteotomy [44].

Prevention of postoperative nausea and vomiting begins with delineation of patient risk. Many guidelines exist to help the surgeon not only determine the risk of PONV but also determine how aggressive the antiemetic prophylaxis and treatment should be [42, 43]. In general, patients with one or more risk factors should have at least one antiemetic. In patients with moderate-to-severe risk of PONV, two or more antiemetic prophylaxis agents should be used to obtain a synergistic effect [46]. Multimodal PONV treatment relies on both pharmacologic and non-pharmacologic treatment modalities. The pharmacologic antiemetics available for PONV prophylaxis include $5HT_3$ receptor antagonists, NK-11 receptor antagonists, butyrophenones, antihistamines, anticholinergics, and phenothiazines [47, 48]. Non-pharmacologic PONV prophylaxis may include adequate IV fluid hydration or the use of regional anesthetic to decrease peri- and postoperative opiate use (Table 30.4).

After MMA for OSA, it is common to keep a nasogastric tube in place after surgery for decompression of the stomach contents in an attempt to reduce PONV. Unfortunately, this has not been shown effective in the literature [49, 50] Following a maxillary osteotomy, there is inevitably postnasal blood that will be ingested. Nasogastric tube retention after surgery may be a low-cost, low-risk intervention to provide comfort for patients with severe PONV but not a predictable reduction in emesis.

30.3.4 Swelling and Edema

After maxillomandibular advancement, patients may have significant postoperative facial edema. The typical course of swelling in these patients reaches its peak between 3 and 5 days after surgery; however, noticeable reduction in facial swelling to the patient can be up to 6 months and a noticeable reduction in facial swelling to the surgeon can take up to 1 year after surgery [51].

Multiple methods exist to reduce the degree of postoperative edema. The non-pharmacologic management of patients after MMA includes head-of-bed elevation to 30 degrees, cryotherapy, and postoperative head dressings. This variety of management has largely not been proven in the literature; however, pharmacologic therapy with perioperative and/or postoperative corticosteroids has been shown to reduce facial edema [52–54]. Eight mg of dexamethasone at induction of anesthesia and another 8 mg every 8 hours for two additional doses are an effective treatment regimen for the prevention of significant postoperative swelling.

30.3.5 DVT Prophylaxis

Venous thromboembolism (VTE) is a common and preventable cause of death in many surgical patients. Orthognathic surgery in general is a low-risk procedure for development of deep venous thrombosis (DVT) and resulting thromboembolism; however, there are many systemic factors that can increase patient risk. VTE pro-

Table 30.4 Antiemetic medications for adult postoperative nausea and vomiting

Medication	Route(s) of administration	Dose (mg)	Dose frequency (hr)	Onset of action (min)	Duration of action (hr)
Promethazine	Intravenous Oral Suppository	12.5–25 12.5–25 12.5	4–6 4–6 4–6	5 20 20	4–12 4–12 4–12
Ondansetron	Intravenous Oral dissolving tablet	4 8	4–6 4–6	30 30	4–8 4–8
Droperidol	Intravenous	0.625–1.25	4–6	10	2–4
Dexamethasone	Intravenous	4–8[a]	Once at induction	–	–
Scopolamine[b]	Transdermal patch	1.5	Once prior to induction	240	72 hours

[a]Intravenous dexamethasone, as well as other corticosteroids, have been found to decrease both PONV and pain. Emerging data show that an induction dose of 8 mg may be more beneficial than the 4-mg dose [65]

[b]Scopolamine transdermal patch can cause distressing pupillary dilation due to the topical anticholinergic effect on the ciliary reflex. Patients should be instructed not to touch the patch and to wash their hands if they have touched the patch

Table 30.5 Postoperative venous thromboembolism prophylaxis recommendations

Surgical risk groups	Caprini score for plastic and reconstructive surgery	Estimated VTE risk without prophylaxis	Recommended VTE prophylaxis
Very low risk	0–2	< 0.5%	Early ambulation
Low risk	3–4	Approximately 1.5%	Mechanical prophylaxis until ambulation
Moderate risk	5–6	Approximately 3%	Chemoprophylaxis
High risk	≥7	Approximately 6%	Chemoprophylaxis

Care must be taken in selecting appropriate chemoprophylaxis [55]

phylaxis recommendations use a combination of systemic factors, based on the Caprini Score for risk stratification, and the region of surgical intervention [55].

VTE prophylaxis can be achieved with early ambulation, mechanical prophylaxis (intermittent pneumatic compression), or chemoprophylaxis (low-dose unfractionated heparin, low molecular weight heparins, vitamin K antagonists, direct thrombin inhibitors, or factor Xa inhibitors) [56]. Patients that undergo head and neck reconstructive surgery have a baseline reduced risk of VTE compared to general surgery or pelvic surgery, thus typically requiring only early ambulation as their primary prophylaxis. The current recommendations for VTE prophylaxis, both chemoprophylaxis and mechanical prophylaxis, are outlined in Table 30.5.

Patients undergoing maxillomandibular advancement for obstructive sleep apnea may have a multitude of comorbidities that increase the risk of DVT and VTE. These risk factors must be taken into consideration with the patient's ability for early ambulation. Mechanical VTE prophylaxis is often all that is necessary until ambulation.

30.3.6 Nutrition

Nutritional status for optimal wound healing may be compromised after maxillomandibular advancement due to postoperative maxillomandibular fixation, postoperative orofacial pain, an instrumented airway, and presurgical malnutrition. Malnutrition has been associated with higher rates of surgical site infections, longer hospital stays, poor functional status after discharge, and increased hospital costs [57]. The daily energy requirements for a healthy adult is 30–35 kcal/kg of body weight per day; however, after MMA, patients have increased metabolic needs for the healing process and may require in excess of 40 kcal/kg of body weight per day [58]. In order to optimize wound healing, there are a variety of options available to increase caloric intake for these patients.

Preoperative nutritional assessment should be a part of any surgical workup. Prior to surgery, patient nutrition should be optimized to afford maximum wound healing potential. Preoperative nutritional supplementation for orthognathic surgery has been explored in order to reduce complications and increase postoperative healing but with no significant difference in outcomes [59]. Nutritional intake after orthognathic surgery without supplementation can be anywhere from 33% to 52% lower than preoperative intake [60]. Postoperative nutritional supplementation with high caloric and high proteinaceous content helps to improve wound healing and decrease postoperative complications.

30.3.7 Antibiotics

Postoperative antibiotics following maxillomandibular advancement begins with preoperative intravenous antibiotics and follows with a 5-day course of oral antibiotics. Typical antibiotic regimens during the perioperative period are weight-based cephazolin or clindamycin 1 hour to 15 minutes prior to incision. Following surgery, 5 days of postoperative antibiotics has been reported to lower surgical site infections to <1%. Additional oral antibiotic administration beyond 5 days has not been shown to add benefit [61–63].

30.4 Outpatient Postoperative Management

30.4.1 Follow-Up Regimen

Patients should follow a 1- to 2-week follow-up schedule for the first 6 weeks. Follow-up may then be expanded to a 6-month and 1-year follow-up as indicated. Postoperative polysomnography should be obtained to document postoperative treatment or postoperative cure

of the obstructive disease. These results can then be used to determine postoperatice conservative therapy if postoperative cure is not achieved.

30.4.2 Postoperative Occlusal Guidance

Postoperative occlusion may not coincide with presurgical planning and adjustments must be made to guide the patient back to the desired centric occlusion. In patients that require adjustment to their occlusion, elastic bands providing approximately 3.5–6 oz. of tension may be used to correct simple malocclusions. Many patients undergoing MMA for OSA do not have preoperative orthodontics and archbars are required for placement of the maxillomandibular complex into both intermediate and final splints. These archbars can be used to attach elastic bands to guide the jaws into final occlusion. In maxillomandibular advancement, the Le Fort I osteotomy is the site with the weakest fixation and care must be taken to avoid aggressive force on the maxillary fixation.

The orientation of elastics depends greatly on the type of malocclusion and the timing of malocclusion. In patients with immediate postoperative posterior open bite, simple posterior vertical elastics can help to compensate for decreased muscle tension and maximize occlusal contacts. However, if there is a midline shift, elastic bands can be used to correct the midlines and make them coincident.

Elastic bands may be worn from the time of surgery to the 1-week follow-up appointment at which time their need may be assessed. If elastic bands are deemed necessary to correct malocclusions, they should be changed at least once daily and removed during meals [64]. Discontinuation of elastics will ultimately depend on if the causative factor is postoperative edema, tooth malocclusion, or error in surgical technique. Thorough investigation as to the cause of the patient's malocclusion should begin if elastics are required longer than 1 month postoperatively.

30.5 Conclusion

There are many similarities in the postoperative management of maxillomandibular advancement for obstructive sleep apnea and for dentofacial deformities. Obstructive sleep apnea increases the postoperative risks of desaturation, postoperative pain, and increased pharmacologic requirements. Care should be taken to address these possible complications before they arise with a well-prepared team. When the patient, anesthesiologist, post-anesthesia care unit, floor nurses, and surgeon work in concert, the patient will most certainly have a reduction in postoperative risk and increased satisfaction.

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Post-surgical Myofunctional Therapy and Physical Therapy

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Contents

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Core Message

Rehabilitation by a skilled team of professionals is critical to optimizing long-term success and can significantly improve airway patency, functional, orthodontic, and aesthetic outcomes after surgery. This book chapter provides an introductory overview to help the practitioner appreciate the role of pre- and postoperative therapies in the care of patients who undergo orthognathic surgery for treatment of sleep apnea.

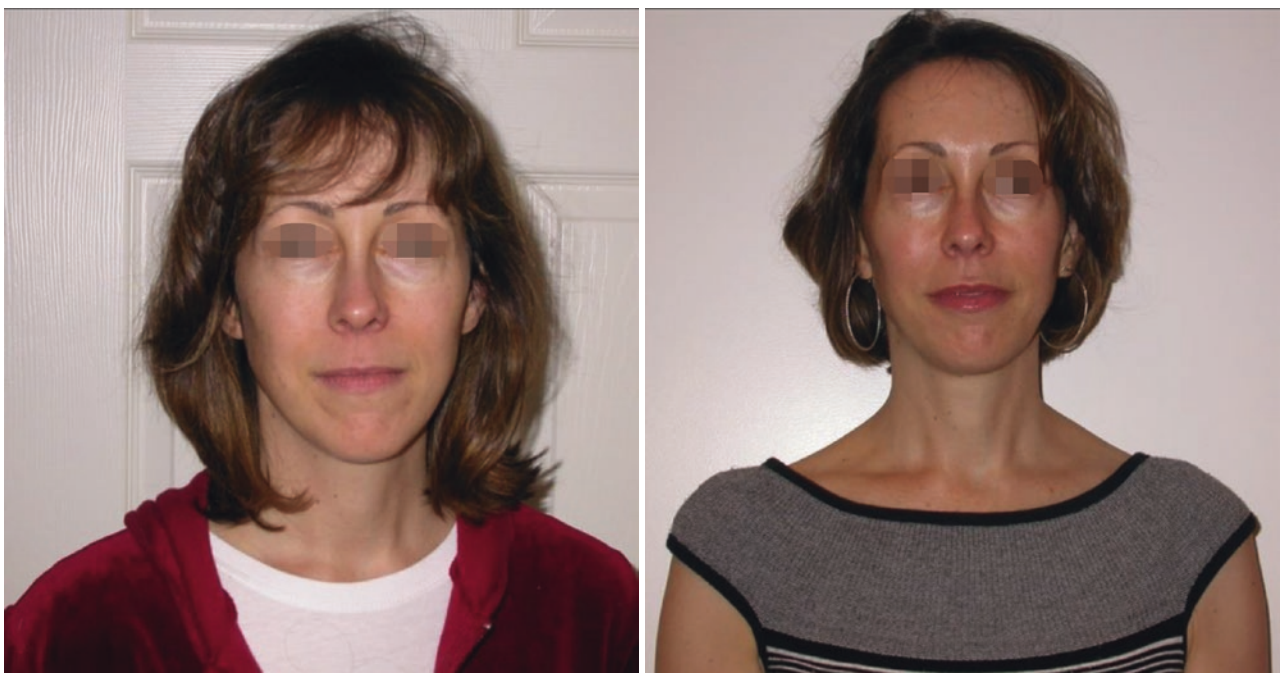
Maxillomandibular advancement (MMA) is a highly effective surgical option for obstructive sleep apnea (OSA) that achieves enlargement of the upper airway by physically expanding the facial skeletal framework [1, 2]. After surgery, however, patients may develop swelling, facial numbness, oral incompetence, drooling, difficulty with mouth opening, and nasal obstruction [3]. Myofunctional and physical therapy can be used before surgery to identify and eliminate potential barriers to successful outcomes, work to identify hurtful habits, educate and empower the patient, and begin to establish healthy habits, postures, and movement patterns. Post-surgical therapy helps maximize the benefits of surgery by restoring motion, strength, and establishing healthy postures, habits, and normal oral and masticatory function, as well as to re-pattern the stomatognathic functions of the oral facial muscles [4] which can improve long-term outcomes (see ◘ Fig. 31.1).

Myofunctional therapy is the neurologic re-education of the oral and facial muscles to promote healthy orofacial habits. It is a rehabilitation therapy program that focuses on optimizing chewing, swallowing, orofacial posture, and nasal breathing. Restoration of these basic functions is important to maximize patient satisfaction, long-term success, and quality of life after jaw surgery. In the United States, a postgraduate course in myofunctional therapy is taught to medical doctors, dentists, dental hygienists, physical therapists, speech language pathologists, occupational therapists, and osteopaths. It will be important that professionals on the rehabilitation team work within their scope of practice but are trained in and working toward the healthy functions now referred to in the research as myofunctional therapy.

Physical therapy for the jaw can include myofunctional therapy with proper postgraduate training, and can optimize surgical results by providing skilled manual, neuromuscular and myofascial techniques and customized treatments, training and therapeutic techniques to decrease pain and inflammation, restore mobility, stability, strength and function of the joints, nerves and muscles, improve posture and alignment of the spine and cranium, and a healthy resting position of the mandible.

The rehabilitation team should work collaboratively with the surgeon to develop and implement mutually agreed upon patient care protocols. Modalities used may vary as outlined by surgeon preference, therapist experience, and each state's professional practice act.

A preoperative assessment is performed prior to surgery to evaluate for the presence of tongue thrust, ankyloglossia, poor oral rest posture, and other signs of



◘ **Fig. 31.1** Myofunctional therapy as adjunct to surgery: improved facial aesthetics, symmetry, and posture

Fig. 31.1 (continued)

orofacial myofunctional disorders as well as postural dysfunction, hypermobility and mal-alignment, and bone, joint, muscle, fascia, or nervous system dysfunctions that may need to be addressed or taken into consideration. Patients are taught to:

- Practice nasal breathing day and night
- Develop a lip seal
- Achieve and maintain palatal tongue rest posture
- Practice bilateral chewing (with clearance from the oral surgeon) and healthy swallow
- Maintain healthy balanced postures, including sleep and eating posture
- Avoid hurtful habits and parafunctions by
  - Keeping hands and objects away from the face
  - Eliminating lip biting and clenching teeth

Indeed, constant pressure on the teeth from the tongue due to poor oral rest posture and/or tongue thrusting during swallow may have a dramatic effect on the dental occlusion and alignment of teeth within the mouth [4].

To maximize surgical results and minimize relapse, it is essential to address the dysfunctions that likely contributed to the need for surgical intervention in the first place. Ideally, pre-surgical therapy including myofunctional therapy is initiated two to three months prior to surgery to create a foundation for the post-surgical long-term phase of care. Post-surgical rehabilitation that includes a sequential series of therapeutic techniques described as myofunctional therapy is then essential to optimize the results of surgery and avoid relapse (see Fig. 31.2).

Post-surgical therapy aims to help rehabilitate the patient in both the acute and long-term phases of recovery after jaw surgery. In the acute setting, the objectives of postoperative therapy may include the following:

- Improve facial sensation, proprioception, and neural networks
- Reinforce nasal saline rinses and encourage nasal breathing
- Encourage proper swallowing, chewing, and feeding techniques

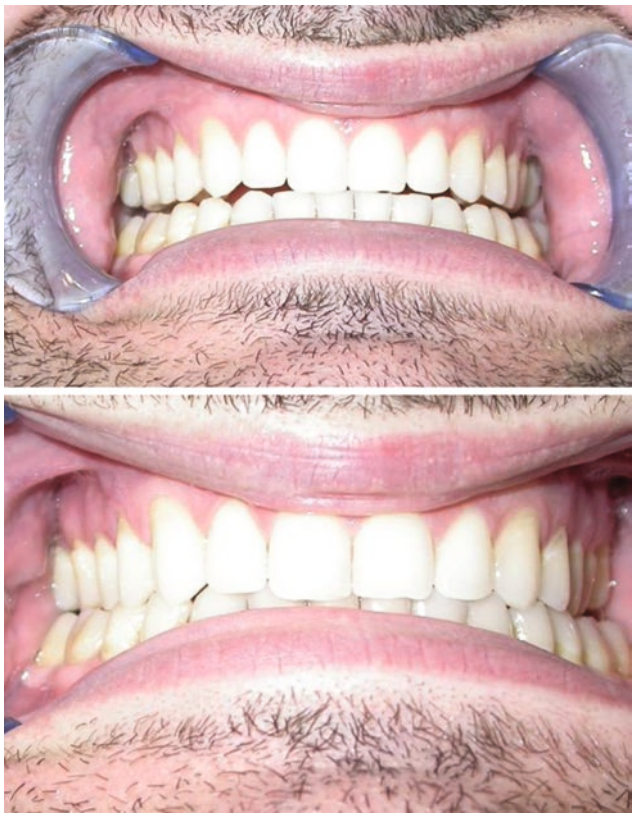


Fig. 31.2 Orthodontic relapse after jaw surgery treated with myofunctional therapy via retraining of the orofacial muscles and improving tongue resting posture

- Avoid maladaptive orofacial habits (touching the face, resting the jaw on the hands, applying asymmetric pressure to the surgical osteotomy and fixation site)
- Improve the resting position of the tongue by isolating and activating different tongue muscles
- Promote stabilization of the mandible and avoid over-opening
- Teach symmetrical muscle patterns that promote a stable orthodontic result
- Establish healthy sleep habits and hygiene

Dental hygienists practicing myofunctional therapy are also able to provide oral care for the patient to reduce inflammation in the tissues surrounding the oral appliances using various rinses such as chlorhexidine and fluorides with soft brushes. Nutritional counseling as to the consistency of foods and identification of foods with a high value of nutrients is valuable to assist the patient into optimal healing. Because of the hygienists' knowledge of dentistry, nutrition, and occlusion, they can communicate between the orthodontist and oral surgeon effectively. They can support and motivate the patient to think positively about the result while in the healing phase and may decrease patient's anxiety.

Speech and language pathologists (SLPs) can be helpful in assisting patients before and after any surgery to the face, mouth, and often to the nose and the pharynx. By applying diagnostic principles and myofunctional therapy techniques, SLPs address any impact the surgery may have on oral functions such as chewing, swallowing, and resting position of tongue and lips. In addition, SLPs may provide patients with nasal breathing techniques, as well as voice and speech articulation therapy when needed as they may be impacted by surgery.

Chewing functions are always disrupted by MMA surgery and quite often patients are left to recover chewing spontaneously and they are recommended to eat soft foods, which is as necessary an approach as avoiding load bearing on a fractured leg. However, most patients do not receive formal instructions on how to properly recover chewing with the unfortunate result of being on soft diet longer than is necessary. It would be akin to having a broken leg and never receive physical therapy to recover proper gait.

Chewing involves many neuro-muscular-occlusal circuits which surgery usually disrupts. Therapy techniques to normalize chewing might include chewing on silicon wafers or tubes or sticks (sham chewing), which allow a balanced and gradable activation of the buccinators, masseters, and temporalis muscles, which can also be toned before surgery and then re-patterned post-surgery.

Since, in the very beginning, just after significant MMA surgery, most meals are to be consumed in liquid form, a proper activation of the buccinators and the orbicularis oris may promote a more efficient sucking through straws or from spoons. Toning and strengthening these muscles prior to surgery can assist with post-surgery recovery.

Nasal breathing is fundamental to preserve a proper lip seal but often, even after the rhino-maxillary surgery provided the physical space for better breathing, nasal breathing is still not spontaneous because nasal breathing is a complex neuro-chemical-muscular activity that needs to be taught or taught again after extensive surgery, as the neuro-sensory parameters changed. Moreover, breathing is involved with voice, not just as the flow of air creates sounds but also as the oronasal cavities create resonance.

Surgery involving the soft palate, like uvulo-palato-pharyngo-plasty (UPPP), often creates a temporary but significant loss of air through the nose during speech, so that sounds that require a build-up of oral pressure, such as /p/, /b/, /t/, /d/, /k/, or /g/, cannot be properly produced as the air escapes through the nose because the soft palate is not isolating properly the various cavities. SLPs, through specific exercises can assist the patient in recovering the functionality of the soft palate.

Although speech-language pathologists, physical therapists, and dental hygienists may take care of patients from birth to old age, for patients who undergo MMA or other sleep surgery, they may still need to receive postgraduate training in myofunctional therapy.

31.1 Paradigm

Myofunctional therapy exercises should be individually customized and performed under the guidance of a professional trained in orofacial myofunctional therapy and who has had extra training as to habit awareness and elimination, breathing education, and proper pre- and post-orthognathic goals. In general, the post-orthognathic surgery paradigm includes the following elements and practice exercises.

31.2 Preoperative

1. Baseline assessments are acquired. Measurements of tongue and lip strength, rest posture of the tongue, ability of the patient to breathe through their nose, history of habits, oral facial pain symptoms, tongue and orbicularis oris restricted tissues, swallowing and chewing difficulties, facial symmetry, diet survey, functional speech and posture problems, photos and videos, palate width, Mallampati/Friedman scores, and screening for abnormal tissues.
2. Begin teaching principles and practice of proper orofacial rest posture, tongue placement, bilateral chewing, proper swallowing, and begin nasal breathing education with diaphragmatic muscle activation, reduced, relaxed breathing as much as the patient is able, nasal hygiene, isolation and activation of the tongue muscles, range of motion of the lips, buccinators, and zygomatic muscles, and reviewing functional posture therapeutic techniques.
3. If ankyloglossia is present, the patient should be enrolled in pre-frenuloplasty myofunctional therapy, undergo surgical release of the lingual frenulum, and then complete post-frenuloplasty exercises for 4–6 weeks before embarking on maxillary mandibular advancement surgery.
4. Myofunctional therapy exercises should be STOPPED prior to dental impressions and CT scan for virtual surgical planning. The exercises prescribed may in fact significantly affect the alignment of the teeth and thereby affect the accuracy of intraoperative surgical splints.
5. Physical therapy to identify myofascial restrictions, and work to improve asymmetries of the cranium, mandible, spine, or pelvis that could adversely impact surgical outcomes.
6. Identify and establish a plan to eliminate postural dysfunction and hurtful habits with the appropriate manual techniques, training, or interventions.
7. Fascia, scar, and injury assessment.

31.3 Acute Post-surgical

Techniques are individualized and are specific to each patient's functions. These are a few of many techniques used:

1. Brush (use feather, soft brushes, or washrag) to stimulate cheeks, lips, and tongue (5 minutes, 3x/day).
2. Puff air into cheeks. Count to 5 on 4 sides (five times, 3x/day).
3. Range-of-motion exercises. Say "EEO OOH AHH" (fifteen times, 3x/day).
4. Tube chew (gently), start with 30 seconds and work up to 2 minutes with lightweight surgical tubes cut into two-inch lengths. Encourage bilateral chewing. Re-engage the muscles of mastication and avoid atrophy of the muscles.
5. Power pucker (20 times, 3x/day). Close lips, lip seal, suck back and release. This may assist in controlling excess saliva.
6. Diet: Increase textures slowly. Start with thin liquids, then thick liquids, then soft food, increase up to more solid.
7. Follow modalities and techniques to decrease pain and inflammation.
8. Limit range of motion to rotation, avoiding translation (Richardson JK reference).
9. Intraoral massage and myofascial release.
10. Nerve desensitization and re-education.
11. Scar tissue management and mobilization.
12. Exercise sequential progressions to improve function, strength, and stability.
13. Emphasize on healthy sleep hygiene and habits.
14. Postural activities and self-cervical mobility exercises.
15. If jaw joint replacement was performed, the patient and therapist must work together to assist the patient to be cognizant of lateral movements with both normal functions and the exercise protocols. Many patients/therapists are not aware of habits and movements that could cause discomfort in the muscles surrounding the joints.

31.4 Long-Term Post-surgical

Habituation of these healthy habits and movement patterns will be essential to prevent relapse and return to those dysfunctions that contributed to the need for surgery (see Fig. 31.3). Multidisciplinary approach to

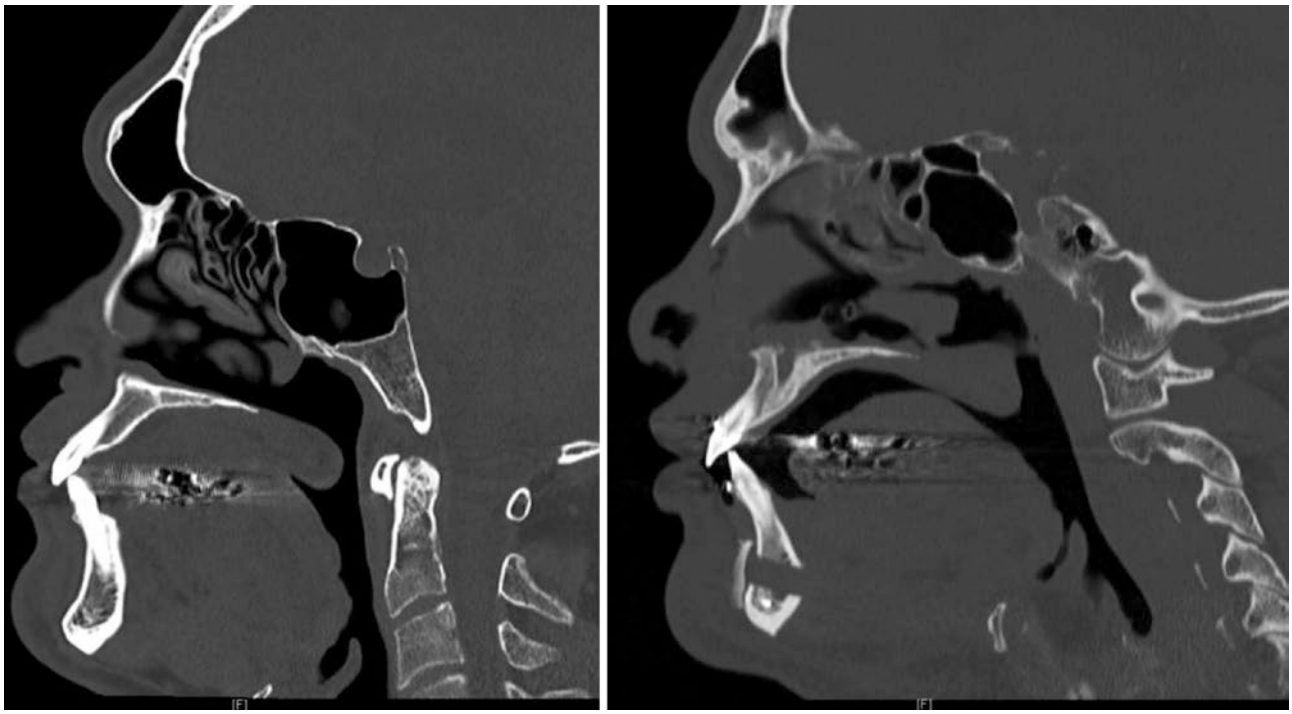


Fig. 31.3 CT scan before and after MMA + GTA surgery showing improper tongue resting positioning. There is now more room for the tongue with the enlarged facial skeleton framework, but the tongue relapses to the preoperative position with a propensity for posterior inferior displacement. Myofunctional therapy will help him learn to position his tongue anteriorly and superiorly to rest against the hard palate

management that includes myofunctional therapy, speech therapy, and physical therapy can alleviate the burden of managing postoperative complications from the surgery and significantly increase patient outcomes and satisfaction. Optimal results are achieved when the therapy is initiated 3–4 months (depending on the surgeon's preference) preoperatively and continued for at least 6 months and ideally 1 year after surgery.

31.5 Other Benefits of Myofunctional Therapy

Recurrent or persistent OSA after maxillary mandibular advancement surgery can be frustrating for both patients and practitioners alike. OSA relapse after maxillary mandibular advancement surgery is not uncommon. Among those patients who have been cured of sleep apnea with jaw surgery, recurrent OSA has been reported even 10–15 years after surgery [1]. The most predominant site of obstruction among these patients with persistent or recurrent OSA is the tongue-base [5]. Myofunctional therapy is an effective adjunct to sleep apnea surgery that has been shown to further reduce the AHI by approximately 50% in adults and 62% in children. Myofunctional therapy rehabilitates the tone of the genioglossus muscle to prevent tongue-base collapse [6] and promotes closed-mouth nasal breathing [7]. It is a highly effective, noninvasive, and much safer alternative to other potential interventions (such as tongue-base reduction and tongue-base suspension) for patients with persistent OSA after MMA surgery, especially among those with upper airway resistance and/or mild-to-moderate sleep apnea.

31.6 Who Are Myofunctional Therapists and Where Can One Find a Well-Trained Therapist?

In different countries, different professionals have taken postgraduate training. In the United States, dental hygienists, physical therapists, osteopathic physicians, occupational therapists, or speech pathologists are likely candidates for the postgraduate training. Surgeons in the United States could encourage physical therapists they work with to do additional training in myofunctional therapy. In Brazil, it is mostly done by speech pathologists. In Japan, many dental hygienists have received the training. In other countries, it may be a combination of dental professionals, physical therapists, or speech pathologists.

The relationship between therapist and patient is the one in which trust, support, and motivation are all intertwined so the patient feels they have their concerns addressed and are proactive about getting back into "shape" (procedures that a doctor might not have time for). Along with ongoing physiologic and emotional support and sleep hygiene, the therapist is the linchpin for the best possible surgical outcome/result, especially in the long term.

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Complications Associated with Maxillomandibular Advancement

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Contents

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Overview

Complications of maxillomandibular advancement (MMA) can occur at any time during the diagnosis and treatment of obstructive sleep apnea (OSA) and throughout the treatment continuum, including the preoperative periods of treatment planning, perioperative orthodontic care, intraoperatively and postoperatively. MMA has become a well-established orthognathic procedure with a relatively low rate of major complications at roughly 1%, according to the 2010 systematic review and meta-analysis of 22 MMA studies by Holty and Guilleminault, who also reported that there were relatively few minor complications over the course of a mean postoperative follow-up of 5 months (range, 3 to 7.7) [1]. Notably, they found older patients and individuals with the presence of one or more preoperative comorbidities at higher risk of complications [1].

Some postoperative complications are so common that they are expected to accompany MMA, including the daily challenges of breathing and eating, as well as low mood and some satisfaction concerns [2]. While orthodontic treatment and orthognathic surgery are intended to improve the patient's appearance, not all patients are able to sufficiently adapt to the challenges from undergoing a major surgery with an extended follow-up healing period. Despite a successful procedure, low mood and patient satisfaction are expected challenges that should be worked through actively by the surgeon and patient. Like other major surgical procedures, recovery from MMA requires days of inpatient hospital time and potentially several months of healing and recuperation. MMA patients in particular require a mean of 3.5 days of in-hospital care following the procedure, and most patients typically return to their normal functional status between 2 and 10 weeks [3].

The incidence of various complications, however, may increase inpatient hospital times, delay, or severely compromise healing and rehabilitation, and postpone the return to regular activities, particularly if one or multiple reinterventions are required. Any number of problems, such as hardware/device failure, incidence of infection, and/or adverse drug reactions, may also complicate recovery and significantly hamper quality of life (QOL), disparage the patient's outlook for a full recovery, and reduce satisfaction of the both patient and provider. Complications must therefore be carefully considered and discussed together as a patient–provider alliance, and similarly prepared for together prior to the procedure to prevent their occurrence or—if complications do occur—to mitigate and manage these events to the best of the ability of both patient–provider team.

32.1 Collaborative Prevention and Management of Complications

32.1.1 First Complications Reported in the 1970s

One of the first major complications of orthognathic surgery was reported in 1979 by Piecuch and West [4]. They reported a spontaneous pneumomediastinum in a 24-year-old male surgically treated for maxillary hyperplasia and mandibular hypoplasia. The man received a total maxillary alveolar osteotomy with expansion as well as bilateral mandibular sagittal osteotomies. Two days later, signs of pneumomediastinum appeared in the form of sharp, non-radiating anterior chest pain without dyspnea. Chest X-ray revealed clear lung fields and electrocardiogram was normal, but the cardiac outline was double density, and appeared to be an air shadow that included the great vessels. Pneumomediastinum was diagnosed and surmised to be attributable to a spontaneous rupture of an alveolus and not related to the specific intervention or use of a respirator during the procedure. Although spontaneous pneumomediastinum has proven to be an extremely rare complication during orthognathic or other major head and neck surgeries, this early case highlights the general risk of spontaneous complications requiring systematic vigilance in complication prevention and aggressive treatment when they occur.

Since then, orthognathic surgery has become more widely accepted over time while it has simultaneously benefitted from innovations in imaging and planning both before and during procedures, and greater capacity for data-intensive 3D computer-assisted surgical techniques, developments in various biomaterials, improved surgical design and quality of instrumentation in bone cutting and fixation, significantly enhanced precision, and superior procedural strategies and approaches, all driven by the improved training and skill of both surgeon and staff [5]. By 2012, there were 14 papers focused on describing complications, and currently, dozens more, though it is believed that complications are underreported [6].

32.1.2 Effective Provider–Patient Risk Communication and Shared Decision-Making

It is critical that each surgeon approach complications and their management seriously and systematically (with multidisciplinary help, as needed). It is also crucial to establish a strong therapeutic alliance where patients are apt to play an active role in their care [7]. This facili-

tates an open discussion of the risks and challenges the therapeutic alliance faces throughout the care continuum and is meant to equip patients and their families with the knowledge of measures they have in their power to most effectively prevent and/or minimize complications. Empowerment is an important predictive variable and foundational to the therapeutic alliance, ultimately strongly influencing patient satisfaction [8]. A highly communicative, participatory working relationship in which the patient is empowered to take an active role of their own care can also facilitate better health outcomes, which cultivates mutual respect and gratitude, and may play a role in decreasing potential risk of malpractice litigation [9–13]. There should ultimately be a clear difference between what is a complication and what is malpractice.

Shared decision-making is a central component in the definition of high-quality care. Epstein et al. identified five specific goals clinicians should aim for, in their 2004 landmark paper: (1) listen and understand the patient's experience and expectations; (2) cultivate a working partnership; (3) supply a balanced discussion of evidence and uncertainties; (4) make recommendations informed by clinical judgment tailored to patient preferences; and 5) confirm that the patient (and family/caregivers) understand risks and treatment plan and agreement to achieve informed consent [14]. It is incumbent upon providers to lead by prompting and directing patients and their families (and other caregivers) to facilitate understanding and agreement to be able to tackle the challenge of managing complications together [7]. But communicating risk can be a challenging endeavor for clinicians since they receive limited training in communication skills for patient-centered care [15]. A 2014 systematic review of risk communication concluded that patients better comprehend risks when evidence is presented visually [16]. It is prudent for surgeons to periodically reassess their approach in the prevention and management of complications associated with MMA [17].

32.2 Why Complications Matter in MMA

32.2.1 Psychological Challenges and Patient Satisfaction

There is evidence that changes brought by orthognathic surgery can provide patients with several positive benefits in addition to the functional successes of airway and occlusion, the intangible benefits of greater self-confidence and empowerment, improved self-image, motivation, quality of life, and feelings of well-being [18, 19]. The psychological aspect of care is therefore very relevant to treatment success, and the focus of much research has been to study how patients cope with the dentofacial changes after orthognathic surgery factors into their psychological health and assess their need.

Several psychological challenges have been commonly reported after orthognathic surgery, such as low mood, decreased quality of life, and decreased satisfaction with their care. Khattak et al. studied 74 patients who underwent orthognathic surgery and found that their patient's main self-reported postoperative complications were challenges with breathing, eating, and low mood [2]. The authors recommended that clinicians should not only be aware of this dimension of care but also to proactively ascertain the patient's state of mind and facilitate psychological support if needed. Reported patient satisfaction may provide a glimpse into one's psychological health, but it is important to remember that it is not the whole story. Nonetheless, psychological health may factor or otherwise color a patient's reported satisfaction with their own treatment. Perioperative changes in mood and affect signal that there are more unseen challenges beyond the patient's perception of their facial changes alone, but also how they process changes that contribute to their overall self-image and, potentially, even their locus of control. Patients with different dentofacial abnormalities, from prognathism and retrognathism to congenital craniofacial morphologies, may have also developed various psychologic disorders (including body dysmorphic disorder [BDD], characterized by dissatisfaction and a preoccupation with a perceived defect of appearance resulting in functional impairment), emotional vulnerabilities, and potentially maladaptive behaviors, all of which need to be recognized and appropriately addressed as early as possible before an irreversible treatment decision is made and carried out [20].

Moon and Kim [21] posited that the patient's perceived needs frequently differ from the needs and focus of the orthodontists and oral surgeons treating them. This requires that the clinician exert earnest effort in discovery of the patient's perspective and expectations. Only after this is it prudent to temper a patient's unreasonable expectations, not with dismissiveness, but with reflective listening, acknowledgment and affirmation, and by preparing them with education and reminders of the types of things they may experience [21]. For instance, Phillips et al. [22] emphasized that presurgical counseling explaining the impact on activities of daily life was an important part of the patient–provider communication process for some patients more than others. They randomized patients who received BSSO to either an opening exercise group or a sensory retraining exercise group. The opening exercise group was reported to be more likely to report numbness and bothersome loss of lip sensitivity interfered with their activities of daily

living (ADL). Furthermore, patients who reported ADL interference tended to be older and/or those who scored high levels of psychological distress on their presurgical self-reports and were more likely to report a higher burden of alarming sensory dysfunction ($P < 0.02$) [22].

On the younger side of the age spectrum, it is important to recognize that children, adolescents, and young adults are not only developing physically, but emotionally as well. Belucci et al. report that emotional vulnerability can be significantly challenged by the stress of surgery, which requires that these patients are closely followed up for at least up to 1 year post-procedure. Furthermore, the authors posited that treatment success also be measured by psychosocial health and improved quality of life in addition to the standard functional markers of success, such as a functional occlusion and improved morphology [23].

Fundamentally, patient satisfaction is a reflection of the patient experience, and thus, an empathetic focus on improving that experience at all stages of care is called for. Surgical patients are more prone to depression and anxiety if treatments last for more than 6 months (Kiyak et al. 1985). It has been estimated that between 60% and 80% of patients experience at least a short bout of depression when adapting to their new appearance [24, 25].

Because some patients may dwell on dissatisfaction from their current state of challenges or in anticipation of future burdens, the surgeon should attempt to elicit discussion to work toward resolving those concerns through listening and understanding, education and empowerment, reassurance, and by providing leadership and direction with concrete tasks and milestones to work toward. In some cases, low mood may not be helped by changing the diet or other practical care modifications and goal setting. Therefore, consulting with multidisciplinary resources such as the patient's family practice physician, a social worker, and/or psychiatrist, who can identify and treat behaviors and conditions such as anxiety and depression is essential, regardless of whether these conditions are related to their surgery.

In 2007, Desforges et al. [26] studied 92 patients who underwent orthognathic surgery and documented routine points of follow-up to gauge quality of life and the psychological impact of surgery. The authors found that patients were generally surprised at the amount of facial deformation from the "important" postoperative swelling, but that ultimately 90% reported that they were satisfied with their treatment to the degree their "complex" was eliminated. Several patients had also reported weight loss, which also likely improved this self-image. The authors suggested that patient satisfaction and quality of care may be directly influenced by the strength of communication, understanding, and therapeutic alliance between the patient and the expert [26]. This further supports the work of Edgerton and Knorr, who showed in 1971 that a patient's realistic expectations of discomfort and recovery were correlated to higher rates of patient satisfaction. The 2016 systematic review by Pacheco-Pereira et al. [27] reported that factors associated with satisfaction were the final aesthetic outcome, its perceived social benefits, type of orthognathic surgery, sex, and improved self-concept during treatment.

On the other hand, factors of patient dissatisfaction were length of treatment, functional impairment, and the perceived omission of surgical risks [27]. Kiyak et al. reported that some dissatisfaction may stem from two significant predictors: 1) external sources of motivation, such as pressure from family and perceived peer pressure, and 2) neuroticism [28], both cases that could likely benefit from focused communication strategies and a psychiatric consult. Other reasons for patient dissatisfaction are lengthy waiting times, crowded clinics, and the need for a large number of office phone calls and follow-up appointments [2].

For the patient, MMA requires adaptation and perseverance through potentially formidable major complications or, at the very least, annoying daily disruptions. The surgeon should endeavor to fully appreciate that patients experience different psychological and emotional reactions throughout all stages of treatment, from presurgical planning, through the perioperative period, and into long-term follow-up. The patient should be the beneficiary of care, and not the unintended beneficiary of the risks and complications they were (or were not) warned about.

32.2.2 Evolution of Fixation Materials and Related Complications

Over time, major orthognathic surgery for OSA has significantly evolved. Bioabsorbable materials have been of great interest in the past 15 years to determine if they hold benefits in healing and reduction in complications. While such materials have demonstrated successes, these are muted by their limitations. For instance, one interesting 2013 study by Yoshioka et al. [29] studied 169 MMA patients who received biodegradable plate systems. Device failure was reported in 6% of patients (10/169), determined to be attributable to a few significant factors: asymmetry (odds ratio [OR] 5.35; $P = 0.02$) and presence of an open bite (OR 5.20; $P = 0.02$). As a result, the authors recommended the use of biodegradable plates in cases of minimal loading only [29].

A 2013 systematic review compared absorbable fixation with titanium fixation among 20 studies and 1673 pooled subjects. The review reported that across the range of orthognathic procedures, there were significantly more complications in the absorbable fixation

group compared to the titanium group (RR = 1.20; 95% CI: 1.02–1.42; $P = 0.03$). The subgroup of only bimaxillary surgery (3 studies) failed to show this same finding and showed a non-significant difference of complications between groups (RR = 1.89; 95% CI: 0.85–4.22; $P = 0.12$), with a similar non-significant finding for the MMA subgroup (6 studies; RR = 1.45; 95% CI: 0.84–2.48; $P = 0.18$) and Le Fort I osteotomy subgroup (2 studies; RR = 0.65; 95% CI: 0.34–1.23; $P = 0.18$). In the domain of fracture fixation, however, the absorbable fixation group showed a significantly lower rate of complications vs. titanium fixation (5 studies; RR = 0.71; 95% CI: 0.52–0.97; $P = 0.03$) in fracture fixation. Absorbable fixation appears to show advantages in the domains of palpability, dehiscence, infection, paresthesia, foreign body reaction, dehiscence, malocclusion, any material-related complication, exposure, and mobility ($P > 0.05$).

This systematic review clearly showed distinct trade-offs to using different materials in different procedures. While absorbable materials have shown some promise, overall outcomes appear better for titanium fixation across all maxillofacial procedures. The high cost of absorbable fixation systems presents another barrier to their use. The authors suggested that there is a paucity of data adequately profiling the safety of absorbable materials, and ultimately recommended that absorbable materials be avoided as a first-line choice across the gamut of maxillofacial surgeries with the exception of fracture fixation, where it has demonstrated superior stability and relatively fewer complications, particularly in palpability.

32.2.3 Mortality

Mortality is certainly a major adverse event (MAE) and the worst possible complication, but fortunately, deaths are very rare [30]. However, it has been acknowledged that some major complications of orthognathic surgery could potentially result in fatality if signs are promptly recognized and intervention of the complication is performed. Conditions reported have included postoperative pneumomediastinum or pneumothorax [31], as well as severe vascular injury to the internal carotid and/or internal maxillary arteries, such as pseudoaneurysm or dissection [32–35], severe intraoperative and delayed secondary hemorrhage, an obstructed airway, and deaths attributable to general anesthesia.

In 2017, Kim et al. reviewed causes of death in South Korea during or after orthognathic or jaw bone surgery from 2000 to 2016, citing 10 surgeries resulting in death or resulting in a vegetative state and 4 cases of death as a result of facial contouring surgery. Of these 14 deaths, apparent causes were bleeding (2 cases), respiratory problems (4 cases), surgical errors (1 case), and "unknown" causes (6 cases); 12 of 14 (85.7%) of the surgeries were performed by plastic surgeons, and the remaining 2 cases by an unknown department at a university hospital and a dentist. It is noteworthy that only 4 of these deaths were reported in the time period studied.

The underreporting of complications up to and including death renders it difficult to ascertain actual rates, particularly since the estimated number of orthognathic procedures performed every year has likely fluctuated. Their current annual estimated number of orthognathic surgeries is approximately 5000 cases. In the United States, no deaths attributable to orthognathic surgery have been reported in the literature; however, the US surgical community is aware of 2 or 3 cases this decade [36]. More transparent reporting of serious cases is needed to determine causes of death and identify risk factors. Despite these incidences, orthognathic surgery is generally considered safe if performed by experienced surgeons in appropriate operating theaters equipped with the necessary tools, as well as access to bailout procedures by specialists who perform them, such as general and vascular surgeons, and interventional radiologists [37, 38].

32.2.4 Short- and Long-Term Safety and Effectiveness of MMA in Context

At present, an abundance of evidence shows that MMA is both safe and effective in improving OSA outcomes and quality of life (QOL), first-line therapeutic alternative to continuous positive airway pressure (CPAP) [39]. Makovey et al. confirmed in 2017 that MMA for OSA was highly effective for their group of patients treated for moderate-to-severe OSA, showing a 2.5-fold volume in airway increase in their sample of 20 patients (mean age, 48.8 ± 12.3 years).

A 2017 overview of 11 systematic reviews concluded that MMA reliably and positively improves the dimensions of the pharyngeal airway in patients with OSA. Meta-analyses of postoperative MMA data by Holty and Guilleminault [1] in 2010 and Zaghi et al. [36] in 2016 showed statistically significant reductions in the apnea–hypopnea index (AHI) across their pooled studies [1, 36] and improvements in other measures, such as the respiratory disturbance index (RDI) [36] and lowest optimal values of nocturnal oxyhemoglobin (SpO_2 nadir) [1]. Recently, another marker of surgical success was reported to be stability of the lateral pharyngeal wall post-MMA using the AHI, oxygen-desaturation index (ODI), and Epworth sleepiness scale (ESS) [40]. MMA has been reported with high treatment success rates [1, 41]. For instance, Zaghi et al. demonstrated

a success rate of 85.5%, and a cure rate of 38.5% [36]; furthermore, this systematic review confirmed that predictors of surgical success were younger patients, lower preoperative AHI, and higher range of maxillary advancement.

32.2.5 Long-Term Follow-Up, Risk of Treatment Failure, and Complications

Evidence of MMA's longer-term safety and effectiveness for OSA was recently confirmed in 2015 by Boyd et al. in their prospective, two-center cohort study (n = 30) with a mean follow-up of 6.6 ± 2.8 years post-MMA, in which they reported that their patients experienced adverse events (AEs) that mirrored shorter studies (e.g., malocclusion, minor bleeding, local infections successfully treated with antibiotics, frequent neurosensory changes of the inferior alveolar nerve which commonly resolved by 12 months, and worsening of facial appearance noted in a low percentage of patients) [42]. In 2009, Blumen et al. also reported no major complications in their study of 50 patients post-MMA, citing that their most common complication was mental nerve sensory loss, which patients overwhelmingly deemed as secondary to the positive surgical outcome [43].

In 2017, Vigneron et al. [44] reported long-term results of MMA for OSA with a minimum of 3-year follow-up and a mean follow-up of 13.8 years ±3.9, with 88 MMA patients treated between 1995 and 2009. The authors divided patients into groups of either treatment success or treatment failure. AHI between treatment failure and success was 33.4 ± 18.7 vs. 4.7 ± 3.2, respectively ($P < 0.004$). Factors of long-term success included younger age (success rate was 100% for patients younger than 45 years of age), BMI <25 and IAH <45 and SNB <75% and narrow retrobasinlingual space (<8 mm), and patients who were treated with preoperative orthodontics. Risks of treatment failure are shown in Table 32.1.

Treatment success and the risk of complications may not be mutually exclusive. Many studies define treatment success as dependent upon meeting OSA research endpoints (e.g., AHI, blood pressure [BP], sleepiness (ESS), quality of life [QOL], etc.); however, this does not necessarily mean that complications were entirely averted. Although MMA has become an indispensable, permanent treatment for OSA with excellent outcomes, it must also be recognized for what it is: a major invasive surgical procedure for the correction of mandibular deformities, not unsusceptible to risk and the incidence of complications. In 2017, de Ruiter et al. [45] studied factors of treatment success *and* failure in 62 patients who underwent MMA for OSA. They confirmed that the AHI measure was useful to gauge treatment for both endpoints. They reported a 71% success rate (mean AHI reduction of 69%) and also identified that key predictors of treatment failures were age (58 vs. 53 years, respectively; P = 0.037) and a significantly larger neck girth (P = 0.008). Notably, the most common complications in MMA procedures were sensory impairment of the inferior alveolar nerve (60%) and malocclusion (24%) [45].

Table 32.1 Risk of treatment failure (Vigneron et al. 2017 [44])

Risk Parameterp	Odds ratio	95% CI
Preoperative BMI >24.8 vs. <24.8 kg/m2	14.00	1.43; 137.32
Preoperative age >45.01 vs. <45.01 years	14.00	1.43; 137.32
Gender Men vs. women	33.33	2.83; 392.60
Preoperative AHI >44.5 vs <44.5 events/h	6.25	1.03; 38.08
Preoperative SNB >75% vs <75%	14.17	1.83; 109.86
Maxillary advancement >11 vs <11 mm	11.00	1.06; 114.09
Postoperative MRBL >8 vs <8 mm	6.25	1.03; 38.08

BMI body mass index, *AHI* apnea–hypopnea index, *SNB* Sella-nasion-sub chin point angle measured on cephalogram, *MRBL* minimal retrobasinlingual distance measured on cephalogram

Generally, treatment failures may correlate with complications, but evidence to date is anecdotal. However, expert opinion of the authors believes that there may indeed be a relationship between incidence of complications and treatment failure. This relationship may be impacted by several variables, including surgeon training and skill [46], longer procedure duration, infection susceptibility and wound contamination, psychologic resiliency of the patient, and the quality of postoperative care management.

32.2.6 Anatomic Complexity Adds to Procedural Difficulty

The head and neck are anatomically complex, packed with a litany of functional structures either intricately connected with or very close in proximity to one another. Mere centimeters and millimeters may separate multiple body systems, including circulatory, digestive, endo-

crine, integumentary, lymphatic, nervous, respiratory, and skeletal systems [47]. Safety is of paramount concern throughout the entire course of treatment, and certainly one of the key elements in the benefit–harm calculus, which also considers effectiveness and risk of complications. A discussion and counsel surrounding the topic of safety should be communicated to the patient for several reasons: (1) it is the central motive of the profession of medicine to provide high-quality health care and do no further harm; (2) guiding an informed discussion of safety serves to establish trust in the shared decision-making process, promoting the therapeutic alliance; patients may be more comfortable with their decision after being well-informed of risks and protective safety measures were thoroughly explored; (3) this point is emphasized as a best practice in the 2010 practice parameter for surgery for OSA in adults in conjunction with a discussion of alternative treatments (Aurora et al. 2010) [48], ▶ Sect. 32.2.7:

» "The patient should be advised about potential surgical success rates and complications, the availability of alternative treatment options such as nasal positive airway pressure and oral appliances, and the levels of effectiveness and success rates of these alternative treatments."

Furthermore, informed consent requires the surgeon to appraise every prospective MMA candidate of both general and patient-specific risks of MMA treatment and potential complications and their likelihood, of which the patient's perceived morbidity may affect their decision to proceed [47].

32.2.7 Differences in Surgical Procedures Leads to Differences in Recovery Time

Differences between procedures may also explain why some patients return to full activity sooner than others. Dickerson et al. [49] reported that BSSO patients returned to work or school faster than those with Le Fort I osteotomies, the latter of which were observed to have larger average intraoperative and postoperative blood loss, longer procedure duration, and greater weight loss. By 21–28 days post-surgery, 81% of BSSO patients returned to full activity compared to approximately 50% of the Le Fort I group.

32.2.8 Identification of At-Risk Populations

There are several potential predisposing factors and risks for complications following orthognathic surgery and specifically in patients who undergo MMA for OSA. Please note that many of these at-risk populations are detailed in each subsequent topical section by complication type throughout the rest of this chapter.

It is certainly advantageous to recognize patients at risk for known complications specifically in MMA, as well as other orthognathic procedures, and indeed, other maxillofacial or otolaryngology procedures of the oral cavity, head, and neck simply because of general similarities and in objectives and approach in relatively similar anatomic areas. It is also important to not be blind to evidence from other specialties, particularly other surgical specialties, such as pharmacotherapy/pharmacy, interventional radiology, trauma, geriatrics, and pediatrics, psychiatric care and social work, as well as the role of patients exposed to myriad educational sources and media (including social media). Data from other specialties are valuable in informing MMA surgeons. Regardless of its weaknesses, multidisciplinary cooperation across the range of medical specialties (and with the patient) is a critical strength in providing the best-quality care possible featuring the objective to reduce the risks of complications via vigilant mitigation strategies and, when they occur, aggressive interventional strategies.

One particularly important at-risk population with import for MMA in the treatment of OSA is obesity. While there are clearly differences in the risk for complications following MMA between various patients and patient characteristics (e.g., younger age vs. older age [including adults vs. pediatrics], race, and patients with congenital conditions [e.g., Marfan syndrome]), obesity is one of the most common characteristics seen across MMA patients, with a disproportionate number of MMA patients likely having a higher BMI or neck circumference, which raises concern for a number potential risks that should be recognized and even discussed with patients. Certainly not all candidates of the procedure are obese; however, it appears to be one of the most frequent common denominators in patients suffering from OSA and, therefore, it is briefly explored here.

32.2.9 Obesity and Neck Circumference

Obesity is a major risk factor contributing to the natural course of OSA and is directly associated with OSA severity [50]. Furthermore, metabolic and hormonal disorders have also been associated with obesity and the incidence of cardiovascular events [51]. As previously discussed regarding treatment success and failure, de Ruiter et al. [45] identified significantly larger neck girth as a predictor of treatment failure [45], and neck circumference is largely correlated with obesity. In 2014, Cizza et al. reported that neck circumference was associated with OSA and metabolic syndrome in both obese men and premenopausal obese women sleeping less than

6.5 hours per night [52]. Neck circumference was also validated as a screening tool for pediatric OSA by Katz et al. [53], who demonstrated children in the 95th percentile of neck circumference (adjusted for age and sex) who were reported to have a significantly increased risk for OSA. It has been suggested that OSA relapse may be related (at least in part) to weight gain, among other variables and comorbid illnesses [54].

It is interesting to note that untreated severe OSA (AHI >30) has a reportedly higher risk of fatal and non-fatal cardiovascular events vs. patients with treated severe OSA or untreated moderate OSA [55]. A follow-up study in 2012 reported that compliance with OSA treatment (CPAP) was correlated with a reduced risk of developing hypertension compared to untreated OSA patients or in poorly compliant patients [56]. Many OSA patients exceed a BMI of 30, and therefore necessitate specialized education and additional care. Additional postoperative nasopharyngolaryngoscopy has been recommended for obese patients [57]. Some data suggest that treating OSA reduces obesity and that the reciprocal is also true: treating obesity decreases OSA severity [58]. Obesity or patients of advanced age who have received a mandibular setback only are at higher risk for developing sleep-disordered breathing; therefore, a 2-jaw procedure has been advocated for skeletal class III patients to anticipate pharyngeal space reduction known to be caused by mandibular setback [59].

32.3 Intraoperative and Postoperative Complications

32.3.1 Intraoperative Hemorrhage

Although there is a lack of any uniform criterion that defines excessive bleeding as a complication, bleeding is certainly of particular concern when performing MMA or total joint replacement (TJR) surgery, and one of the most common complications of MMA. Van de Perre in 1996 suggested that the most common complication in maxillary surgery is excessive blood loss, whereas in mandibular procedures, the most common complication is compromised airway attributable to local effusion. Of course, MMA is a combination of both procedures, but it is unclear which of these complications is more frequent [30].

MMA and/or TJR procedures subject the inferior alveolar vessels, superior alveolar, maxillary, retromandibular, facial, and sublingual arteries and veins to potentially severe damage. Bleeding itself may be directly managed by pressure, bone wax, gauze packed with thrombin or epinephrine, or by electrocautery. Considerable blood loss can occur because of the region's rich vascularity and blood supply. Mean intraoperative blood loss was 436 mL in a 2011 systematic review of 7 bimaxillary orthognathic studies by Piñeiro-Aguilar et al. [60]. Bleeding severity may, of course, vary considerably between patients and the type of procedure. MMA requires a longer procedure duration and greater blood loss than for procedures of the maxilla or mandible alone. A 2016 study by Thastum et al. reported intraoperative blood loss among 356 orthognathic patients treated at a University Hospital in Denmark, and found that relative blood loss was significantly increased in patients with reduced body mass in extensive procedures with long durations [61]. That same year, some of the same researchers led by Andersen et al. also reported that higher relative blood loss and longer procedure duration predicted longer hospital length of stay post-orthognathic surgery [62] and suggested that reductions in blood loss and shorter procedure times are prime targets not only to reduce inpatient morbidity and prolonged healing but also to decrease the financial burden that often accompanies these two metrics.

32.3.2 Hemorrhage Control: Hypotensive Vs. Normotensive Anesthesia

In 2001, Praveen et al. [63] conducted a randomized trial in 53 consecutive orthognathic surgery patients ranging from 15 to 33 years of age that compared hypotensive and normotensive anesthesia to determine the effect of each approach on intraoperative bleeding. They found significantly less blood loss in the hypotensive group vs. the normotensive group (median blood loss 200 mL [range: 90–400 mL vs. 350 mL [range 130–1575 mL], $P = 0.01$). This advantage has been confirmed by several subsequent studies, as well as the aforementioned systematic review [60, 64–68]. Furthermore, reduced intraoperative blood loss under hypotensive anesthesia also reduces the need for blood transfusion and its accompanying perioperative/postoperative risks, such as infection, acute lung injury, myocardial infarction (MI), tumor recurrence, and greater mortality [69]. Therefore, bleeding would increase if anticoagulants are used in long-duration procedures (such as MMA and TJR), whereas reduced bleeding is achieved if hypotensive anesthesia is used in concert with local vasoconstrictors. No matter which anesthesia is decided upon, excellent teamwork between the anesthetist and operator is essential to prevent or mitigate major intraoperative adverse events and immediate postoperative problems [30].

32.3.2.1 Surgeon Skill Paramount in Managing Intraoperative Bleeds

Managing bleeding requires a good view of the surgical field, anatomic visualization, the selection of appropriate local drugs, and employing caution during the procedure. Surgeon skill is especially important in respecting margins of vessels in the surgical field and directly managing hemorrhagic areas with gauze compresses, cauterization, or vessel ligation [70]. The temporal artery, masseteric artery, facial artery, and associated vessels all may be encountered during dissection. Additionally, TJR requires stripping of the masseter, the medial pterygoid, and the lateral pterygoid, all of which can cause significant bleeding. In multiple operated joints, anticipating bleeding can be difficult because of the obliteration of anatomical planes and the presence of scar tissue. Furthermore, bleeding control, especially of the masseteric artery, can be very difficult in the presence of ankylosis.

Careful surgical technique and methodical dissection is key in TJR. Knowledge of surgical planes and methodical dissection through these planes will help the surgeon identify and isolate vessels prior to the approach of the joint capsule or pterygomasseteric. Careful stripping of muscle from the bone with controlled force can also prevent bleeds. This is of particular concern when preparing the condyle for the condylectomy, as the masseteric artery lies just medial to the sigmoid notch. When removing the condyle, the attachments of the lateral pterygoid should be carefully stripped instead of simply pulling it out with the muscle still attached.

When performing bony cuts, adjacent soft tissues must be protected, and the surgeon should try to avoid "blind" maneuvers where the tip of the cutting instrument cannot be seen. Specialized instrumentation can protect adjacent soft tissues while performing bony cuts through the neck of the condyle in preparation for hardware. The authors of this chapter prefer to use a piezo cutting instrument for condylectomy to prevent inadvertent damage to adjacent structures.

When bleeding does occur, however, most vessels in the superficial anatomical layers can be isolated and ligated. As noted in the section on nerve damage, indiscriminate use of electrocautery is discouraged. Fine-tipped bipolar electrocautery is an elegant alternative to the bovie, but caution is still advisable. Muscular bleeds can usually be controlled by packing the wound with epinephrine or thrombin-soaked neuro patties or Surgicel® (Ethicon US, LLC, a subsidiary of Johnson & Johnson), Fibrolar Surgicel, FloSeal, and allowing sufficient time to pass while applying proper wound pressure if necessary. For major arterial bleeds that cannot be controlled with local measures, isolation of the branches of the external carotid and embolization may be necessary. Occasionally, it may be necessary to consult interventional radiology for arterial embolization, particularly in cases of severe ankylosis where access can be limited. Careful preoperative planning with appropriate imaging can allow the surgeon to identify potential hazards and embolization can be performed 1–2 days prior to surgery.

32.3.3 Postoperative Hemorrhage

Secondary delayed bleeding may be prevented by vessel ligation or angiography must be performed for large vessel injuries. Arterial epistaxis that occurs a few weeks following Le Fort I osteotomy is considered severe and must be treated immediately with packing of the nasal maxillary antrum or by reintervention with clipping or electrocoagulation of the damaged vessels and other hemostatic techniques, albeit with the a risk of causing aseptic necrosis [35].

As mentioned above, severe vascular injury is a risk that must be quickly managed. Although rare in orthognathic surgery, pseudoaneurysm has been reported to occur in the larger blood vessels, such as the maxillary, facial, and inferior alveolar artery [71, 72]. Patients may present with a pulsatile soft mass, facial effusion, and delayed hemorrhage. Pseudoaneurysm, also known as arteriovenous fistula, or dissection of the internal carotid and/or internal maxillary arteries must be rapidly managed by reintervention (exploratory vessel ligation or vessel embolization via interventional radiology) to prevent severe morbidity or mortality [32–35, 71, 72].

Some causes of blood loss may be mysteriously confounding. Diligent and persistent investigation is required to determine the underlying etiology. For instance, the physical stress of orthognathic surgery may have a distinct vascular impact that, for reasons not completely clear, results in a greater propensity for excessive menstrual bleeding and irregularity [73]. It has been postulated that increased fibrinolysis during menses is responsible for this phenomenon [74]. Prophylaxis, as previously mentioned, is the use of hypotensive anesthesia, which has been found to attenuate bleeds intraoperatively [64].

Another cause of blood loss may be momentarily elusive, as was the case of a patient of the author. A gastrointestinal (GI) bleed of unknown etiology developed in a 58 -year-old male post-MMA for OSA. The patient had reported bouts of syncope and presented with significantly reduced blood pressure and a hemoglobin level of 5.6. Infrared (IR) endoscopy of the maxillofacial region failed to find the source of the hemorrhage, but a full GI endoscopy revealed three very large, actively bleeding ulcers. It was believed that long-term use of non-steroidal anti-inflammatory drug (NSAID) was likely responsible for the ulcerations. The patient required blood transfusions, a 5-day intensive hospital stay, and ulcer ablation. The patient made a

Fig. 32.1 Pre- and postsurgical profile photographs

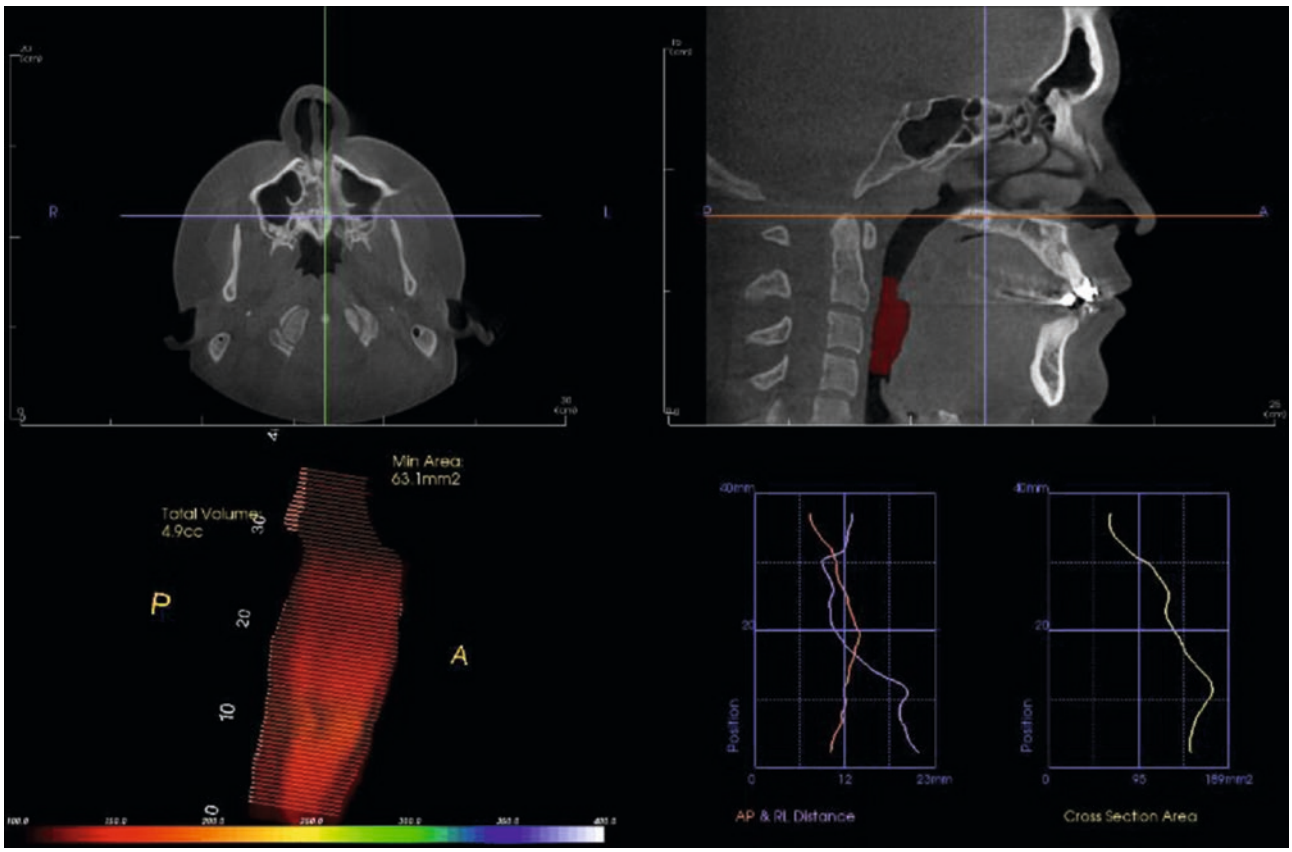


Fig. 32.2 Presurgical airway dimensions

rapid and full recovery without further bleed complications. The patient made a rapid and full recovery without further bleed complications.

Another case of NSAID use resulted in an MAE of gastric perforation post-MMA for OSA (see Figs. 32.1, 32.2, 32.3, and 32.4). A 21-year-old male experienced an uneventful course of healing post-surgery. The patient was managing his pain preferentially with ibuprofen in lieu of oxycodone because of concern of dependency. At 1-week post-MMA, the patient pre-

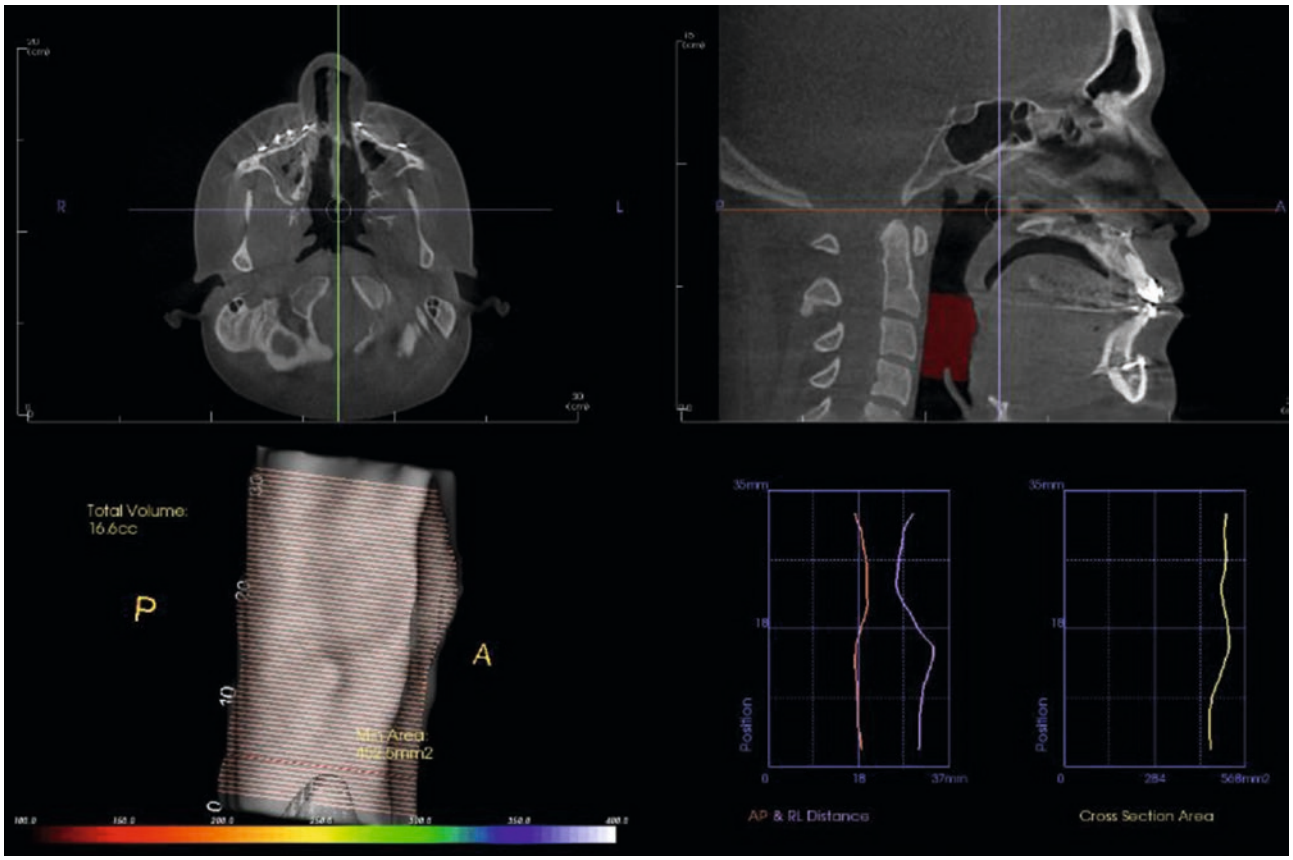


Fig. 32.3 Post-surgical airway dimensions. Minimal axial airway

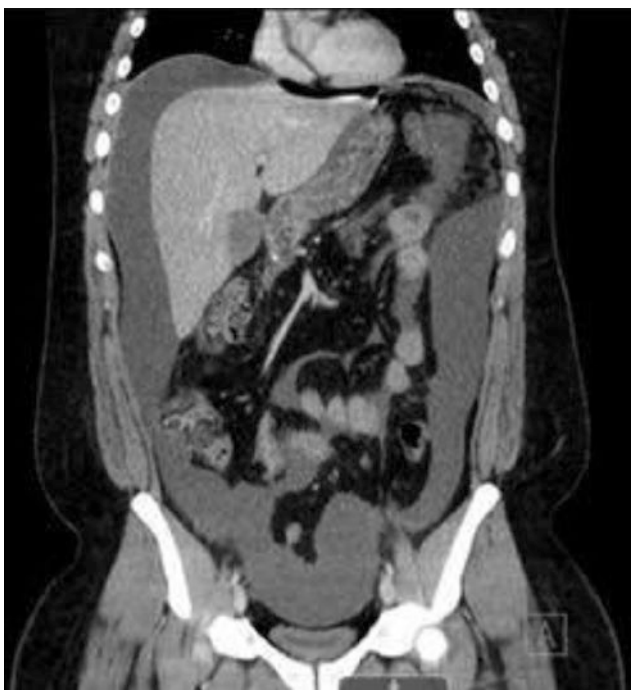


Fig. 32.4 Anterior-posterior abdominal CT with contrast

sented to our office with fever, nausea, tachycardia, and malaise. At this point, IV resuscitation was administered, and the patient was admitted to the hospital for evaluation. A hospitalist consult was requested. Upon hospitalization, the patient further complained of gastric pain, which was not initially reported. Proper labs and abdominal imaging were acquired. A high white blood cell count was revealed. Gastric perforation was suspected. As a result, general surgery went on to perform an emergency laparotomy. Even though the patient was a fairly young, healthy male (other than OSA), it was suspected that gastric perforation developed from NSAID use. The patient was admitted to the ICU post-surgically from gastric repair. Infectious diseases and general surgery followed the patient for the week-long stay, after which the patient was discharged and followed up by all engaged physician services (general surgery, infectious diseases, and OMS).

Over-the-counter (OTC) NSAIDs, including high-dose aspirin, are nearly used universally by MMA patients in the postoperative healing period. They may be particularly relied upon when transitioning off prescription pain medication from days to weeks post-

procedure. NSAIDs have long been known to directly impair the upper and lower GI tract via the depletion of COX-1-derived prostaglandins, which ultimately cause topical injury to the mucosa, allowing ulceration [75] . NSAID-related GI bleeds requiring hospitalization are more common than previously thought. A 2017 study by Sostres et al. [76] conducted a case–control study among patients with an endoscopy-confirmed major upper GI bleed (n = 3785) and compared their characteristics to control subjects (n = 6540). They found that NSAIDs significantly predicted a fourfold greater risk of GI bleed in the GI-confirmed group vs. controls (adjusted relative risk of 4.86 [95% CI, 4.32–5.46]). In 2017, Chen et al. [77] reported that even low-dose aspirin (taken for the primary prevention of cardiovascular events) was associated with the development of a lower GI bleed within 1 year in 53,805 aspirin users compared to 269,025 aspirin-non-user controls (0.20% vs 0.06%, respectively), confirmed with a very high statistical probability ($P < 0.0001$). It is also important to note the risk of NSAIDs, including aspirin.

A 2016 systematic review and meta-analysis [78] reported that the majority of MAEs attributable to aspirin use included GI bleeds and cerebral hemorrhage (risk of 1 death and 1 stroke per 1000 individuals taking aspirin for 10 years) [78, 79]. The authors did not find, however, a significant difference in the incidence of fatal or severely disabling GI bleeds with use of aspirin. MAEs associated with aspirin use are rare, although these data illustrate the potential threat of a ubiquitous OTC agent used by millions, including the vast majority of MMA patients, who also may have a history and inclination toward use of an NSAID/aspirin for other conditions, or have inculcated routine use of low-dose aspirin for the primary prevention of cardiac events. This underscores the need for physicians to be actively vigilant detectives in all areas of care, including in the arena of pharmacotherapy (whether prescribed or OTC), to direct it and determine the source of complications and, if a solution is elusive, to recruit multidisciplinary help. This help may include consults with specialists, pharmacists, and even the patient's family and/or other caretakers to determine risk of drug interactions and side effects.

32.3.4 Potential Complications of Le Fort I Segmental Osteotomy

32.3.4.1 Oroantral Fistula

Oroantral fistula is the pathological communication between the maxillary sinus and the oral cavity, and may be caused by trauma of the procedure, infection, osteomyelitis, and other iatrogenic causes. The goal of closing oroantral fistula post-segmental osteotomy is to achieve complete fistula closure, optimize patient comfort during to promote expedient healing, and avoid other complications, including reopening and site infection [80]. Closure of an incident oroantral fistula has been proposed to be resolved using a variety of methods and harvest sources of bone and connective tissue, including ranging from septal cartilage graft [81], modifications of other various connective tissue grafts, such as use of auricular cartilage [82], use of bony closure [83], closure with tongue flap, and closure with buccal fat pad flap and endoscopic drainage of the maxillary sinus [84] (see Figs. 32.5 and 32.6).

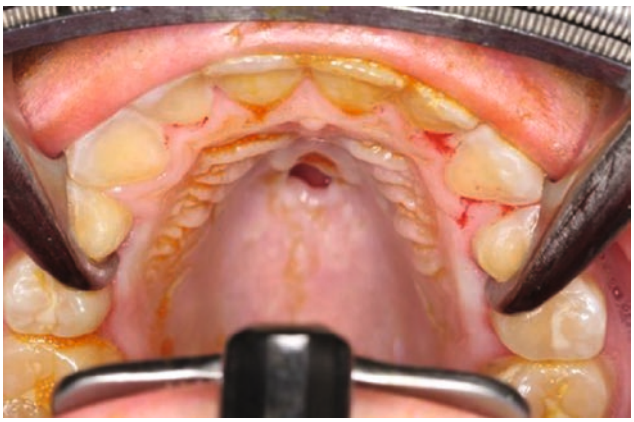


Fig. 32.5 Intraoperative image of matured orantral fistula 7 months post-surgery

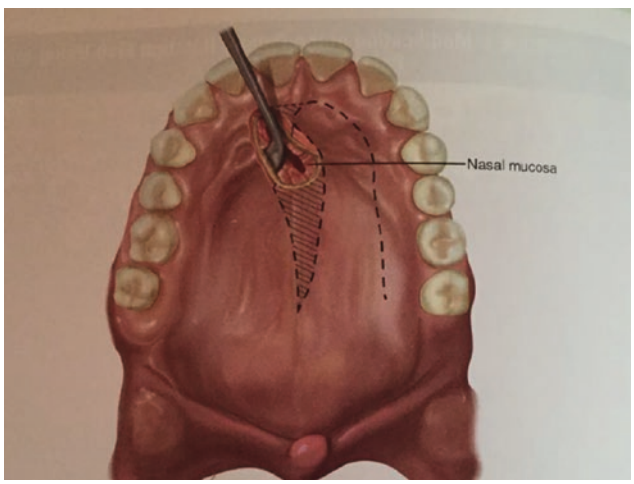


Fig. 32.6 Proposed palatal flap for repair of orantral fistula. (Adopted from the Atlas of oral and maxillofacial surgery)

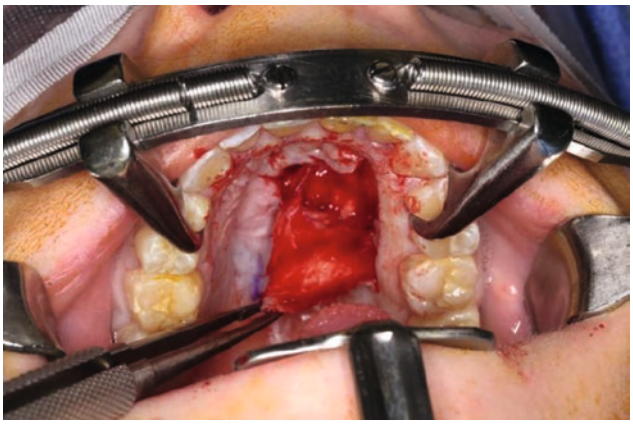


Fig. 32.7 Elevation of passively moving flaps was approximated over the orantral fistula. In addition to layered closure of nasal mucosa, fibrin glue was used to seal and for closure of palatal flaps

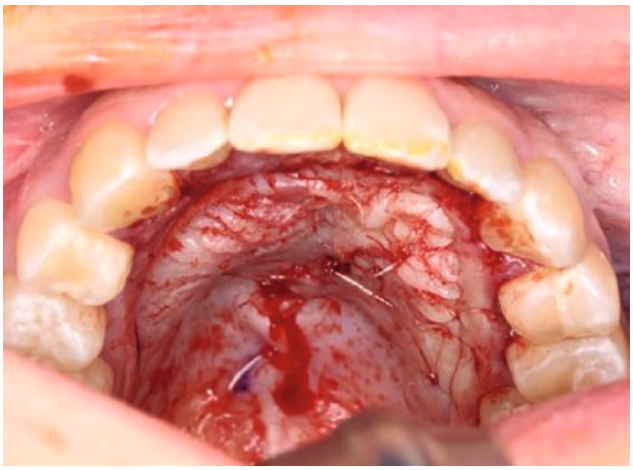


Fig. 32.8 Repaired palatal closure with a full-coverage palatal splint

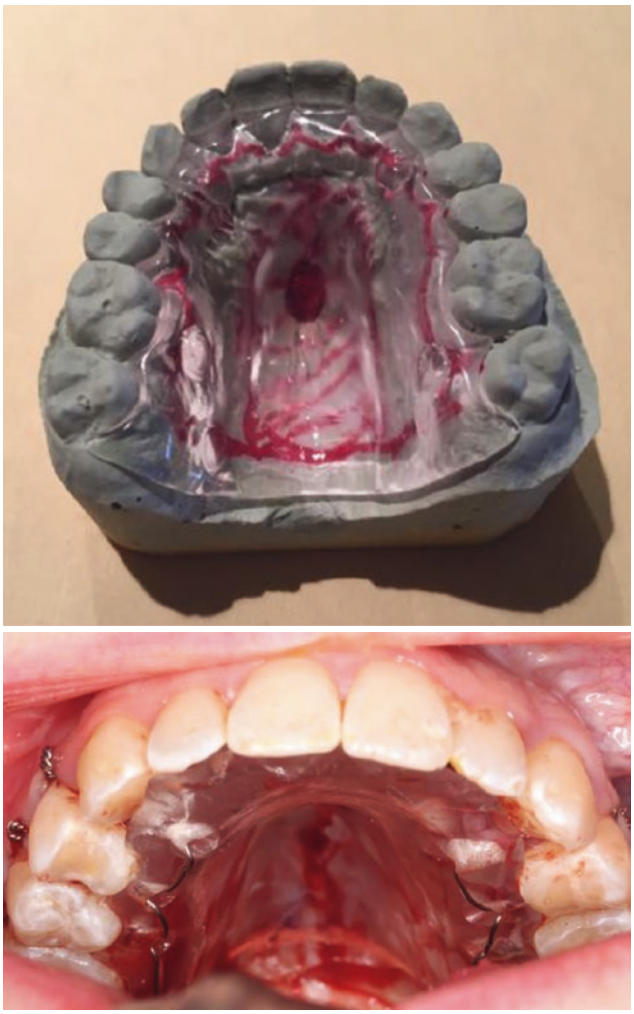


Fig. 32.9 Fabricated palatal splint

There are three main categories of flap implant, each useful in different scenarios [85]:

1. Buccal flap (useful for large fistulas) [83, 84]
2. Palatal flap
3. Vestibular flap

One of the present authors performed closure of an oro-antral fistula in a patient post-segmental osteotomy, as shown below (Figs. 32.7, 32.8, 32.9, and 32.10).

32.3.4.2 Segmental Hypovascularity, Necrosis of Bony Segments, and Delayed Wound Healing

Although rare, some transoral vertical ramus osteotomies have reported the very rare occurrence of proximal segment necrosis, presumably attributable to local isch-

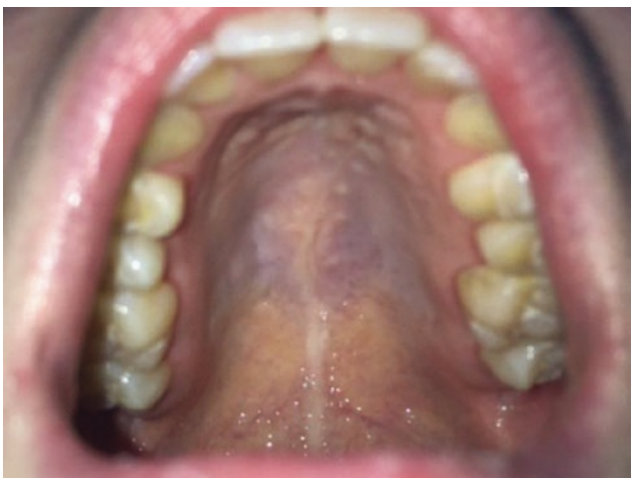


Fig. 32.10 Palatal splint was removed at 21 days post-surgery and this image shows healing at 2 months post-surgery; 2-year follow-up showed no reoccurrence

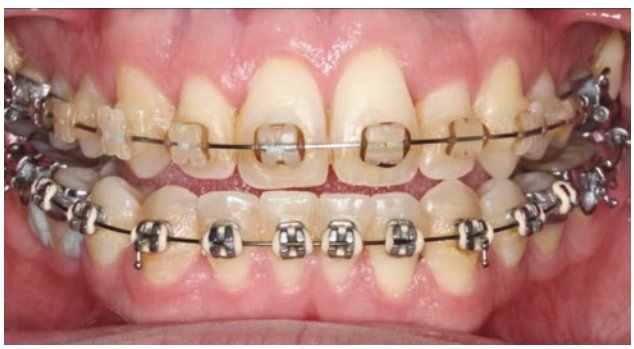
emia, although the exact cause in many cases may be unclear. In the late 1970s, Bell and Kennedy performed animal studies to further understand bone healing and revascularization. One notable 1976 paper [86] reported that they performed pedicled and non-pedicled vertical ramus osteotomies in 15 adult Rhesus monkeys. When the proximal segment was pedicled to the articular capsule and lateral pterygoid, minimal vascular injury or ischemia was observed. Alternatively, intraosseous necrosis and delayed wound healing were observed to result from the abrupt vascular ischemia when the proximal segment was not pedicled to soft tissue. They postulated that necrosis of the proximal segment's inferior aspect could be prevented with osteotomies performed further posterior and superior to the gonial angles. However, Lownie et al. in 1980 reported on wound healing and avascular necrosis in 10 baboons. Their results are in conflict with Bell and Kennedy's conclusion that necrosis can be completely avoided by repositioned osteotomies. Lownie et al. showed positive outcomes following reattachment of not only the periosteum, but also reattachment of the lateral pterygoid attachment and temporomandibular joint (TMJ) capsule.

More recent human research suggests that transient ischemia of the bony segments can and may occur in the acute postoperative phase and require reintervention, even in healthy, non-diabetics. In 2014, Kim et al. [87] reported a rare case of necrosis of the proximal segments in patients who underwent transoral vertical ramus osteotomies. They noted that more frequent complications of vertical ramus osteotomies are infections and hemorrhage due to major vessel injury, bad splits, and neurosensory deficit. Necrosis is a surprisingly rare complication, particularly in an otherwise healthy 37-year-old female for whom a Lefort I osteotomy and transoral vertical ramus osteotomy were indicated for mandibular prognathism. However, the patient reported that she was experiencing swelling and submandibular pain on the right side, and she was therefore readmitted 10-weeks postoperatively with progressive pain and purulent discharge. A panoramic X-ray radiograph revealed a radiolucent lesion causing discontinuity of the proximal segment. Further axial CT imaging at the same time revealed the right mandible sustained destructive bony changes and eroded cortical layers. Upon admission, she was treated with IV combination therapy of a third-generation cephalosporin plus metronidazole, and an intraoral operative plan, which led to resection of roughly 15 mm of the dead tissue at the inferior aspect of the proximal segment. Kim et al. surmised that necrosis of bony segments follows local ischemia attributable to periosteal detachment and the subsequent formation of a hematoma. Further, the authors suggest that circulation to the bony segments is likely to be reestablished given successful reattachment of the periosteum. Similar to other rare complications, surgeons should be aware of this complication of bony segments and that, while it is rare, has been known to occur in healthy subjects without a history of chronic vascular ischemia and resultant delay of wound healing, such as in diabetic patients.

Osteotomies may also present with delayed union or non-union because of insufficient healing tissues, and may be present in patients identified with known systemic conditions that compromise wound healing, such as in diabetics, diseases affecting connective tissues, vascular diseases, congenital anomalies (including cleft palate), osteoporosis, and genetic and behavioral disorders associated with nutrition deficiencies [88], as well as idiopathic trauma during surgery. Many of these illnesses run the gamut of detection and severity. If warranted by signs or symptoms and if not previously diagnosed, surgeons may need to conduct investigative measures prior to or following surgery and take necessary steps to intervene.

32.3.4.3 Case: Hypovascularity of Segmental Osteotomy and Related Infection

A 36-year-old male patient with severe OSA underwent MMA with counterclockwise rotation. Course of surgery was uneventful, but intraoperatively closer to the end of the procedure, some signs of hypovascularity of the Le Fort segment were becoming apparent when the gingival tissue of the maxilla showed some signs of ischemia (see ◘ Figs. 32.11, 32.12, and 32.13). In the following 3 days post-surgery, the maxilla (especially the anterior segment) became more and more ischemic. Additionally, the pinprick tests were negative for bleeding and relief of palatal splint did not aid the maxilla's ischemic status. At this point, the patient was referred to hyperbaric oxygen therapy (HBO_2) to rescue vascularity. HBO_2 therapy consists of placing the patient into a pressurized chamber up to 3 atmospheres (ATM) of pres-



◘ **Fig. 32.11** Pre- and post-MMA photographs

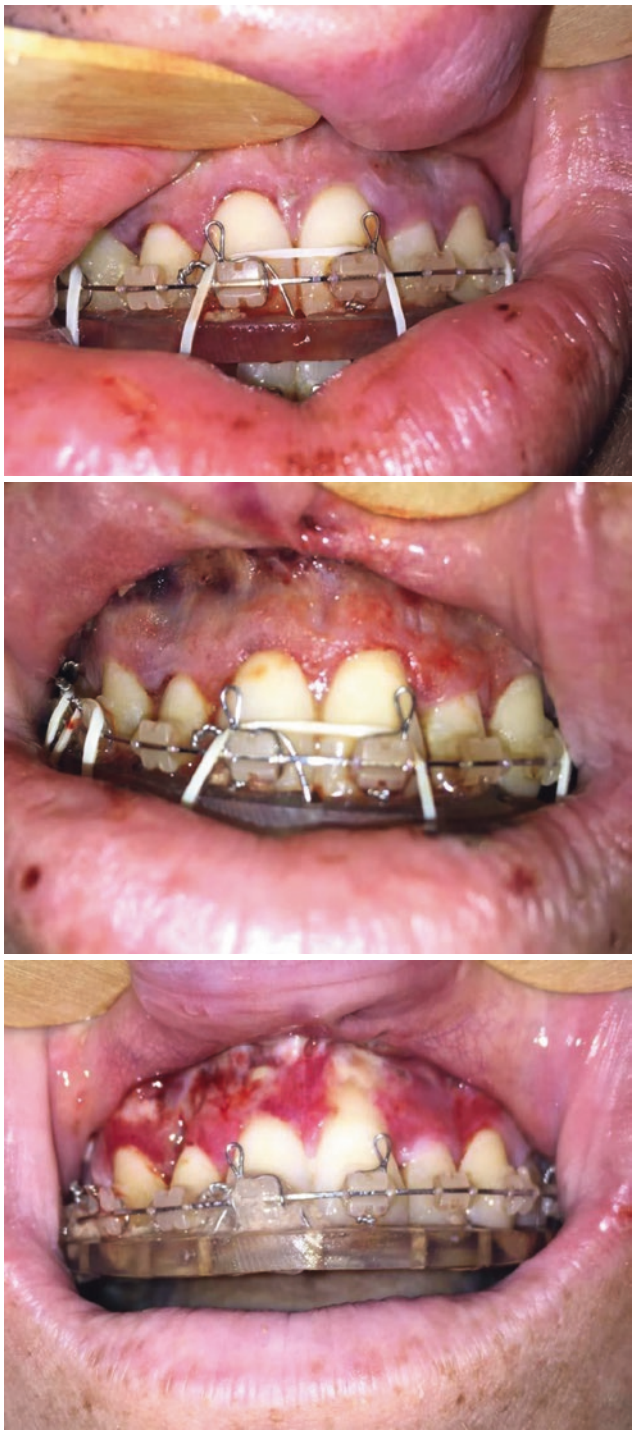


Fig. 32.12 Post-surgical onset of necrosis

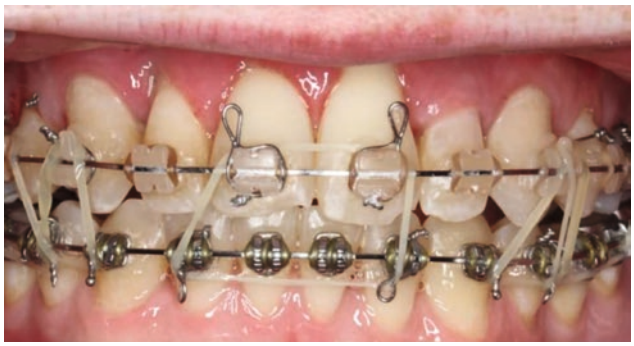


Fig. 32.13 Perfusion was completely restored on day 10 post-surgery

sure, with each additional atmosphere equal to 33 feet depth of seawater (or 14.7 lbs. per square inch [psi]).

Several dramatic physiologic changes occur when the human body is placed in an environment of increased atmospheric pressure coupled with 100% oxygen, first of which is the doubling of plasma O_2 concentration from 3 mL/L to 60 mL/L, which thereby promotes the perfusion of oxygen to ischemic tissue [89]. Other effects of HBO_2 therapy include the potentiation of various antibiotics (aminoglycosides and quinolones), and it is believed to prevent protease and free radical release in some injuries, which is believed to decrease vasoconstriction, edema, and cellular damage. HBO_2 therapy is also thought to neutralize alpha exotoxins produced by bacteria (e.g. *clostridium*). All these elements may be responsible for improved wound healing. Several conditions are known to be treatable with HBO_2, including some off-label indications (such as stroke and autism).

HBO_2 therapy was first investigated for use in plastic surgery in 1965 when McFarlane et al. [90] experimented with necrosis prevention in experimental pedicle flaps, and the following year in 1966 when McFarlane and Wermuth [91] investigated it in a rat model for composite graft salvage. A 2003 report by London maxillofacial surgeons Shanker and Farrell [92] documented use of HBO_2 therapy initiated for a 17-year-old male following excision of an odontoid peg via Le Fort I osteotomy. Peg excision occurred while the down-fractured segment was retracted and displaced for more than 8 hours. In the 10 days following the procedure, ischemia was observed on the patient's left side of the maxilla, including both the labial and palatal sides. A total of 9 HBO_2 sessions were administered over the course of 5 days, with each treatment of 100% O_2 lasting 90 minutes at 242.4 kPa with an air break and safe decompression, with all procedures free of adverse events (100% O_2). HBO_2 therapy resulted in significantly improved tissue perfusion, albeit with slightly reduced pallor of the labial side of the teeth. By the end of all treatments, the hard palate was observed to be well perfused.

A 2011 paper by El-Din Eid and El Sayed [93] reported their experience with 16 Le Fort I maxillary advancement patients, 8 of whom received HBO_2 therapy compared to 8 control patients who did not receive HBO_2. Relapses in the control group and time points 2 and 3 among all parameters were significantly different than the intervention group, ultimately showing that

HBO_2 therapy is feasible and effective at improving postoperative stability following Le Fort I maxillary advancement. A 2018 systematic review of HBO_2 concluded that the treatment appears to be generally effective in combination with standard wound therapies to heal complicated acute wounds, though more data are needed before being recommended as a mainstay in wound therapy [94].

After 15 dives, the patient started to show signs of revascularization and improvement of vascularity to the anterior segment of the maxilla. Furthermore, the patient's other clinical outcomes were dramatic. His AHI improved from 32 to 5, and the lowest oxygen saturation improved from 82% to 90%. The patient is still undergoing periodontal surgery to restore the defects, but the anterior segment is perfused and thriving.

32.3.4.4 Tooth Loss After Segmental Osteotomy

A 16-year-old female presented with severe anterior open bite that required closure (See ◘ Fig. 32.14). She underwent double-jaw surgery with segmentation and maxillary expansion. Preoperatively, the patient was notified that tooth loss was immanent due to proximity of the canine and premolar roots (see ◘ Figs. 32.15, 32.16, and 32.17).

32.3.5 Postoperative Nausea and Vomiting

Postoperative nausea and vomiting (PONV) is the most common postoperative complication following orthognathic surgery. It is also one of the most distressing and annoying complications, and not without significant morbidity that could potentially be life threatening. It is oftentimes temporarily disabling, sometimes rendering the patient adversely affected for not only minutes, hours, or days, but for potentially longer periods of time. It is also not without serious consequences, often extending outpatient recovery duration, inpatient hospital stay, and thereby increasing costs of hospitalization.

Several factors are associated with PONV and may be categorized into the following, as Silva et al. summarized in their 2006 study:

1. Non-anesthetic factors
   (a) Patient self-reported characteristics (e.g., age, gender, smoking status, prior history of motion sickness, or prior PONV report)
2. Anesthetic factors
   (a) Mask ventilation
   (b) Volatile anesthetics
   (c) Intraoperative use of opioids
   (d) Patient hydration
3. Surgical-related factors
   (a) Type and length of surgery
   (b) Use of postoperative analgesic opioids

◘ **Fig. 32.14** Patient pre- and post-surgeries

Before After

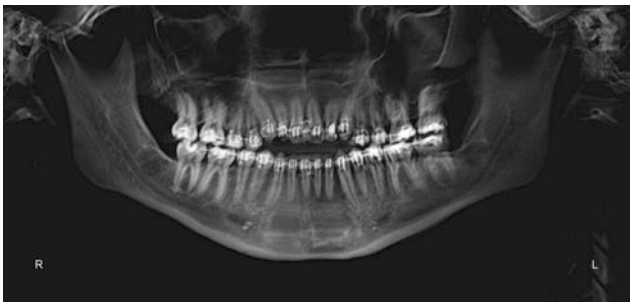


Fig. 32.15 Presurgery panoramic showing a severe anterior open bite and close proximity of canine and premolars where the segmental osteotomy was planned to be performed

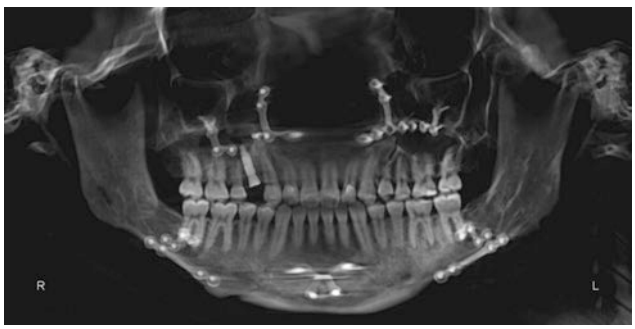


Fig. 32.16 Post-surgery panorama showing that tooth number 5 was lost on the right side, as the possibility was advised to the patient. The site with tooth loss was grafted

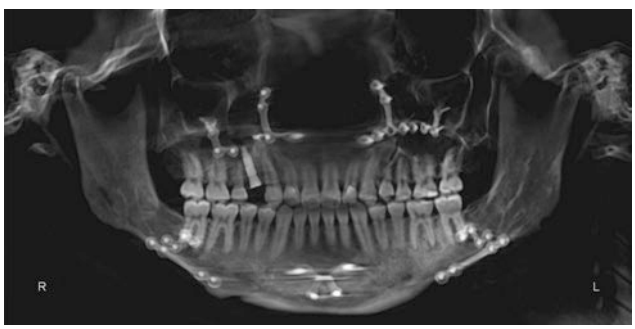


Fig. 32.17 Panoramic radiograph shows implant placement awaiting restoration

The incidence of PONV, during the ether era, ranged from 75% to 80%, improving gradually to approximately 9% to 43% over the past 40 years. In a 2006 retrospective, cross-sectional survey of 553 patients who underwent maxillary and/or mandibular osteotomies, more than 40% (206/514) of patients experienced PONV [95]. The female-to-male ratio was 1.61:1, although female gender was not significant (P = 0.0654; alpha set at 0.05). Younger patients (between 15 and 25 years of age) reported PONV the most, and this group had the largest prevalence as a result (age was statistically associated with PONV [P = 0.0340]). The Koivuranta score [96] and the Apfel score [97] are 2 common risk assessment scores for PONV. These simple risk assessments have been proposed for PONV, but their usage is complicated in the orthognathic surgery population by multiple factors [98]:

1. The score's low sensitivity and specificity (65% to 70%).
2. Continued debate over optimal antiemetic prophylaxis.
3. Differing medical risks between nausea and vomiting, since they are each discrete medical signs.
   (a) Nausea poses no significant health risk, albeit disturbing a patient's sense of well-being and amplified anxiety.
   (b) Vomiting, however, could lead to significant health risks.
      (i) Hematoma
      (ii) Wound dehiscence
      (iii) Dehydration
      (iv) Electrolyte imbalances
      (v) Esophageal damage
      (vi) Aspiration

32.3.6 Bad Splits

A "bad split" is an unfavorable and troublesome fracture that may occur intraoperatively during sagittal split osteotomy or postoperatively, and possibly could lead to relapse. Frequently cited reasons for bad split include incomplete osteotomies, using osteotomes that are too large, attempting to split the segments too rapidly, presence of impacted third molars, and misdirecting the medial osteotomy upward toward the condyle and placement of the medial osteotomy too far superior to the lingula [99]. Synonyms used for bad split include "buccal cortical plate fracture" (proximal segment) and "lingual cortical plate fracture" (distal segment) [100]. A bad split can occur during SSO of the mandible (see Figs. 32.18 and 32.19). The rate of bad splits during sagittal split ramus osteotomy (SSRO) has been reported to be approximately 2.3% (pooled incidence rates vary from 0.5% and 14.6%). Interestingly, despite technical innovations and improved operator experience and training, the incidence of bad splits has not appeared to appreciably decrease as expected [100], confirming the need for additional focused research efforts to determine different ways to prevent and treat it.

There are a number of potential reasons underpinning the risk of bad splits. First, MMA is a difficult procedure in a highly complex anatomical region. More complex morphology is reportedly a risk factor for bad splits [99]. Second, inexperienced surgeon skill or poorly

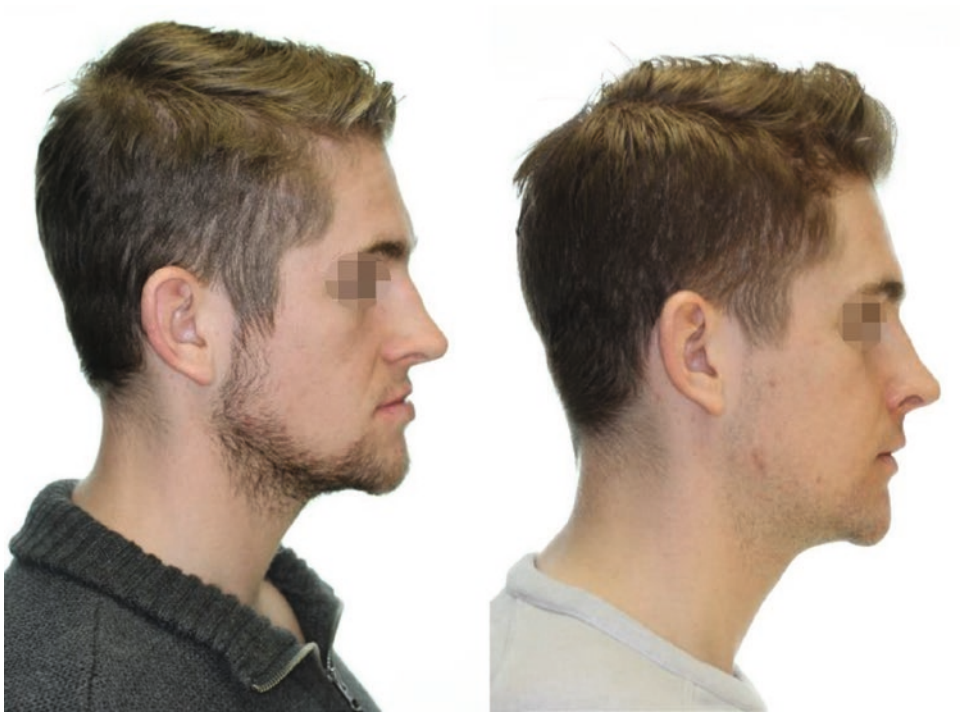


Fig. 32.18 Pre- and post-MMA profile photographs

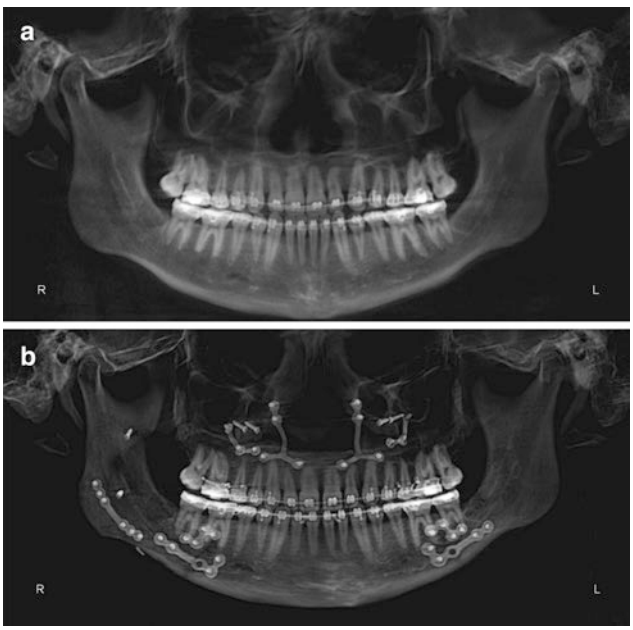


Fig. 32.19 Panoramic view of presurgery and 1-year post-double jaw surgery. The second image shows repair of the buccal segment and subcondylar bony fractures that were managed during surgery. (**a**) Presurgery panoramic radiograph. (**b**) Post-surgery panoramic radiograph

executed technique is purportedly an important causative factor for bad splits [101, 102]. Finally, impacted third molars have been controversial, with debate surrounding whether impacted teeth should be extracted 6 to 9 months prior to MMA or concurrently. Some evidence suggests that age may also be a risk factor, with older patients suspected to be at higher risk than younger patients [100, 103–107]. Some studies, however, argue the opposite that younger patients are at increased risk [108, 109].

It has been reported that third molars are the most frequently reported risk factor for bad splits, with an increased risk in younger patients younger than 20 years of age with impacted third molars and >40 years of age without, with concern surrounding root contact with the MMA fixation screws [107]. Furthermore, the incidence of bad splits has been reported to be higher if third molars are unerupted and have divergent roots or a distoangular/vertical orientation [105]. Although gender appears to be correlated with bad splits, several studies report that the risk of bad split is similar in both males and females.

In 2015, Zamiri et al. reported that neither a short length nor long length of the medial osteotomy line appeared to affect the incidence of bad splits, since the ramus' thickness could dictate the type of fracture pattern on its medial side [110]. Some anatomic differences in the mandible may also increase the risk of a bad split [99]. However, while the use of splitters and separators in lieu of chisels has been suspected to increase the risk of bad splits, this has not been supported by the data [111]. It has been reported that the risk of bad splits are especially more frequent when the forced separation of bone segments is performed after incomplete osteotomy of the mandibular inferior border [112–115].

32.3.7 Infections

Rates of postoperative infection are also relatively low. A 2015 retrospective analysis of 336 patients reported that the infection rate post-MMA was 11.3%, which is suspected to still be within a normal range for a clean/contaminated procedure [116]. A 2016 retrospective study reported a postoperative rate of infection at approximately 8% [Davis et al.]. In 2016, Verewij et al. conducted a meta-analysis of 59 studies and 4123 patients, of which 333 were reported with a postoperative infection. The mean pooled incidence was 9.6% per patient, with an incidence range between 2.0% and 25.9% per patient.

Several types of infections have been reported in the literature, ranging from acute or chronic maxillary sinusitis or otitis media, cellulitis, osteomyelitis, abscesses, and operative site infections requiring removal of osteosynthesis material [117]. Verewij et al. also reported that the mean pooled incidence of material removal was 11.2% per patient, with smoking cited as one of the most important risk factors. Risk of infection is surmised to be greater with any implanted hardware that provides havens for or otherwise increases the areas conducive for bacterial colonization, such as has been reported in the use of miniplates with monocortical screws [118].

The importance of judicious prophylactic use of antibiotics cannot be overstated, as it is a valuable tool with which to mitigate the risk of infection and the potentially serious sequelae that may accompany it. Even the use of first-generation cephalosporins (e.g., cefazolin) has been reported to be more effective than combination prophylaxis (penicillin/clindamycin) for any orthognathic surgery [119]. However, the 2015 Cochrane systematic review by Brignardello-Petersen et al. [120] suggested that amoxicillin-clavulanic acid with clindamycin was nearly "always effective," though this therapy is not appropriate for patients with a penicillin allergy and those who cannot tolerate its myriad adverse effects. The Cochrane authors suggested that evidence supporting single doses or short dosing was unclear. The preponderance of data supported their recommendation for long-term antibiotic prophylaxis in orthognathic surgeries, suggesting that the evidence indicates a probable mitigation of the risk of infection, though stopped short of recommending exact dosages or antibiotic types. They defined long-term antibiotic prophylaxis as administration prior to surgery, during surgery, and >24 hours postoperatively, particularly in smokers and procedures greater than 3 hours in duration. In 2017, however, Posnick et al. suggested that the use of third-generation cephalosporins may reduce the rate of infection as low as 1% [121].

32.3.8 Neurologic Injury

32.3.8.1 Facial Nerve Weakness

Temporomandibular joint replacement (TJR) surgery requires a preauricular/endaural incision as well as a Risdon or posterior mandibular incision. Both of these incisions put branches of the facial nerve (cranial nerve VII) at risk for injury. Proper placement of the preauricular or endaural incision with careful dissection down to the root of the zygoma just over the tragal cartilage will usually keep the surgeon well behind the pathway of the frontal branch of the facial nerve as it traverses the zygomatic arch. This is particularly important when dissecting to a multiply operated joint, as the soft tissue planes tend to be obliterated and retraction is very difficult.

Similarly, when using the Risdon's incision or retromandibular approach, the surgeon should use careful surgical technique, identifying layers and controlling bleeding in order to prevent "blind dissection" and inadvertent damage to the marginal mandibular branch of the facial nerve. Nerve testing can also be used to ensure that the nerve is identified and protected during dissection down to the joint capsule or the pterygomandibular sling. Nerves can be injured due to indiscriminate use of electrocautery, and as dissection proceeds into the deeper structures, the surgeon must use caution when controlling bleeding.

Traction injuries are the most common injuries to branches of the facial nerve. When operating on a multiple-operated joint, the incidence of traction injury increases due to the presence of scar band which does not "give" like the unoperated tissue and, thus, translates more shearing force to the nerve. Careful attention while retracting, proper placement of instruments and avoiding excessive retraction pressure can help prevent nerve injury during TJR surgery. In some cases, it may be necessary to extend the incision to either avoid a nerve encountered during dissection or relieve excessive retraction pressures.

Facial nerve weakness can happen postoperatively after TJR surgery. The patient should be well informed of this potential complication preoperatively (in verbal and written form) and reassured during the immediate postoperative period. Most facial nerve weakness will resolve within 6 months and the patient should be brought back periodically for monitoring. We recommend taking photos at rest and while having muscles of facial expression active every 1–2 months to track the progress of recovery both to reassure the patient and for legal purposes.

32.3.9 Venous Thromboembolism (VTE)

Venous thromboembolism (VTE) is a relatively infrequent complication in orthognathic surgery and distraction osteogenesis cases [122] [123]. There are 90 million cases of VTE per year in the United States with an incidence of roughly 0.1–0.3% per year, and remains underappreciated [124] [125]. In maxillofacial surgery, Lowry et al. estimated the incidence of VTE as 0.00035% based on the memory recall of 103 maxillofacial surgeons [126]. In 1998, Moreano et al. [127] identified 34 cases of postoperative VTE following 12,805 otolaryngology or head and neck procedures and reported a VTE incidence of 0.1% following general otolaryngologic procedures and 0.6% following head and neck surgery.

Clinically, VTE is the overarching term that encapsulates either pulmonary embolism (PE) *and/or* deep vein thrombosis (DVT) [128], and is a preventable cause of morbidity and mortality in hospitalized patients, particularly in the perioperative period, accounting for approximately 10% of hospital deaths [129] and could result in permanent work-related disability and economic burden [130, 131]. Obesity, higher BMI, adverse cardiovascular and metabolic characteristics have long been suspected to be major risk factors for VTE, including C-reactive protein, measures of oxidative stress [132–134], as well as hyperthyroidism (levels of FT4 at the upper range of normal are a "strong" risk factor for VTE) [135].

Interestingly, oral infection was recently revealed to be a major independent risk factor for VTE, which may be particularly applicable for some orthognathic surgery cases. In a 2018 match case–control study by Cahoon et al., the authors reported that specific sites of infections, which included oral infections (75% of which were attributable to oral candidiasis; the remainder had dental infection/abscess, concurrent oral HSV, or sublingual salivary gland infection), significantly predicted VTE compared to infected controls with no reported VTE. Specifically, the odds of a VTE event in the presence of oral infections were an 11.61-fold increased risk (95% CI: 2.22, 60.82; $P = 0.004$) behind only systemic blood stream infections, which held a 17.8-fold greater risk (95% CI: 1.17, 269.7; $P = 0.004$); the risk of VTE with concurrent pneumonia was 3.64-fold (95% CI: 2.00, 6.63; $P < 0.0001$), and symptomatic urinary tract infection had a and 2.24-fold risk of VTE (95% CI: 1.29, 3.91, $P = 0.004$). The odds of the combination of all other infections were 1.56-fold higher risk of VTE vs. patients with no infection (95% CI: 1.08, 2.25; $P = 0.017$). Therefore, it may be prudent to initiate VTE prophylaxis in the presence of any infection, particularly oral and systemic infections [136].

It is also important to note that VTE is known to occur in several reconstructive procedures to remove oral and maxillofacial cancers, some of which share similar approaches to orthognathic surgery [137–139].

Table 32.2 Signs and symptoms of PE and DVT and preventive and therapeutic options [143]

VTE Condition	Signs and symptoms	Prophylaxis/treatment for both PE and DVT
Pulmonary embolism	Pulmonary embolism Recent or sudden shortness of breath Sharp chest pain Coughing up blood Sudden collapse	*Compression therapy* Use of intermittent pneumatic compression boots while hospitalized or in bed at home Wearing preventative compression hose (typical strength between 10 mm Hg and 18 mm Hg. *Medical prevention* Low-molecular-weight heparin (enoxaparin 40 mg daily or dalteparin 5000 units daily) Fondaparinux 2.5 mg daily Unfractionated heparin 5000 units every 8 hours Warfarin (used after hip surgery) *Patient prevention measures* Avoiding dehydration Resuming activity as soon as possible to promote circulation Consulting provider about DVT/PE risks, including identification of genetic propensity Lifestyle changes (exercise)
DVT	Recent swelling of one leg Unexplained pain or tenderness of 1 leg	

Routine prophylaxis for cancer procedures have been justified in the literature [140], and there is generally wide agreement across several types of major surgeries that VTE prophylaxis is indicated in patients at moderate or high risk for VTE, [141]. In their 2012 economic systematic review, Thirugnanam et al. identified that the most economically attractive VTE prophylaxis agents for inpatients were low-molecular-weight heparins and fondaparinux [142]. Prevention and treatment are detailed in Table 32.2.

32.3.10 Relapse and Risks Leading to Reoperation

Causes for relapse may be considered a complication that is caused by a litany of factors, some of which have

been previously described. As noted above, a relapse of OSA could be attributed to not only postoperative weight gain and the natural history of other comorbidities and changes in medications [54], but also bone remodeling or absorption and its related skeletal instability, implant device failure or postoperative trauma. In some cases it is impossible to identify the acyual cause of relapse. However, several structural factors have been associated with relapse, including changes attributable to any excess bone movement and/or rotation, changes of the teeth, and any unresolved malocclusion during pre-procedure orthodontic treatment, any change in position of the condyles, and any significant changes in ramus inclination and the mandibular plane [144, 145].

Lee et al. in 2010 performed a retrospective review of 110 cases orthognathic surgical cases in South Korea, which reported that six patients (5.5%) who relapsed early postoperatively (within 2 weeks) required reintervention repair. Several reasons for reintervention have been reported in the literature: protruded mandible in concert with facial asymmetry (5 cases; the other with facial asymmetry only), insufficient occlusal stability, anterior open bite, and unilateral mandibular shift. Several risk factors were identified which were believed to contribute to these unstable results: preexisting temporomandibular disorder (TMD), including dual bite, condylar sagging, counterclockwise rotation of the mandibular segment, asymmetrical mandibular setback (and related soft tissue tension), and potentially inadequate final wafer fabrication [144]. Late skeletal relapse and reoperation have also been reported to be required for secondary to progressive condylar resorption (PCR). In 1994, Crawford et al. reported 7 cases of following orthognathic surgery, the etiology of which is still unclear. Reoperations are also necessary for the removal of osteosynthesis material, commonly complicated by postoperative infection (mean pooled incidence of 11.2% per patient).

32.3.10.1 Condylar Resorption

PCR is the progressive morphologic change of the condyle to a finger-shaped figure and reduced volume of the mandibular condyle, frequently characterized by noticeable changes in facial height. Even though it has been routinely reported in the literature, its development following orthognathic surgery is still not fully understood. A 2012 systematic review reported that PCR occurred in 5.3% (137/2567) of orthognathic surgery subjects aged 14–45 years, 97.6% (122/137) of whom were female [146]. The same review showed 118 subjects with mandibular deficiency and a high mandibular plane was associated with PCR. Further complicating its etiology and pathogenesis, reintervention outcomes for PCR are unpredictable, with reported reoperation cases varying considerably between studies. And while orthodontics may be employed in some patients in whom skeletal relapse has occurred to reach a satisfactory occlusal result, this outcome, too, may be highly variable and similarly unpredictable [147]. Interestingly, resorption appears to cease after roughly 2 years [148].

Counterclockwise rotation of both proximal and distal segments of the mandible and posterior displacement have also been surmised to be risk factors for PCR and may be contraindicated for at-risk patients. In 2000, Hwang et al. [149] confirmed this in a consecutive series of 452 orthognathic subjects, 17 of whom presented with postoperative PCR confirmed by a standardized preoperative and postoperative imaging protocol. They found that both distal and proximal segments of the mandible were significantly rotated counterclockwise in PCR patients vs. the non-PCR patients (P = 0.005 and.007, respectively). Furthermore, PCR patients experienced significantly more posterior displacements of the condyle (P = 0.007). In 2004, Hwang et al. [150] compared 2 groups: 17 PCR subjects vs. 22 subjects without condylar resorption, but with mandibular hypoplasia and a high mandibular plane. A relevant non-surgical risk factor was a posteriorly inclined condylar neck. While there were no significant gender differences between the two groups, non-PCR patients confirmed were significantly older than the PCR group (P = 0.001). Furthermore, these results were confirmed by the 2016 systematic review by Catherine et al. [151]. PCR has been shown to be, to various extents, successfully treated by pharmacotherapy, including NSAIDs, tetracyclines, omega-3 fatty acids, inflammatory cytokine inhibitors [152]. Definitive treatment, however, lies in reintervention of progressively worse occlusive result after a period of activity of at least 6 months. Preventive measures include great care in at-risk patients to optimally position for appropriate postoperative mechanical loading on the TMJ, and performing mandibular advancement only after the condyles has been confirmed by imaging to be stable [153].

32.3.10.2 Intraoperative Tooth Injury

Because MMA requires surgical work in close proximity to teeth, the procedure poses risk of damage to their roots and vascular supply. Maxillomandibular fixation screws have been shown to be safe and useful during implantation, but caution must be exercised to avoid damaging local structures, particularly dental roots.

32.3.11 Risks in the Surgery-First Orthognathic Approach

In 1991, Brachvogel, Berten, and Hausamen [154] posited the surgery-first orthognathic approach (SFOA or

SFA), which bypasses presurgical orthodontic preparation and instead immediately proceeds with corrective orthognathic surgery. The rationale for this approach is predicated on the theory of "regional acceleratory phenomenon" (RAP), postulated by Frost in 1983. Frost drew attention to increased local metabolic activity after sustaining a surgical wound, which is characterized by enhanced bone turnover and the more expedient movement of dentoalveolar tissues. He surmised that this physiologic feature may be "harnessed" to some extent to facilitate more expeditious orthodontic treatment post-orthognathic surgery. Based on this hypothesis, presurgical orthodontics could potentially be *disadvantageous* both before and after orthognathic surgery (because of its very long treatment times ranging from 7 to 47 months), and its known complications of dental cavities, a higher risk of periodontitis, recession of the gingiva, and root resorption [155–157]. However, SFA remains controversial. In 2016, Gandedkar et al. proposed that SFA may be "ideal" for patients with minimal dental discrepancies, but stopped short of recommending the approach for more complex cases, the selection of which should be cautiously reserved [158].

Some evidence suggests that complications may indeed be higher in SFA patients vs. traditionally treated orthognathic patients, including those for MMA. Pelo et al. in 2017 reported data from human SFA and human traditional studies performed between January 2000 and August 2016. Excluded from the review were case reports, any non-surgical reports, and studies that failed to render transparent segmental osteotomy data. The authors reported that recurrent segmental osteotomies were significantly more frequent in patients undergoing SFA compared to patients treated traditionally, with the caveat that there is a notable sparsity of data in this area. With more data, Pelo et al. speculated that SFA-related complications, while still relatively low, may be slightly higher than the traditional approach if analyzed in larger datasets and more rigorously conducted comparative studies. They concluded that SFA should be performed only by skilled operators since it is "a little riskier than the traditional approach." They further noted that there may be success in reducing the incidence of minor complications by preserving blood supply to the bones and teeth, particularly sparing periosteal, mucosal, and muscular insertions.

Complications reported in each respective approach may represent a critical determining factor in intervention choice. The findings of Pelo et al. underscore the importance of closely evaluating complications in the context of determining the appropriate approach, timing, and the most optimal intervention based on available data. Therefore, risks of complications should never be an area of care that is overlooked, nor should the discussion be avoided with patients. It is critical that both surgeons and patients together understand complications and properly calculate risks, particularly since complications are key central elements in swaying a surgeon and/or the patient to one therapeutic approach or another. Complications themselves or their perceived risk may not be a compass that always points North, so to speak, but can certainly inform and, to some extent, guide the critical surgeon–patient calculus that weighs risks and benefits. Thus, there is an ever-present need for surgeons to critically evaluate all potential risks of complications, be familiar with their incidence and predisposing factors, and even actively participate in reporting incidence in the literature, whether it be a single case report, case series, or larger observational or investigative studies.

32.3.12 Nasal Morphology Issues

MMA may not only induce the intentional changes in the dimensions of the hypopharynx, oropharynx, and nasopharynx, but may also cause unintentional morphological changes, such as nasal septal deviation and/or widening of the nose. These changes in nasal morphology may occur as a result of maxillary repositioning, maxillary expansion and/or advancement, or superior impaction, all of which may structurally impact the nasal septum and alar cartilage.

Nasal-septal deviation can occur if there are shortcomings in the amount of bone trimmed from the maxilla's nasal crest or insufficient trimming of cartilaginous septum. Both may incur displacement or compression. A LeFort I osteotomy may also be responsible for nasal deviation. Furthermore, nasotracheal intubation may cause an increase in intranasal pressure, and extubation of a partially deflated cuff could potentially subluxate the quadrangular cartilage; thus, manual inspection should be performed post-extubation. A 2017 study of 379 OSA patients who underwent MMA at Stanford Hospital between August 1992 and December 2015 reported the incidence of post-MMA corrective nasal surgery and was reported to be at least 18.7% (71/379) [159]. Of these, 12.7% (48/379) of patients required functional nasal surgery, and 6% (23/379) required both functional and aesthetic nasal surgery. The authors suggested that this rate of post-MMA is unacceptable and may potentially be improved by refining MMA techniques [159].

One such potential improvement was recently proposed to minimize widening of the nose with a modified alar cinch suture technique. Instead of performing a single, long alar cinch suture (known to anchor the bilateral nasolabial soft tissue medially), which has

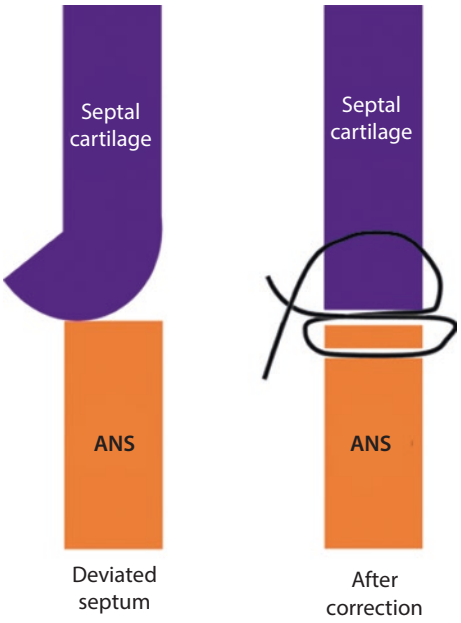


Fig. 32.20 Schematic of a figure-8-style suture of the septal cartilage to the anterior nasal spine (ANS) to stabilize the caudal portion of septal cartilage. (Shin et al. [162] - This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (► http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. ► https://jkamprs.springeropen.com/articles/10.1186/s40902-016-0067-z)

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been criticized for restricting the medial movement of the ala, Yen et al. in 2016 proposed a modified alar cinch suture [160] that essentially restrains alar fibroareolar tissues more selectively via individually placed, non-absorbable sutures at the bilateral lower border of the piriform rim. The technique was tested after Le Fort I maxillary osteotomy in 17 cases. Overall, they found the technique to reduce interference and distortion from intubation and promoted a more symmetrical nasolabial profile [160].

Previously, Van Sickels and Tucker proposed that there were 3 viable approaches to manage deviation nasal septum postoperatively: 1) immediately reoperate, or 2) immediate manipulation using the blind method (similar to that used in reduction of nasal bone), or (3) septoplasty at a later date to avoid complications of the airway [161]. But a more basic technique has also been proposed by Shin et al. in 2016, who proposed that nasal deviation may be managed by performing nasal reduction and caudal septal fixation via a figure-8 suture with 3-0 prolene [162] (See Fig. 32.20). Advantages of this technique may be to more tightly secure the corrected septal position post-extubation and to ultimately mitigate changes in columella morphology over time [163]. Again, it is important to inform patients of the risk of nasal widening and septal deviation post-MMA, that they are risks known to accompany the procedure in some cases, and that corrective rhinoplasty may be needed to correct malformation [164, 165]. Generally, patients need to be counseled on the morphological and aesthetic changes possible post-surgery. Changes are more severe for patients who are already orthognathic vs. patients who have severe retrognathia, for whom the aesthetic changes are more positive and agreeable.

32.3.13 Impact of Orthognathic Surgery on Oral Health

In 1993, Ellingson and Artun conducted a study of 93 orthognathic surgery subjects to determine if ischemia associated with surgical trauma had a long-term impact on the pulp and dental health [166]. Orofacial trauma, specifically to teeth and pulp, has long been known to impact dental health after traumatic injuries, and the question was raised if the trauma of surgery was comparable to other traumatic events. A total of 93 subjects received either LeFort I osteotomy ($n = 42$) or BSSOs (76 patients). Mean age was 38.5 ± 9.4 years (range 21.9 to 63.9), and subjects were followed up long term, an average of 8.9 ± 2.9 years (range 4.7–15.3) post-index procedure. The development of pulp canal obliteration was observed significantly more frequently in the maxillae of patients treated by LeFort I osteotomy than in those without ($P < 0.001$), but a larger proportion of osteotomy patients were affected by pulp canal obliteration ($P < 0.01$), but bilateral split osteotomies had no impact on pathologic pulpal changes observed over time. The risk factors identified leading to necrosis were history of a restoration or presence of dental caries ($P < 0.01$), but few teeth required extraction during the lengthy follow-up.

32.4 Conclusion

A wide range of complications have been reported to accompany MMA and orthognathic surgery generally. Some of the most common are issues with anesthesia and nausea and vomiting, neurosensory disturbances, and less commonly primary and secondary hemorrhage, bad splits, fractures, infections, respiratory issues, or

vascular issues, such as venous thromboembolism or pseudoaneurysm. The psychiatric health of a patient should also be a preeminent concern. Although many complications are unpredictable, the patient's comfort, quality of life, and path of healing can be planned for. The patient and their family or other caregivers should be empowered to play an active role in the patient's care. Providers have a legal and ethical responsibility to understand the gamut of complications and know how to expertly manage them. Providers are also obliged to be thorough risk communicators, and expected to be cautious, meticulous, and expertly precise as surgeons, ultimately willing to help prevent complications or mitigate their impact.

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Virtual Surgical Planning for Osseous Surgery to Manage Obstructive Sleep Apnea

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Contents

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33.1 Introduction

The use of maxillofacial osteotomies for the treatment of dentofacial deformities has a long and storied history [1–3]. Movement of the maxilla, mandible, and chin can be performed in all three planes of space in order to achieve the appropriate skeletal changes. In general, underdevelopment of the maxilla and/or mandible is a more common clinical scenario than overdevelopment [4]; therefore more advancement type procedures have been done over time. Historically, surgeons anecdotally noted improvement in patient-reported signs and symptoms of daytime somnolence, snoring, and nighttime observations of apneic episodes by bed partners in a subset of their orthognathic surgical patients. As such, it becomes clear over time that osseous advancement of the facial bones could provide relief of airway obstruction [5, 6].

The use of maxillofacial osteotomies to manage the patient with PAP-intolerant obstructive sleep apnea has been validated both as a rescue treatment for patients failing other types of OSA procedures (such as UPPP) and as a primary treatment [7–9].

Such treatments advance the osseous insertion points of soft tissues that support airway patency. Specifically, anterior movement of the maxilla advances the soft palate, superior aspect of the pterygomandibular raphe, and associated connections to the superior pharyngeal constrictor while also permitting intra-nasal modifications such as septoplasty, removal of bone spurs, osseous recontouring, and turbinoplasty. Advancement of the mandible and chin directly advances the attachments of the genioglossus, geniohyoid, mylohyoid, digastric muscles, and the inferior aspect of the pterygomandibular raphe. A proper term for such airway reconstruction would be *osseopharyngeal reconstruction*, which emphasizes the indirect soft tissue impact via the direct bony reconstruction (see Fig. 33.1).

It is logical to assume that "more is better" when moving these bony structures forward with airway patency as the goal, and to a certain extent this is true. However, this is mitigated by the realities of post-surgical relapse due to a combination of soft tissue stretching (rubber band effect) as well as decreasing bony contact with progressively greater advancement [10–13]. Although not well quantified, there appears to be approximately 50–70% concordance between the amount of bony advancement (particularly of the mandibular soft tissue attachments) and the amount of oropharyngeal and hypopharyngeal airway dimensional change [14–19]. In addition, it is not clear in any one patient how much additional airway volume/dimension is actually needed in order to achieve a successful outcome. Therefore most surgeons will plan to advance the mandibular anterior soft tissue attachments a minimum 10–12 mm to achieve 5–8 mm of AP caliber change.

Historically, maxillomandibular advancement was planned using a decidedly two-dimensional, sagittal plane visual treatment objective (VTO). This process utilized clinical examination, lateral cephalometric films, acetate tracings, and dental model surgery to create a surgical plan that would achieve the desired AP dimensional changes. Surgical guide splints were then created by a laboratory in order to permit accurate transfer of the surgical treatment plan to the patient.

Although this method can still work well, contemporary planning involves the use of virtual three-dimensional methods that can provide significant additional information. The overall process is identical to that used for routine orthognathic surgery. Specific steps include (see Fig. 33.2):

- Comprehensive history and physical examination
- Occlusal records including either surface scan images of the teeth or dental models and bite registration
- Cone-beam or medical-grade computed tomography
- Preliminary treatment planning (2-D)

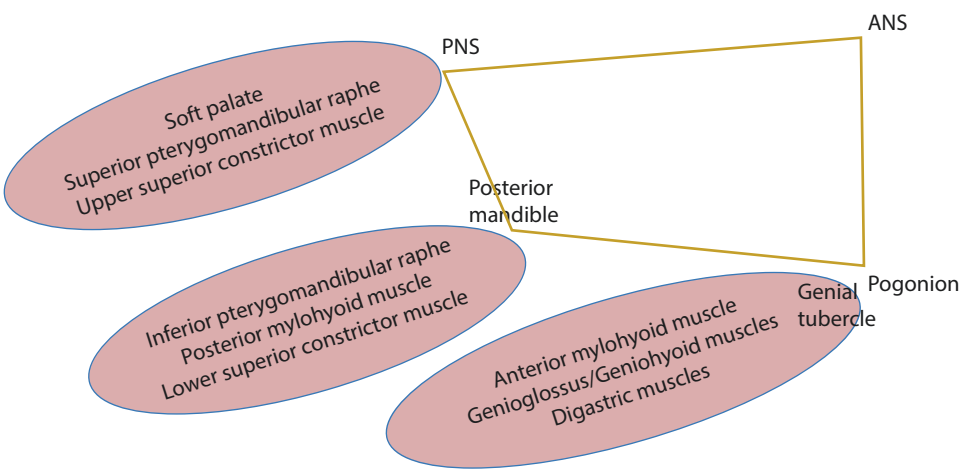

Fig. 33.1 Demonstrating the anatomical principle of osseopharyngeal reconstruction via maxillomandibular advancement

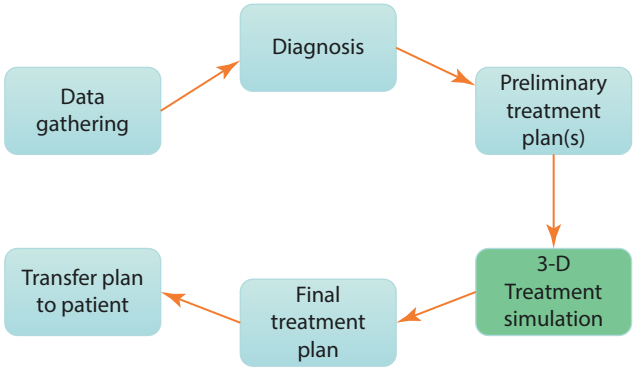

Fig. 33.2 OSA surgical treatment planning process integrating virtual three-dimensional simulation

- Three-dimensional treatment simulation
- Construction of intraoperative guide splints
- Transfer of surgical plan to the patient in the operating room

Treatment planning differs from traditional orthognathic surgery, where the primary goals include correcting the dental malocclusion, harmonizing facial bony and soft tissues to maximize esthetics, and doing so in a manner that will be stable of the long-term time frame. The main goal of maxillomandibular advancement or other operations that address obstructive sleep apnea is clearly focused on resolution of airway obstruction with surgery that will be stable over the long term. In patients not having orthodontic treatment, preservation of current occlusion is an important but ultimately secondary goal. Patients who have orthodontic treatment as a part of their OSA treatment plan also may benefit by also obtaining an improvement in their dental occlusion.

The ultimate goal is to render the patient "surgically cured" of their obstructive sleep apnea (defined as an AHI < 5 events per hour). For all patients, the goal of surgery is to reduce AHI to a level that renders them unlikely to develop medical comorbidities (AHI below 15–20). The specific goal in each patient's case will be impacted by the severity of their pre-surgical AHI and their tolerance for post-surgical PAP therapy if their post-surgical AHI is between 5 and 15 with persistent daytime sleepiness. It is this author's opinion that most patients are not favorably inclined towards the idea of needing some form of PAP therapy after MMA, as it is usually the intolerance of this treatment that leads them to seek surgery in the first place. Clearly some patients with post-surgical AHI of 5–15 with persistent sleepiness may tolerate PAP therapy better as the pressures may be reduced because of their improved airway anatomy, but this is not likely to be viewed as a completely successful outcome by MMA patients.

Occlusal plane manipulation such as counterclockwise rotation can be a useful technique in MMA patients for a variety of reasons. First, even though most MMA patients have multilevel obstruction, retroglossal airway crowding commonly plays a central role in the propensity of the airway to collapse. Therefore, maximal advancement of the insertions of the genioglossus, geniohyoid, inferior pterygomandibular raphe, and associated superior constrictor musculature is crucial to obtaining a successful outcome (see Fig. 33.1). In bimaxillary operations (maxilla and mandible only), a minimum advancement of at least 11–12 mm at pogonion should be the goal. Small amounts of counterclockwise rotation can permit this magnitude of advancement while simultaneously mitigating some of the advancement at the LeFort I level, where increasing degrees of advancement may lead to difficulties with bony overlap, telescoping of segments, and potential malunion or nonunion. In addition, perinasal fullness and nasal anatomy changes may not be well tolerated in comparison with the changes in mandibular and chin position.

Additional advancement of the bony insertion of the genioglossus and geniohyoid can be obtained with the use of nontraditional chin osteotomies. Traditional genioplasty procedures are not useful in these patients (with the possible exception of patients with BMI <25 and AHI <15, which is uncommon) [20]. Chin surgeries must have as their goal the specific advancement of the genial tubercle region of the mandible. Virtual surgery can provide additional information to locate and definitively advance this structure.

The question of whether virtual surgical planning for MMA and other osseous surgeries is "better" than traditional methods of planning remains unanswered. There are no studies that evaluate whether the outcomes achieved (reduction in Apnea-Hypopnea Index, reduction in Epworth Sleepiness Score, resolution of cardiovascular disease, prevention of automobile accidents, etc.) are superior if virtual three-dimensional treatment planning is used as compared to traditional methods. The few studies that compare traditional orthognathic surgery processes (cephalometry, model surgery) with virtual planning techniques have focused mostly on correction of asymmetries and found some evidence that virtual three-dimensional planning conferred benefits in terms of obtaining the desired results [21, 22]. Although it would not be illogical to conclude that creation of AP dimensional change may be more predictable if done with virtual surgical planning, further study is needed.

33.2 Case Examples

Figure 33.3 presents the lateral cephalometric imaging of a 33-year-old female patient with severe obstructive sleep apnea planned for maxillomandibular advancement. This is an example of telegnathic surgery in which the intention is to not change the patient's dental occlusion but to perform an osseopharyngeal reconstruction. As in all cases, careful clinical history and physical examinations, as well as data acquisition in the form of a cone-beam or medical-grade CT, are performed. This data is then integrated to permit a virtual web meeting to proceed.

In this particular case, the high mandibular plane angle presents the surgeon the opportunity to utilize occlusal plane rotation to maximize the advancement of the genial tubercle muscular insertions while simultaneously mitigating some of the midface changes that may be concerning for the patient. Figures 33.4 and 33.5 demonstrate the operative sequence performed in this particular case as well as the planned dimensional surgical movements, with maxilla-first surgical sequence and counterclockwise rotation, followed by mandibular advancement into final occlusion. The patient also had a traditional genioglossus advancement performed, as subjective cosmetic requirements did not permit utilizing a formal anterior mandibular osteotomy to advance the genial tubercles further. Figure 33.4 also demonstrates the potential disadvantage of maxillary-first surgery in cases in which there is both counterclockwise rotation and a need to significantly advance the maxilla (and mandible). The intermediate splint in such cases can be unwieldy.

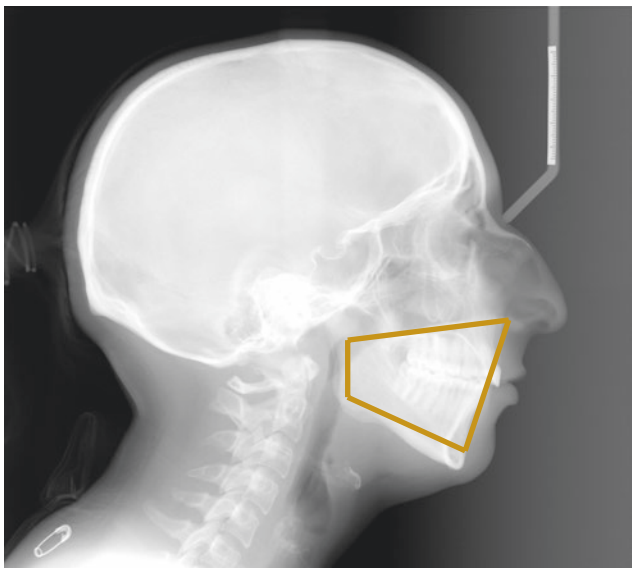

Fig. 33.3 Lateral cephalometric image clearly demonstrating the convex facial profile, high mandibular plane angle, and narrow retroglossal airway

Contrast this case with the 49-year-old male patient with severe OSA whose lateral cephalometric image is shown in Fig. 33.6. In this case, both the occlusal plane and mandibular plane are exceedingly flat. CCW rotation in a case such as this is problematic from a biomechanical standpoint (occlusion). Note the much more manageable shape and orientation of the intermediate splint in this case. Figures 33.7 and 33.8 demonstrate the nearly identical advancement at pogonion as compared to the prior case, but clearly the intraoperative surgical process will be simpler in this case of mandible-first MMA as compared to the prior case.

The use of mandibular-first surgical sequencing can be useful in select cases where counterclockwise rotation is desired and significant maxillary (and mandibular) advancement is desired. Figure 33.9 shows a lateral cephalometric image of a 59-year-old male with severe OSA and clear retroglossal airway crowding. The occlusal plane angle is somewhat steeper than in the second case, which opens the opportunity to use CCW rotation to achieve increased advancement of the mandible and chin. Also note the improved shape and orientation of the intermediate splint achieved with mandibular-first surgery (Figs. 33.10 and 33.11). The disadvantage of mandibular-first surgery is the absolute need for rigid fixation of the mandible in order to continue the operation on the maxilla. If, for instance, an aberrant split were to occur in the mandibular osteotomy(s) and rigid fixation cannot be created, the surgeon may have to abort the maxillary portion of the case, place the patient in IMF with intermediate splint in place, and allow the mandible to heal, returning at a later date to complete the maxillary advancement.

A second advantage of mandible-first surgery is the fact that condylar position during records acquisition becomes a non-issue. This is in contrast to maxillary-first surgery wherein the maxillary repositioning is dependent on records being obtained with the mandibular condyles accurately seated in the glenoid fossa. OSA patients (and non-OSA patients) can protrude their mandible forward, probably subconsciously in OSA patients as a reflex maneuver to help keep their airway open.

The use of genioplasty (either isolated or in addition to other maxillomandibular osteotomies) has been advocated by some for OSA patients, with the concept that this will open the airway by moving the anterior mandibular muscle attachments forward. The data with respect to traditional sliding genioplasty procedures does not support this operation as an isolated surgery to treat OSA, with the possible exception of those patients with minimal disease (AHI <10) who are of normal

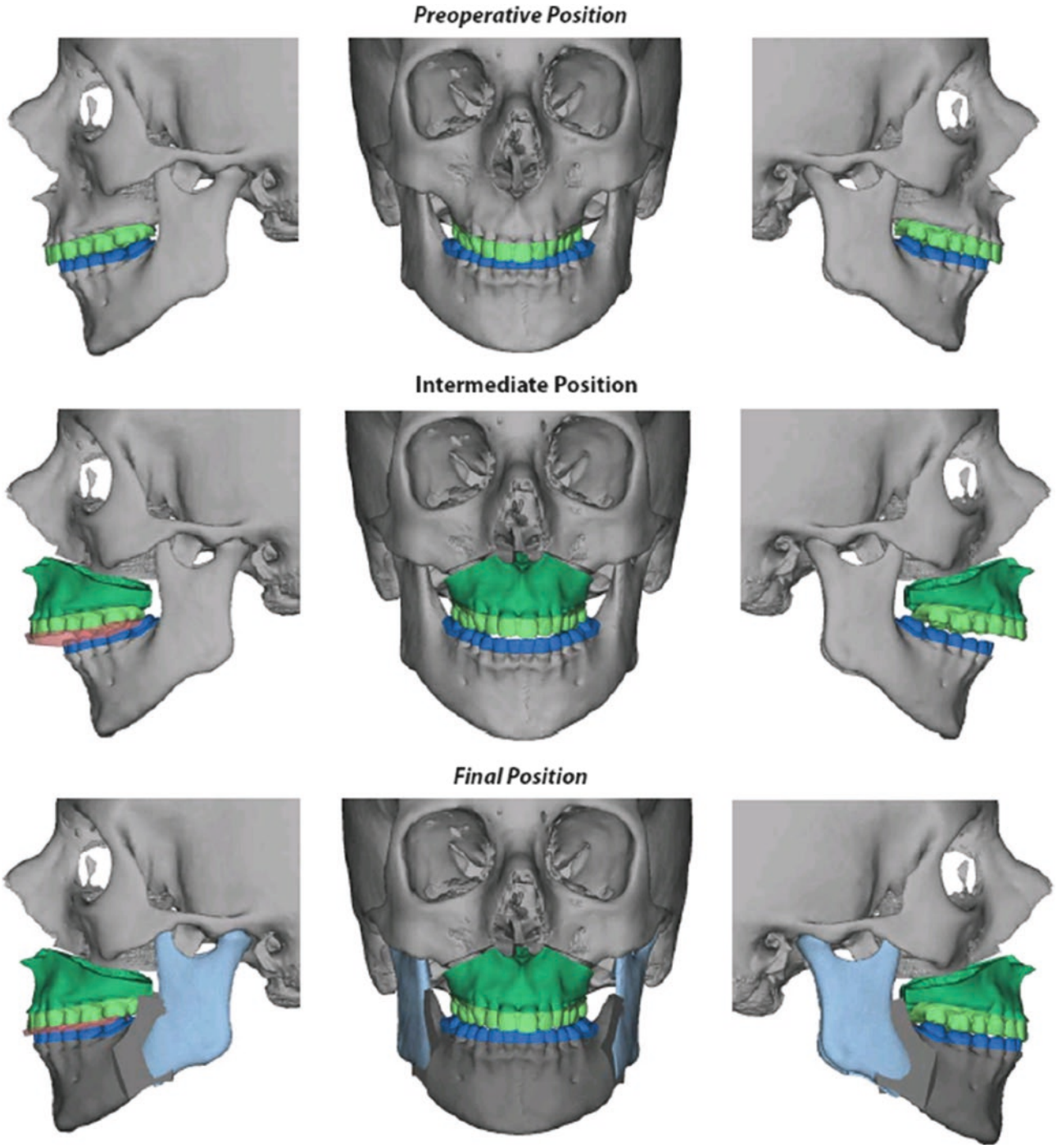

Fig. 33.4 VSP screens shots demonstrated CCW occlusal plane rotation, with maxillary surgery first. Note the thickness and orientation of the intermediate splint

(BMI < 25) weight [20]. This is not a common clinical scenario seen by most surgeons. Traditional sliding genioplasty procedures do not typically capture the actual genial tubercle, with its associated muscle attachments, which likely accounts for the lack of efficacy in isolated sliding genioplasty for OSA.

On the other hand, osteotomies of the chin that do capture the genial tubercle region are theoretically attractive as a mechanism to directly suspend the genial tubercles in an anterior position. Virtual surgical planning can help the surgeon accurately determine the position of the tubercle region of the mandible and, if

Fig. 33.5 Dimensional movements as planned for surgery. Note the difference in advancement of ANS versus Menton

Point	Name	Anterior /Posterior	Left/Right	Up/Down
ANS	Anterior Nasal Spine	3.47 mm Anterior	1.37 mm Left	0.47 mm Up
A	A Point	4.14 mm Anterior	1.08 mm Left	0.09 mm Down
ISU1	Midline of Upper Incisor	6.00 mm Anterior	1.18 mm Left	0.00 mm Down
UK9L	Upper Left Canine	5.06 mm Anterior	0.79 mm Left	0.67
U6L	Upper Left Anterior Molar (mesiobuccal cusp)	4.59 mm Anterior	0.15 mm Left	1.83 mm Down
UK9R	Upper Right Canine	6.46 mm Anterior	0.66 mm Left	0.97 mm Down
U6R	Upper Right Anterior Molar (mesiobuccal cusp)	6.55 mm Anterior	0.03 mm Left	2.12 mm Down
ISL1	Midline of Lower Incisor	8.61 mm Anterior	1.86 mm Right	1.30 mm Down
L6L	Lower Left Anterior Molar (mesiobuccal cusp)	9.00 mm Anterior	1.27 mm Right	3.18 mm Down
L6R	Lower Right Anterior Molar (mesiobuccal cusp)	7.85 mm Anterior	1.22 mm Right	2.59 mm Down
B	B Point	10.05 mm Anterior	1.99 mm Right	1.83 mm Down
Me	Menton	11.80 mm Anterior	2.26 mm Right	2.22 mm Down

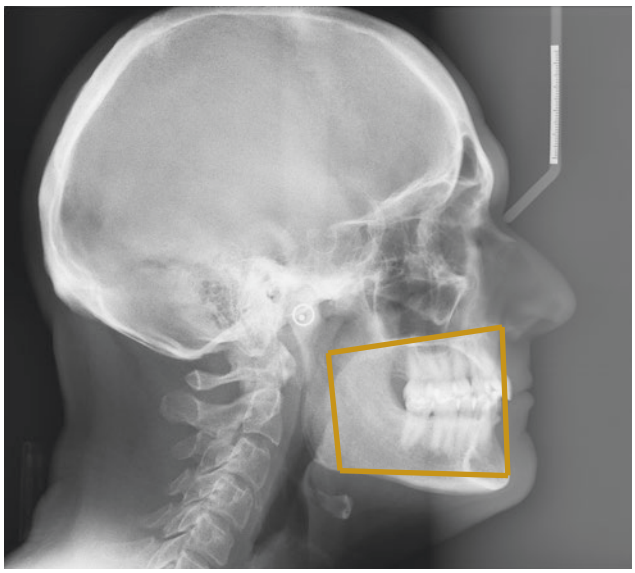

Fig. 33.6 Lateral cephalometric film demonstrating retroglossal airway crowding in a patient with a straight facial profile and very flat occlusal plane angle

desired, create intraoperative surgical cutting and/or repositioning guides to help make certain the intended tubercle is actually captured during surgery.

A common cause of obstructive sleep apnea is idiopathic condylar resorption or temporomandibular joint degenerative disease. These entities result in a predictable constellation of findings including sleep-disordered breathing, chronic facial pain, convex facial profile with mandibular/chin retrusion, high mandibular plane angle, impaction of posterior dentition, and anterior open bite malocclusion. The management of these patients has engendered some controversy historically, with options generally including traditional orthognathic surgery, costochondral graft ramus reconstruction combined with maxillary surgery, and alloplastic TMJ prosthesis insertion combined with maxillary surgery. It is not within the scope of this manuscript to debate the merits of these three approaches, as all have advantages and disadvantages. Each patient's clinical situation is unique, and all approaches merit consideration.

The approach most utilized by this author is alloplastic joint reconstruction with maxillary surgery in the majority of skeletally mature patients who are at least middle-aged and particularly in those patients who have persistent TMJ symptoms despite aggressive nonsurgical conservative therapy. Patients who are younger and whose symptoms can be controlled with conservative therapy are better candidates for traditional orthognathic surgery, understanding that some of these patients may go on to need further surgery due to relapsing symptoms or further condylar degeneration.

Figure 33.12 demonstrates the lateral cephalometric image of a 43-year-old female with severe OSA and progressive (over many years) loss of chin projection with TMJ pain and condylar degeneration. Conservative treatment of her facial pain failed to provide adequate pain relief nor prevent further condylar degeneration. Her sleep-disordered breathing clearly had progressed in terms of her historical recollection of the development of SDB symptoms a number of years after the development of her TMJ disorder.

As in other cases of maxillomandibular advancement for OSA, the advancement of the anterior mandibular region must be adequate to relief the multilevel obstruction, with an emphasis on moving pogonion point forward at least 15 mm. These patients in particular usually can be advanced even further than this, as shown in Figs. 33.13 and 33.14 that demonstrate a

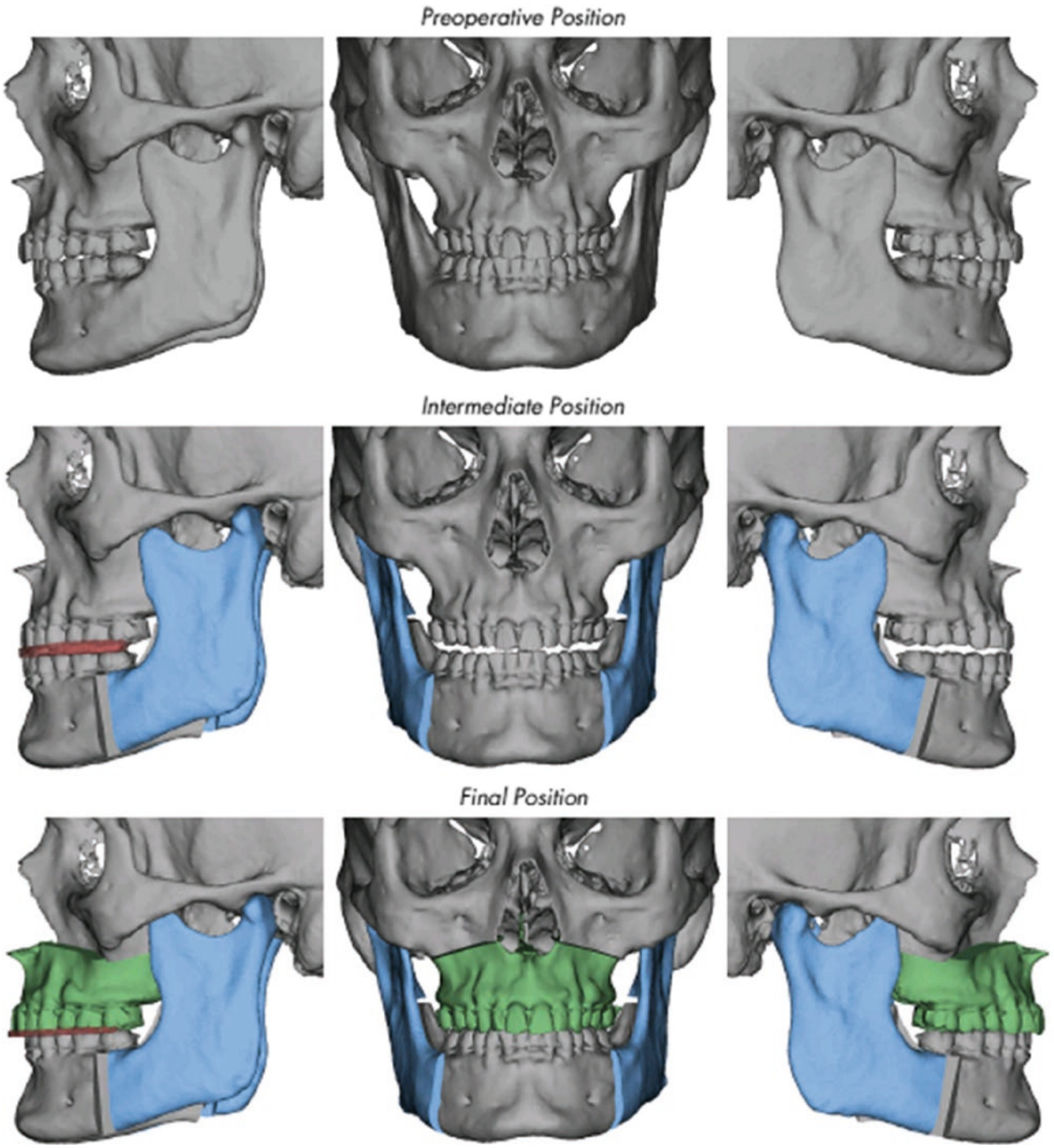

Fig. 33.7 Note the extremely flap occlusal and mandibular plane angles which precludes CCW rotation to achieve adequate genial region advancement

21 mm advancement at pogonion. Virtual planning also permits the construction of an accurate 3D printed model that can be used to fabric the custom TMJ prosthesis. Historically, this process was much less predictable; 3D model printing permitted accurate duplication of the *pre-surgical* anatomy, and this model could then be segmented and reconstructed manually to represent the intended post-surgical anatomy. Given the desire to accurately transfer the intended surgical plan to the patient at surgery, this was a less than confidence-inducing process. Modern virtual surgical planning techniques with the ability to print a 3D model in the intended post-surgical anatomy are a significant advancement, permitting consistent results with high

Fig. 33.8 Note the nearly identical pogonion dimensional change as compared to previous case treated with CCW rotation

Point	Name	Anterior /Posterior	Left/Right	Up/Down
ANS	Anterior Nasal Spine	6.98 mm Anterior	0.10 mm Left	2.15 mm Up
A	A Point	7.32 mm Anterior	0.07 mm Right	1.93 mm Up
ISU1	Midline of UpperIncisor	8.00 mm Anterior	0.00	2.00 mm Up
U3L	Upper Left Canine	7.50 mm Anterior	0.26 mm Right	1.66 mm Up
U6L	Upper Left Anterior Molar (mesiobuccal cusp)	7.24 mm Anterior	0.60 mm Right	1.21 mm Up
U3R	Upper Right Canine	8.42 mm Anterior	0.24 mm Right	1.68 mm Up
U6R	Upper Right Anterior Molar (mesiobuccal cusp)	8.58 mm Anterior	0.58 mm Right	1.22 mm Up
ISL1	Midline of Lower Incisor	8.12 mm Anterior	0.19 mm Left	4.21 mm Up
L6L	Lower Left Anterior Molar (mesiobuccal cusp)	8.19 mm Anterior	0.20 mm Left	2.59 mm Up
L6R	Lower Right Anterior Molar (mesiobuccal cusp)	8.21 mm Anterior	0.20 mm Left	2.61 mm Up
B	B Point	9.60 mm Anterior	0.20 mm Left	4.07 mm Up
Pog.	Pogonion	11.11 mm Anterior	0.21 mm Left	4.55 mm Up

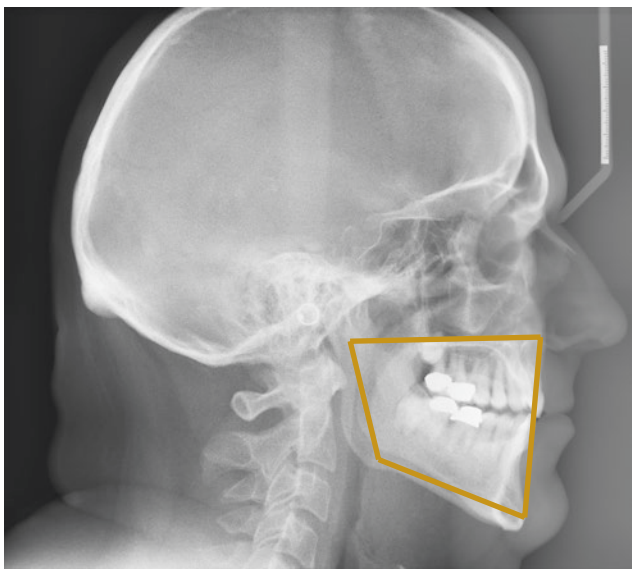

Fig. 33.9 Lateral cephalometric film showing severe retroglossal airway crowding with occlusal plane angle that will permit some degree of CCW rotation to maximize advancement of the chin and genial region

levels of confidence that airway obstruction can be relieved (see Fig. 33.15).

Nontraditional osteotomies (other than standard LeFort and sagittal split osteotomies) are sometimes necessary in order to adequately manage cases where the amount of advancement would render traditional osteotomy technique at risk of osseous healing problems or relapse over time. It is therefore logical that these types of osteotomies would play an outsize role in the management of OSA patients, as the amount of AP advancement needs to be maximized in OSA patients as compared to routine orthognathic surgery patients.

Figure 33.16 depicts the lateral cephalometric film of a 16-year-old female patient with idiopathic condylar resorption and moderately severe obstructive sleep apnea planned for maxillomandibular advancement. This patient's ramus anatomy was such that traditional SSRO was thought to be risky due to the amount of advancement needed and the resulting lack of bony overlap within the ramus. Therefore an inverted-L osteotomy was chosen for the mandibular procedure, to be combined with maxillary LeFort I and chin advancement. The virtual surgical plan is as shown in Fig. 33.17, utilizing mandibular first sequence, followed by maxilla then chin. In addition, Figure 33.18 shows cutting guides that were printed to help accurately place the osteotomies, as well as templates that were utilized to contour the iliac crest corticocancellous blocks that were inserted into the segmental ramus defects.

Finally, maxillomandibular advancement utilizing distraction osteogenesis can be utilized in select cases. Some examples include cases where the magnitude of advancement of the maxilla or mandible is such that traditional osteotomies would end up with inadequate overlap, revision surgeries, and edentulous patients in whom surgical splints may not be adequate to create intraoperative rigid fixation.

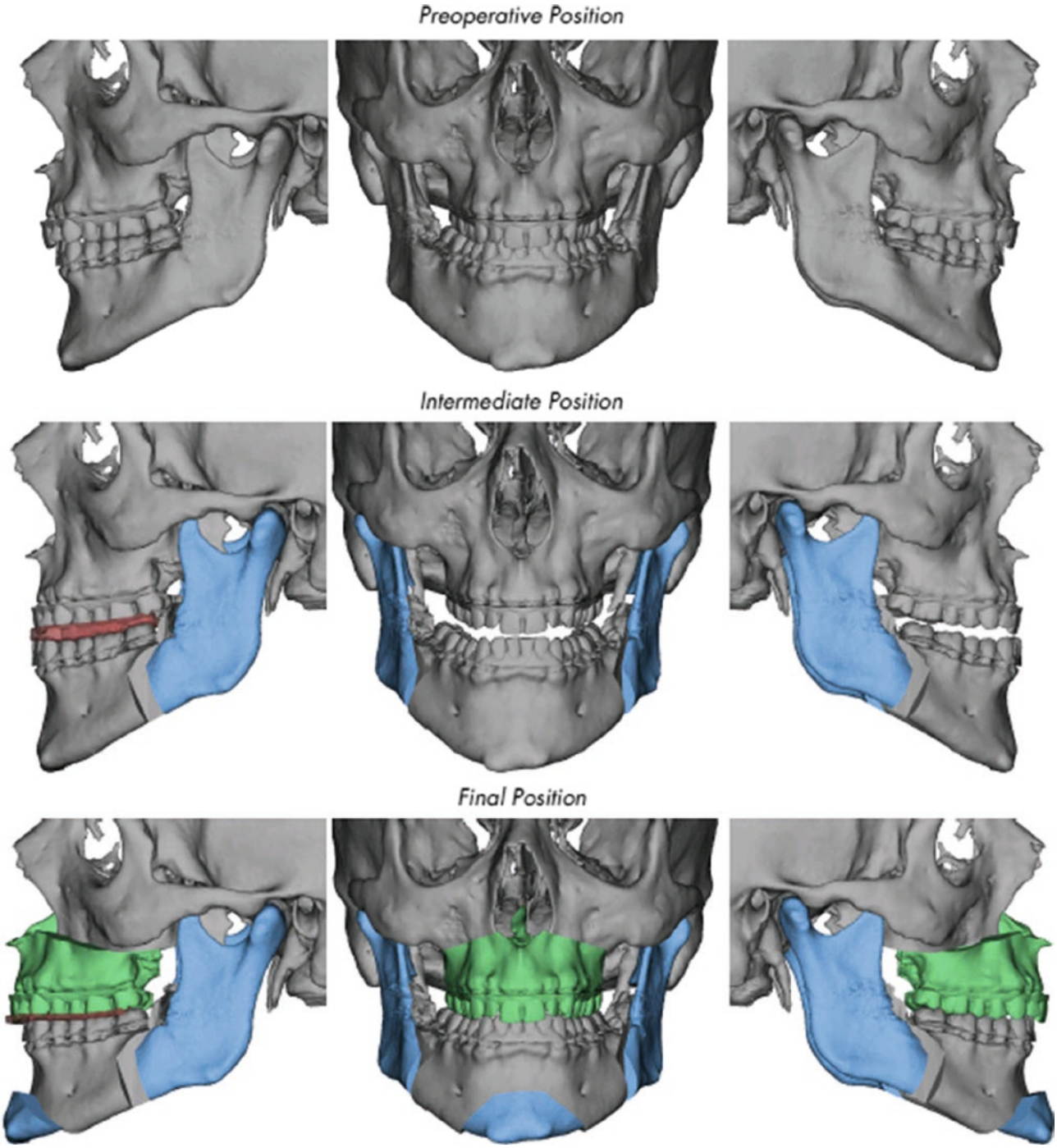

Fig. 33.10 Virtual planning images showing CCW rotation with trapezoidal chin advancement to specifically advance the genial tubercles. Also note the improved shape and orientation of the intermediate surgical splint

Figure 33.19 shows a 53-year-old male patient with persistent severe OSA who had previously undergone an initial genioglossus advancement which failed, followed by maxillomandibular advancement both of which were ultimately unsuccessful in relieving his OSA. Virtual surgical planning was used to plan a maxillomandibular advancement using distraction osteogenesis due to the anticipated amount of anterior movement that might be

Fig. 33.11 Note the differential increase in advancement achieved with CCW rotation from ANS to B point through to pogonion

Point	Name	Anterior /posterior	Left/right	Up/down
ANS	Anterior Nasal Spine	8.20 mm Anterior	1.00 mm Right	3.27 mm Up
A	A Point	8.58 mm Anterior	1.00 mm Right	2.93 mm Up
ISU1	Midline of Upper Incisor	10.00 mm Anterior	1.00 mm Right	3.00 mm Up
U3L	Upper Left Canine	9.89 mm Anterior	1.00 mm Right	2.49 mm Up
U6L	Upper Left Anterior Molar (mesiobuccal cusp)	9.79 mm Anterior	1.00 mm Right	1.23mm Up
U3R	Upper Right Canine	9.91 mm Anterior	1.00 mm Right	2.45 mm Up
U6R	Upper Right Anterior Molar (mesiobuccal cusp)	9.79 mm Anterior	1.00 mm Right	1.12 mm Up
ISL1	Midline of Lower Incisor	9.79 mm Anterior	1.00 mm Right	2.74 mm Up
L6L	Lower Left Anterior Molar (mesiobuccal cusp)	9.79 mm Anterior	1.00 mm Right	1.29 mm Up
L6R	Lower Right Anterior Molar (mesiobuccal cusp)	9.72 mm Anterior	1.00 mm Right	1.38 mm Up
B	B Point	11.33 mm Anterior	1.00 mm Right	2.57 mm Up
Pog.	Pogonion	18.29 mm Anterior	1.00 mm Right	4.84 mm Up

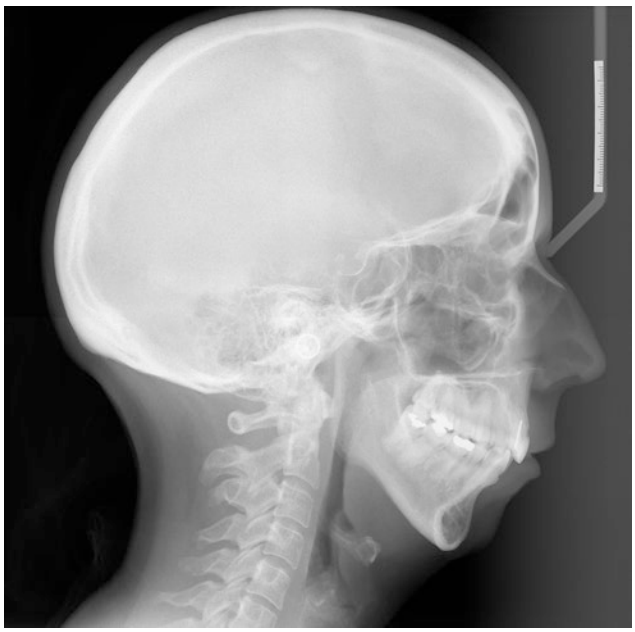

Fig. 33.12 Lateral cephalometric image showing the typical stigmata of airway collapse, clockwise occlusal plane rotation, high mandibular plane angle, impaction of posterior dentition, and lack of mandible/chin position

needed in this patient who had been resistant to previous operations (see Fig. 33.20).

After virtual planning, 3D printed models can be created and used in DO cases to pre-surgically modify and adapt the distractors (see Figs. 33.21 and 33.22). This permits accurate vector recreation that is faithful to the surgical plan and also saves operating room time. In addition, cutting and placement guides can also be constructed to be used intraoperatively.

The post-surgical post-distraction surgical result is shown in Fig. 33.23, which clearly demonstrates a significant improvement in the airway AP dimension. Polysomnography demonstrated complete resolution of the patient's formerly persistent OSA.

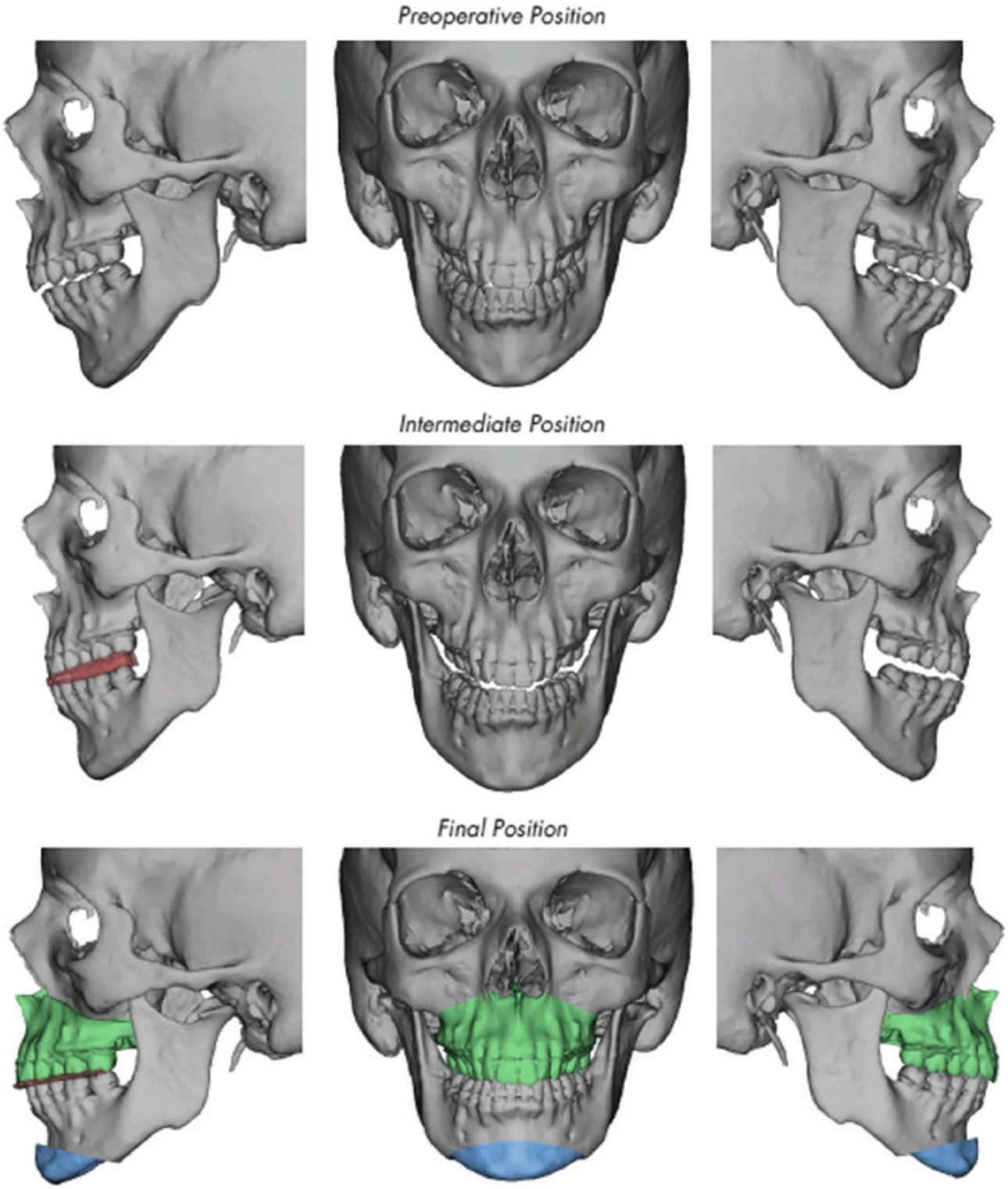

Fig. 33.13 Virtual surgical planning demonstrating the intended surgical reconstruction, including LeFort I osteotomy with anterior repositioning of mandible followed by total joint replacement

Fig. 33.14 Note progressive increase in AP advancement from ANS to incisors and then on to B point and pogonion due to the significant degree of counterclockwise rotation

Point	Name	Anterior /posterior	Left/right	Up/down
ANS	Anterior nasal spine	2.32 mm Anterior	0.62 mm Left	5.79 mm Up
A	A point	4.50 mm Anterior	0.39 mm Left	5.02 mm Up
ISU1	Midline of upper incisor	8.00 mm Anterior	0.00	5.00 mm Up
U3L	Upper left canine	7.61 mm Anterior	0.06 mm Left	3.56 mm Up
U6L	Upper left anterior molar (mesiobuccal cusp)	7.01 mm Anterior	0.14 mm Left	1.66 mm Up
U3R	Upper right canine	7.62 mm Anterior	0.05 mm Left	3.99 mm Up
U6R	Upper right anterior molar (mesiobuccal cusp)	7.14 mm Anterior	0.12 mm Left	2.16 mm Up
ISL1	Midline of lower incisor	8.53 mm Anterior	0.96 mm Left	7.93 mm Up
L6L	Lower left anterior molar (mesiobuccal cusp)	7.63 mm Anterior	0.78 mm Left	4.16 mm Up
L6R	Lower right anterior molar (mesiobuccal cusp)	8.53 mm Anterior	0.76 mm Left	4.39 mm Up
B	B Point	13.06 mm Anterior	0.70 mm Left	5.78 mm Up
Pog	Pogonion	21.08 mm Anterior	0.55 mm Left	7.19 mm Up

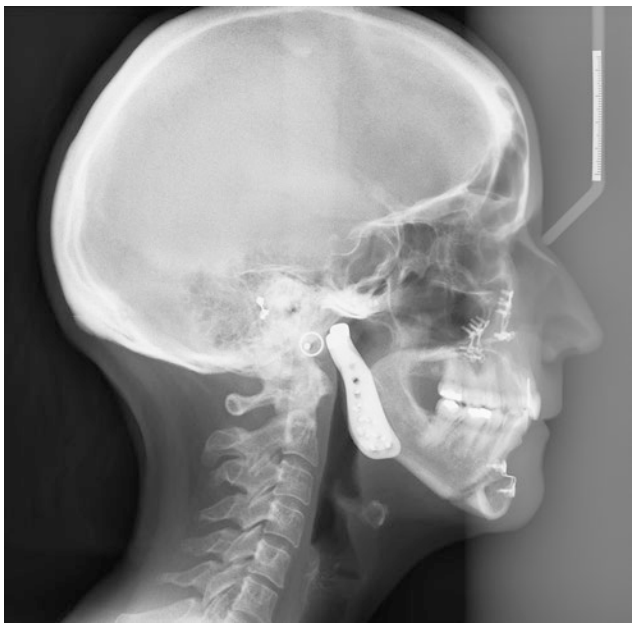

Fig. 33.15 Postoperative lateral cephalometric image showing post-surgical anatomy including total joint replacements, occlusal plane rotation, improved chin projection, and, most importantly, relief of the airway collapse in the retroglossal region

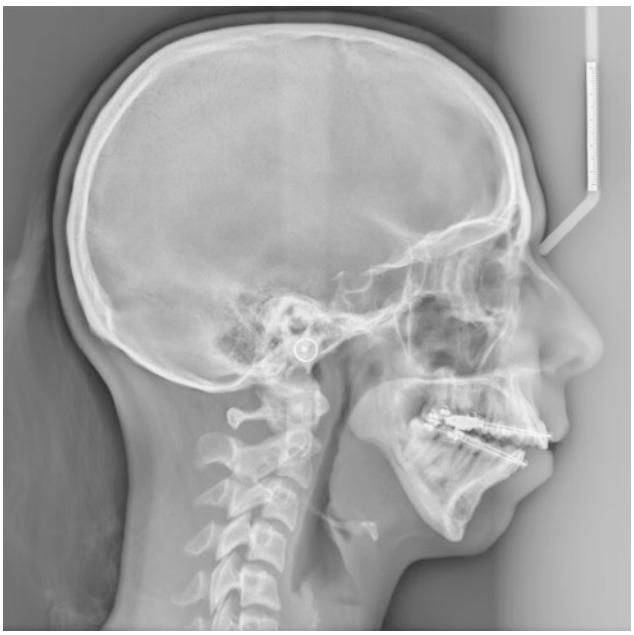

Fig. 33.16 Lateral cephalometric film showing severe condylar resorption, clockwise mandibular rotation, apertognathia, and severe airway obstruction

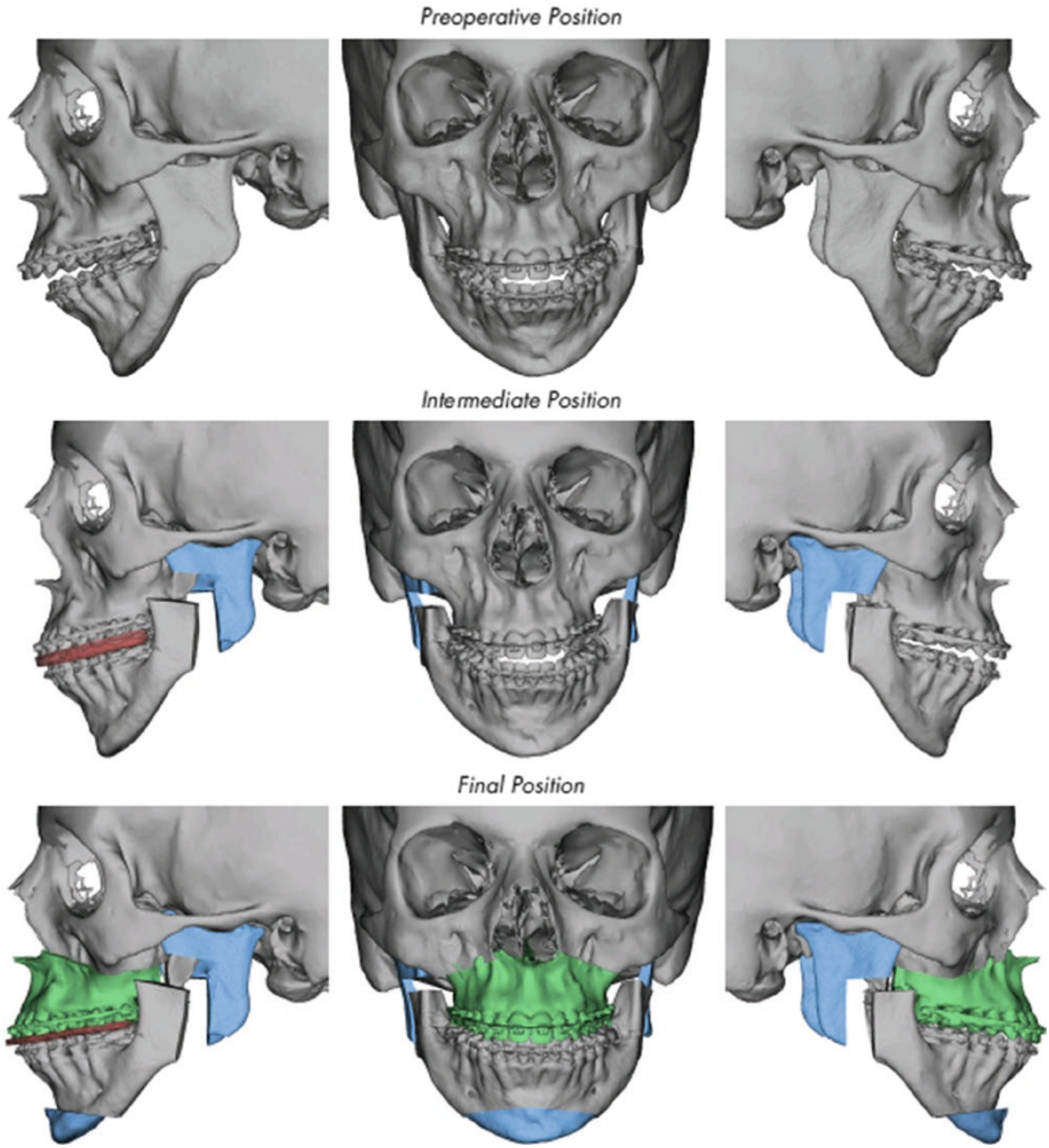

Fig. 33.17 Virtual surgical plan showing mandibular-first surgery utilizing inverted-L osteotomy

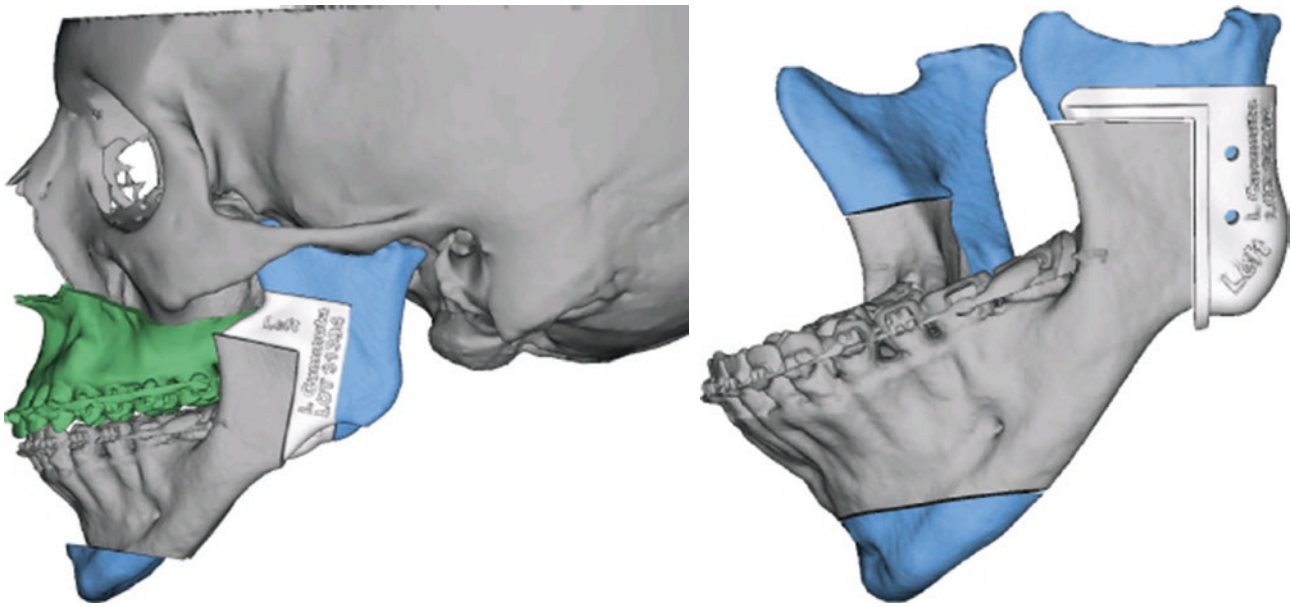

Fig. 33.18 Showing cutting guide and bone graft template for left mandibular inverted-L osteotomy

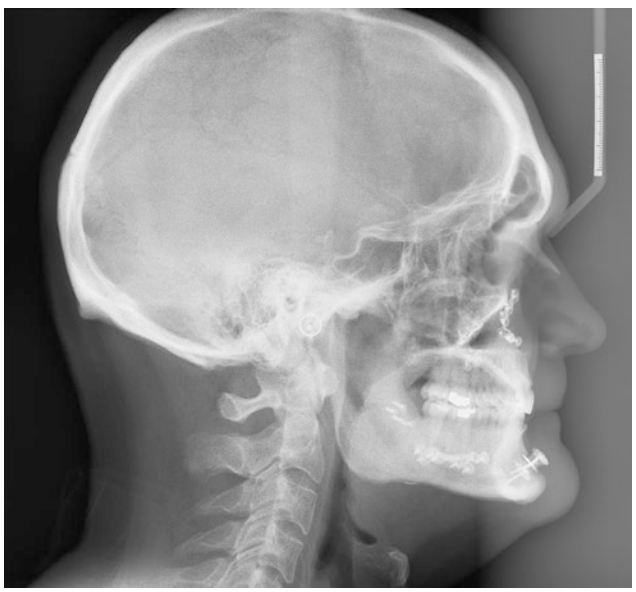

Fig. 33.19 Lateral cephalometric film showing patient who is post-GGA and post-MMA elsewhere who has residual severe OSA due multilevel obstruction and planned for bimaxillary distraction

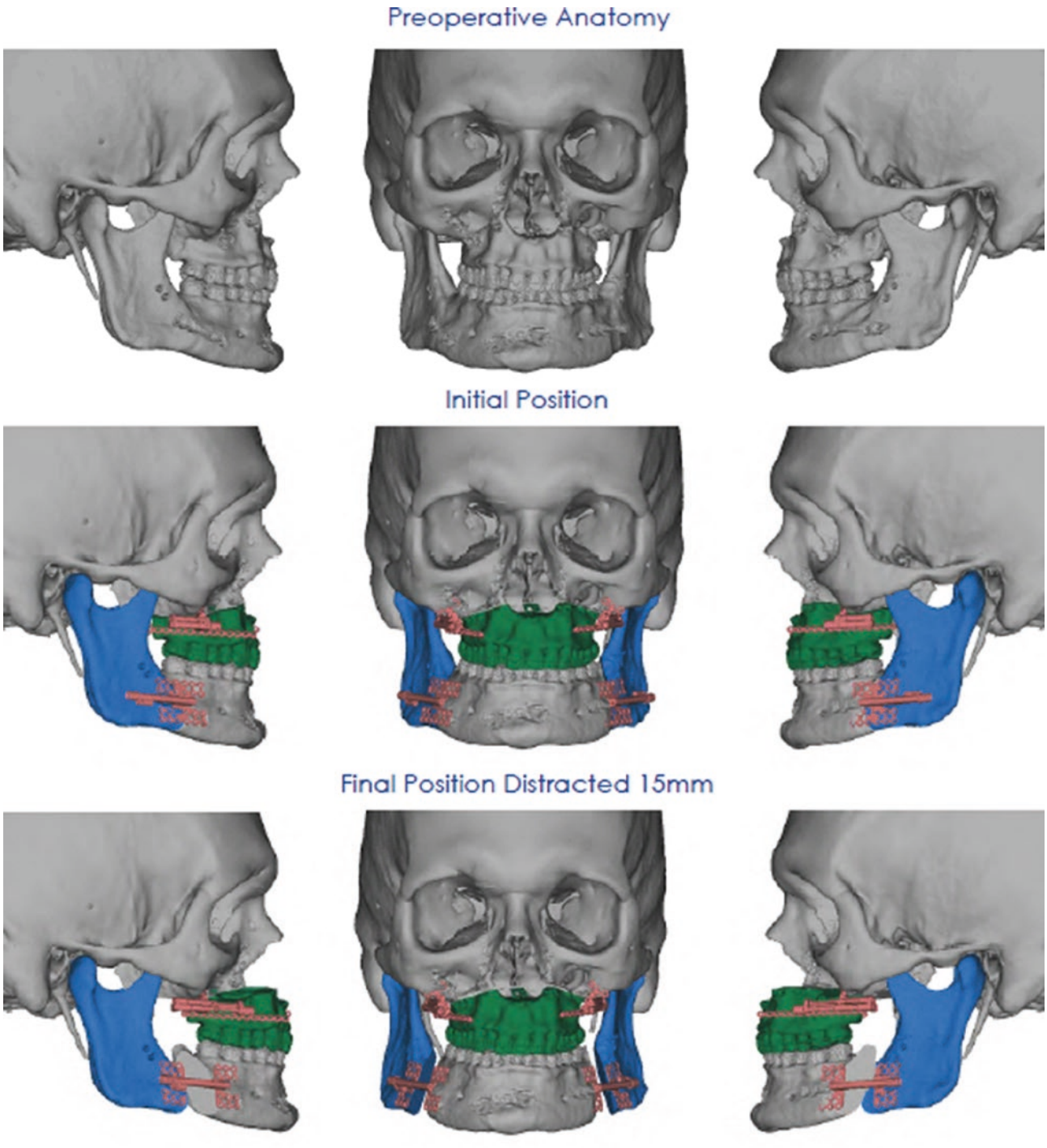

Fig. 33.20 Virtual surgical planning showing planned secondary osteotomies, placement of distractors to create appropriate vector of movement, and planned final position of segments

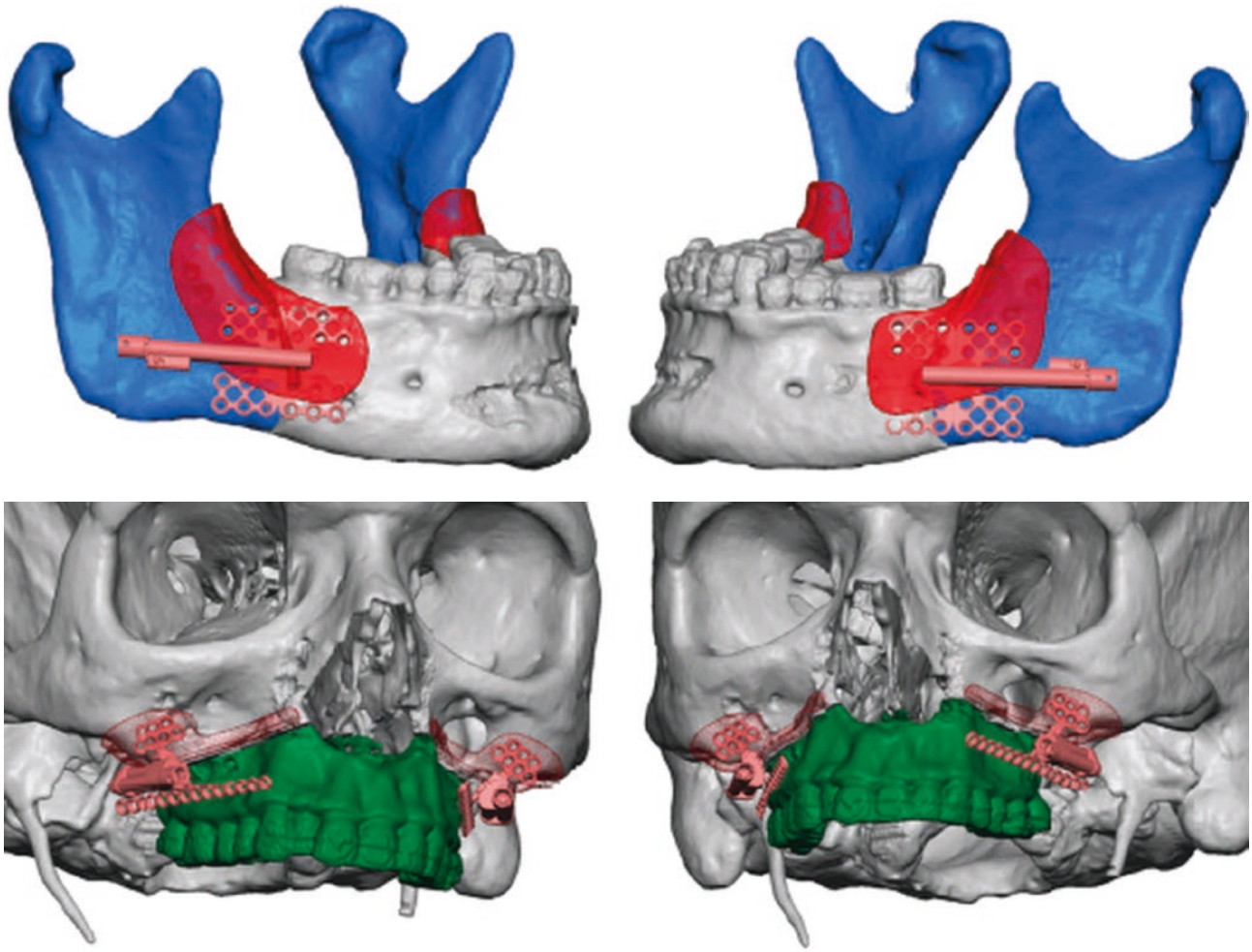

Fig. 33.21 Cutting and positioning guides constructed virtually

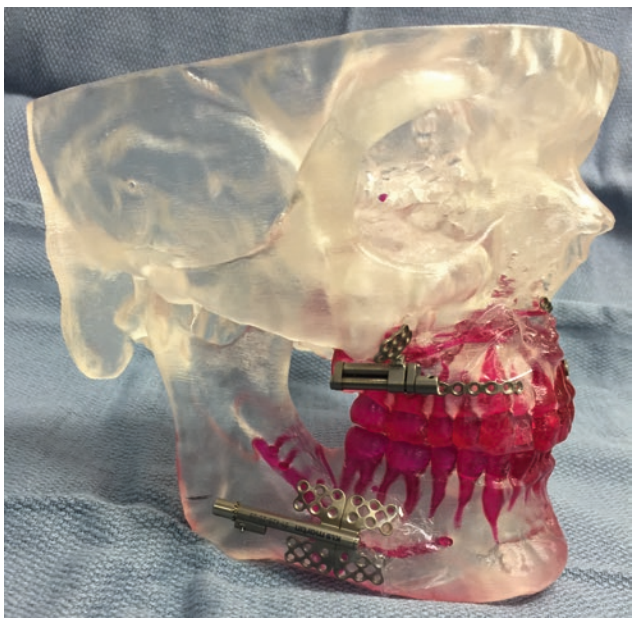

Fig. 33.22 Showing 3D printed stereolithographic model, permitting transfer of virtual surgical plan to the model, with presurgical modification of distractors

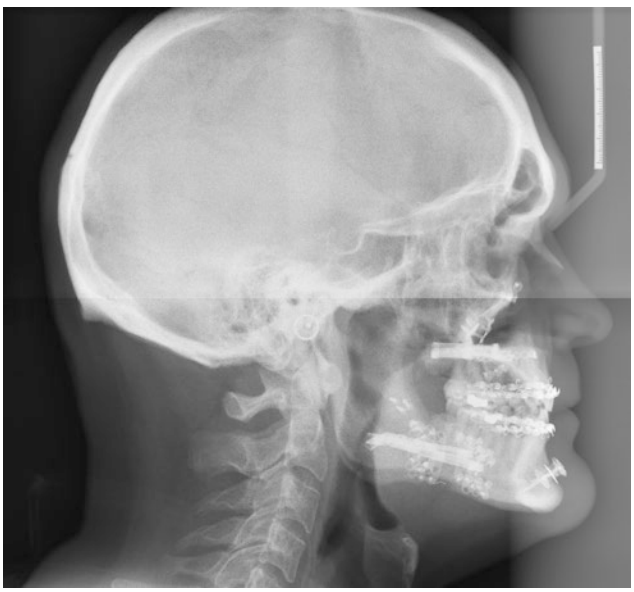

Fig. 33.23 Lateral cephalometric film demonstrating accurate transfer of virtual plan to the facial skeleton, as well as clear anterior repositioning of the maxilla and mandible, with improved airway dimension

33.3 Conclusions

Osseopharyngeal reconstruction via maxillomandibular advancement (and other operations on the facial bones) is primarily focused on opening the airway. As such, it is critical that the surgeon be certain that the planned surgical treatment will actually move the bones and their soft tissue attachments both in the proper direction and by the appropriate dimensional amount to give a high likelihood of success. Virtual surgical planning permits this type of precision to a degree that was not previously possible with two-dimensional methods. In addition, planning and execution of complex osseopharyngeal reconstruction, wherein temporomandibular joint replacement, nontraditional osteotomies, and distraction osteogenesis are used, accrue advantages when done in a three-dimensional manner.

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Temporomandibular Joint Reconstruction

Louis G. Mercuri

Contents

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Key Points

1. Alloplastic TMJ replacement devices do not require a donor site.
2. Alloplastic TMJ replacement devices require less surgery time.
3. Custom alloplastic TMJ replacement devices can be designed and manufactured to conform to the anatomical situation.
4. Alloplastic TMJ replacement device components are not susceptible to prior failed foreign body particles, local, or systemic pathology.
5. Immediately after alloplastic TMJ replacement device implantation, a patient can begin physical therapy hastening regaining mandibular function.

34.1 Synopsis

Presently, there are two options for the reconstruction of the temporomandibular joint: autogenous bone grafting or alloplastic joint replacement. This chapter presents evidence-based advantages and disadvantages for each of these management options to assist both the surgeons and their obstructive sleep apnea patients in making that choice should this option be required to manage the case.

34

34.2 Introduction

Temporomandibular joint (TMJ) reconstruction (TJR) presents unique problems because of the integral role the TMJ plays in establishing and maintaining proper mandibular form and function. The TMJ not only acts as a secondary growth center for the mandible, but its integrity is vital to the functions of mastication, speech, and deglutition, as well as in obstructive sleep apnea (OSA) airway support [1].

TMJ TJR goals are (1) improvement of mandibular function and form, (2) reduction of further suffering and disability, (3) containment of excessive treatment and cost, and (4) prevention of further morbidity [2]. End-stage disease and/or pathology such as OSA, with accompanying anatomic form and physiologic function distortions dictate consideration for TMJ TJR.

The surgeon presented with an OSA patient requiring TMJ TJR has two options, either autogenous or alloplastic reconstruction. This chapter presents an evidence-based discussion of the advantages and disadvantages of autogenous and alloplastic TMJ TJR to assist both the surgeon and their patients in making that choice in the management of OSA.

34.3 Autogenous TMJ Replacement

Autogenous bone grafting has been reported to be "the gold standard" for reconstruction of developmental deformities, end-stage TMJ pathology, and ankylosis using either free or vascularized bone grafts from rib [3], calvarium [4], clavicle [5], iliac crest [6], or fibula [7].

In addition to the reported unpredictability of autogenous bone grafting [8–12], complications frequently occur. Complications associated with bone harvest have been reported up to 19% of cases and include chronic pain, skin sensitivity disorders, and complicated wound healing. This can lead to hypertrophic scarring or infection, fracture, and prolonged length of hospitalization, all associated with additional morbidity and medical costs [13, 14].

The costochondral graft has been the most frequently recommended autogenous bone graft for TMJ reconstruction due to its ease of adaptation to the recipient site, its gross anatomical similarity to the mandibular condyle, and its demonstrated growth potential in skeletally immature patients [3, 15–19].

Reitzik reported that in an analogous situation to autogenous costochondral grafting, cortex-to-cortex healing after vertical ramus osteotomy requires 20 weeks to consolidate in monkeys and 25 weeks in humans [20].

Maxillomandibular fixation is typically maintained for some period in patients after TMJ reconstruction with costochondral grafts. Despite rigid fixation, graft micromotion will invariably occur with early mandibular function. This results in shear stresses on the graft/host interface that potentially can lead to poor neovascularization, nonunion, or failure [21].

In a systematic review of the literature, Kumar et al. assessed the growth potential of costochondral graft for TMJ reconstruction. These authors concluded that there were no randomized clinical trials, and the only evidence is in the form of case series, considered the lowest level of evidence for any study. Therefore, no inference can be interpreted regarding growth potential of costochondral graft. Thus, based on available evidence, they concluded that use of costochondral graft for TMJ reconstruction for its growth potential lacks scientific evidence [22].

The advantages of an autogenous bone graft for TMJ reconstruction:

1. Availability – Part of the human skeletal system. No lead-time to purchase and acquire device components.
2. Biocompatibility – Autogenous tissue, therefore, little concern for issues of biocompatibility or hypersensitivity.

3. Adaptability – Autogenous bone can be shaped at surgery to adapt to the lateral surface of the mandible and glenoid fossa.
4. Less expensive – Alloplastic TMJ replacement components are expensive. No need to maintain an inventory of expensive alloplastic TMJ replacement components and specialized instruments or equipment.

The disadvantages of an autogenous bone graft for TMJ reconstruction:

1. Requires a second surgical donor site.
2. Longer surgery and anesthetic time – Simultaneous autogenous bone harvest and preparation of mandibular implantation sites are most often not technically feasible.
3. Potential morbidity associated with autogenous bone harvesting.
4. Requires neovascularization, bone turnover, and bone healing.
5. Delays physical therapy – Orthopedic surgeons understand that early physical therapy increases the range of motion of reconstructed joints [23]. Keeping a patient immobilized (maxillomandibular fixation) after any open joint surgery, particularly joint replacement, increases muscle atrophy, as well as periarticular fibrosis and the potential for the development of heterotopic ossification and ankylosis [24].
6. Bone is subject to foreign body reactions, local and systemic pathology – Henry and Wolford concluded that a foreign body reaction locally influenced the success of autologous tissue reconstruction [25]. This principle holds true in cases of high inflammatory arthritic diseases, OSA, and condylar resorption [26, 27].
7. Higher relapse potential when autogenous bone grafting to reconstruct the TMJ is combined with orthognathic surgery – Reconstruction of the loss of posterior vertical mandibular height and dental occlusion, as seen in end-stage arthritic disease, condylar resorption, and many cases of OSA, requires counterclockwise rotation of the mandible along with maxillary surgery [27]. This maneuver places great stress on the mandibular condyle. Relapse has been reported high when autogenous costochondral grafting has been used to reconstruct the condyle in such cases [28–30].

34.4 Alloplastic TMJ Replacement

With the potential morbidity associated with harvest of autogenous bone and the inability of these tissues to survive either the transplantation process or the functional demands applied to them, there arose the need for the development and use of alloplastic materials to replace them anatomically and functionally.

The practice of reconstructive orthopedic surgery would be unthinkable and impossible without the availability of alloplastic joint replacement devices. In the 1960s, posed with the problem that resection arthroplasty as an uncertain procedure with recurrent deformity and limited motion as common complications, Sir John Charnley developed a successful low-friction total alloplastic joint replacement device. Since that time, with the evolution of surgical techniques, implant materials and designs, excellent long-term function and quality of life improvement results have been reported along with device survival rates exceeding 90% after 10 years [31, 32].

Over the years, surgeons dealing with end-stage TMJ pathology unable to be managed predictably with autogenous bone grafting developed alloplastic total TMJ replacement systems [33, 34].

Presently, the two US FDA-approved total alloplastic TMJ replacement systems (TMJ Concepts, Ventura, CA and Zimmer Biomet, Jacksonville, FL) have demonstrated long-term successful outcomes in management of end-stage TMJ pathology (Fig. 34.1).

The results of studies comparing the presently available FDA-approved alloplastic TMJ replacement support the surgical implantation of both stock and custom systems. Further, these studies demonstrate that alloplastic TMJ replacement is safe and effective, reduces pain, improves mandibular function, and patients' quality of life, with few complications. Therefore, alloplastic TMJ replacement represents a viable and stable long-term solution for cranio-mandibular reconstruction in patients with irreversible end-stage TMJ disease [35–51].

Lee et al. reviewed published research on TMJ total replacement that compared the outcomes of autogenous costochondral graft and alloplastic TMJ reconstruction. Using PubMed databases, including prospective, retrospective, case–control or longitudinal studies and significant statistical analysis, these authors divided outcomes into "Acceptable" or "Non-acceptable." These authors discovered seven articles that dealt with costochondral graft in 180 patients. Most patients had good outcomes (n = 109, 61%). They found six articles with 275 patients who had undergone alloplastic TMJ replacements. Those patients had excellent outcomes (n = 261, 95%). These authors concluded that alloplastic total joint reconstruction resulted in increased quality of life and fewer complications in comparison with autogenous costochondral grafting. Therefore, alloplastic TMJ replacement was deemed more effective for total joint replacement than costochondral grafting [52].

The advantages of alloplastic TMJ replacement:

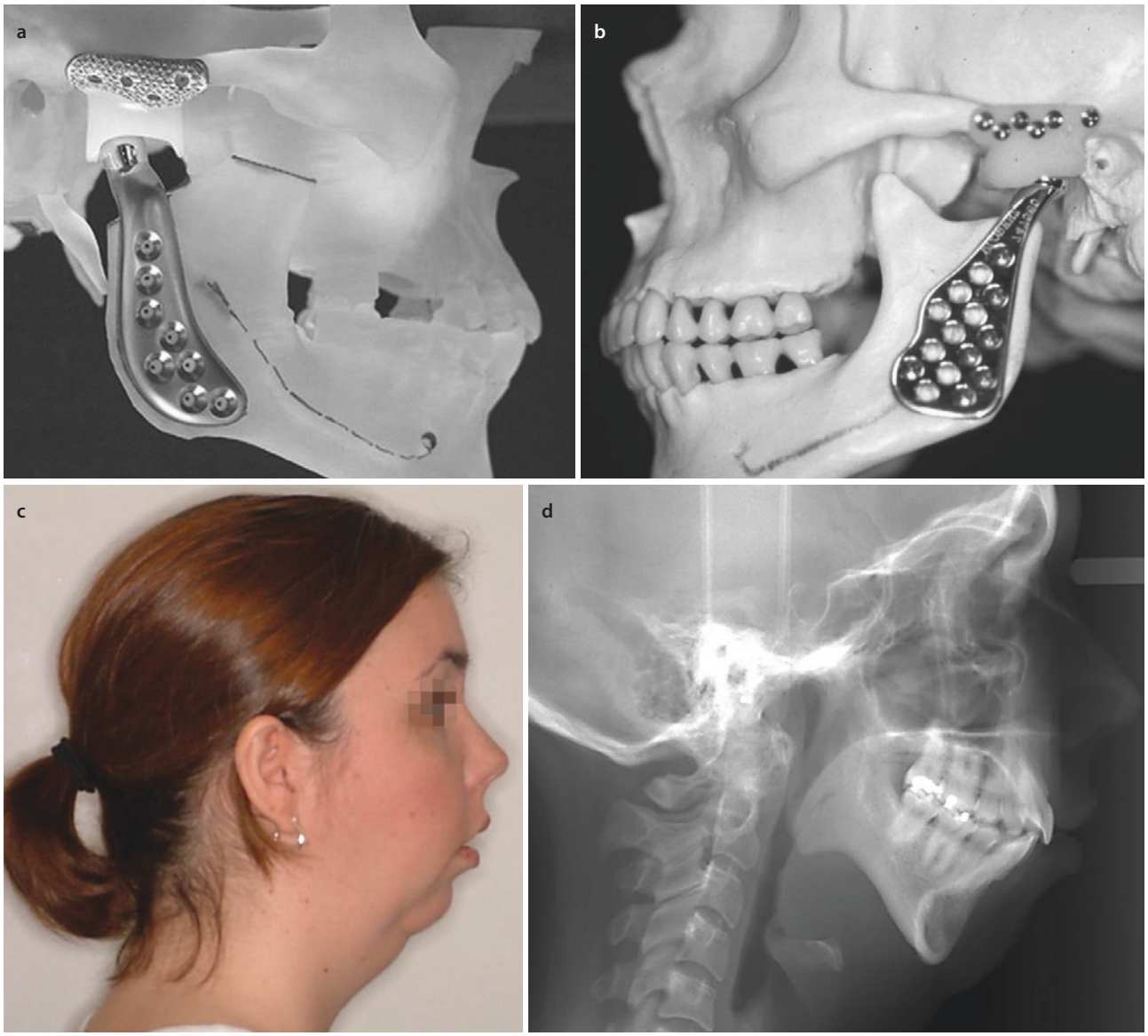

Fig. 34.1 **a** TMJ Concepts (Ventura, CA) total TMJ replacement device consisting of a commercially pure titanium mesh backed ultrahigh molecular weight polyethylene fossa component and an alloyed titanium ramus component with a cobalt/chrome/molybdenum condyle. **b** Zimmer Biomet (Jacksonville, FL) total TMJ replacement device consisting of an all ultrahigh molecular weight polyethylene fossa component and an all cobalt/chrome ramus component

1. Availability – Stock systems can be inventoried for use as needed. Custom devices can be ordered in advance.
2. No donor site morbidity.
3. Decreased surgery time – No donor site.
4. Conforms to the anatomical situation – In the case of a stock system, the surgeon will alter the host bone to allow the components to fit. A custom system provides the surgeon with components that are designed and manufactured for the specific anatomical situation.
5. Components are not susceptible to prior failed foreign body particles, local, or systemic pathology.
6. Patient can begin physical therapy immediately as there is no concern for neovascularization and component mobility.

The disadvantages of an alloplastic TMJ replacement:

1. Expense – Since the operating room, anesthesia, and surgical time charges are much less than with autogenous costochondral graft harvest and implantation, the total cost of alloplastic TMJ replacement is less or at least comparable.
2. Longevity of the components. Studies indicate that alloplastic TMJ replacement devices have a lifespan of at least 10–20 years [37, 38, 41, 42].

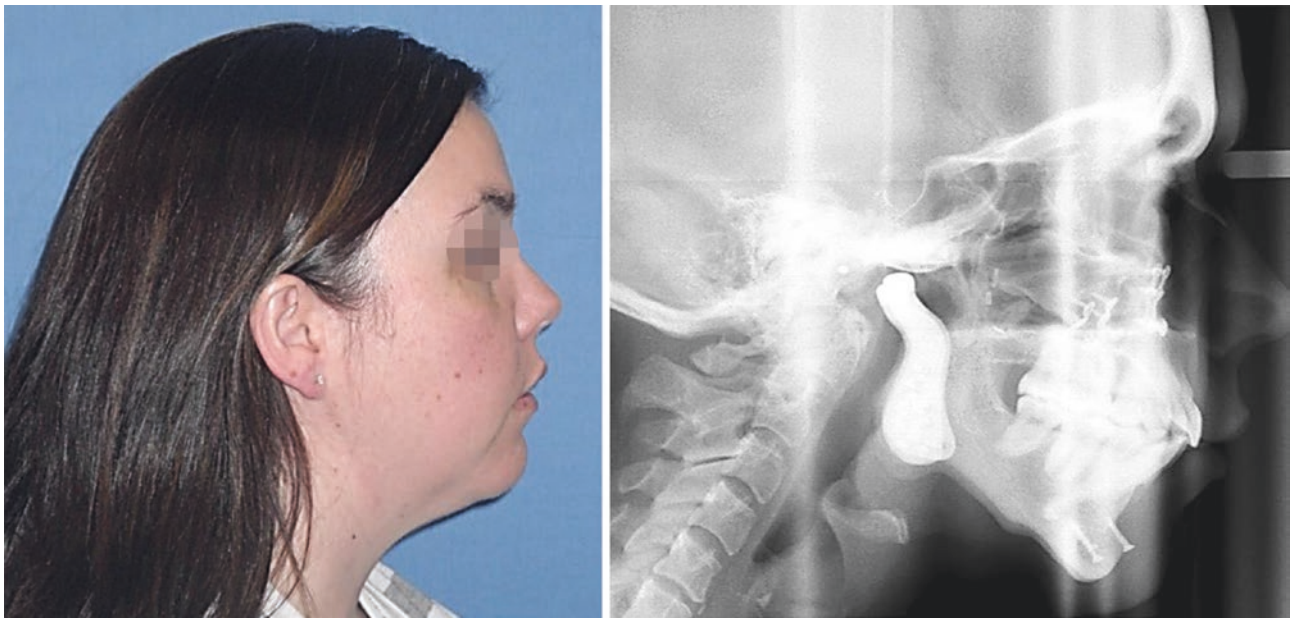

Fig. 34.2 TC preoperative clinical and lateral cephalometric images. Note the restricted airway

3. Material hypersensitivity – Excessive reactivity to implant debris or hypersensitivity to implant debris is relatively rare, where it is estimated that only 1–3% of aseptic failures are due to hypersensitivity responses among traditional metal-on-polymer type total joint replacement hip and knee designs. The percentage of aseptic failures due to biomaterial hypersensitivity in alloplastic TMJ replacement is not known [53].
4. Only indicated for skeletally mature patients – It appears to be myopic to continue to reoperate in children with failed, overgrown, or ankylosed costochondral grafts, with autogenous TMJ replacements, using the same modalities that failed, when there may be an appropriate solution available. These patients would be benefit from undergoing alloplastic TMJ replacements knowing that, depending on functional growth, revision and/or replacement surgery may be required in the future, rather than incurring continued failures of autogenous grafting that will very likely also require future surgical intervention [54, 55].

34.5 Case

TC was a 27-year-old female who presented for consultation regarding maxillomandibular orthognathic surgery to manage her OSA. Polysomnography documented an apnea–hypopnea index (AHI) of 31.1/hour. She was prescribed and had been using continuous positive airway pressure therapy (CPAP), but this modality was becoming an issue between her and her spouse (Fig. 34.2).

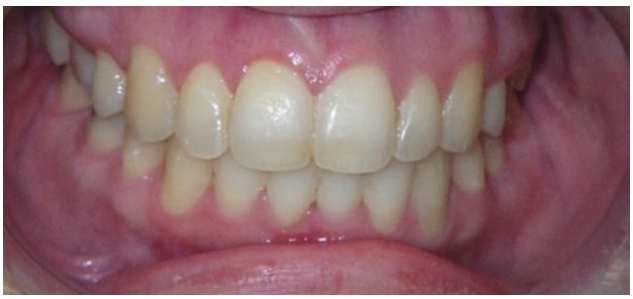

Fig. 34.3 TC 5 years postoperative clinical and lateral cephalometric images. Note the enhanced airway

After clinical, radiographic examinations, TMJ, and orthognathic workups, TC and her spouse were presented with a treatment plan that included bilateral TMJ replacements with patient-fitted prostheses to increase her posterior vertical dimension and advance her mandible, LeFort I osteotomy to align her maxilla with the mandibular advancement, and an advancement genioplasty. Since she had a Class I, well interdigitated, and stable occlusion, orthodontics was not considered necessary (Fig. 34.3).

After the surgery, TC was able to discontinue the use of her CPAP and her AHI improved to <10/hour. She has maintained her occlusion and AHI for 5 years (Fig. 34.4).

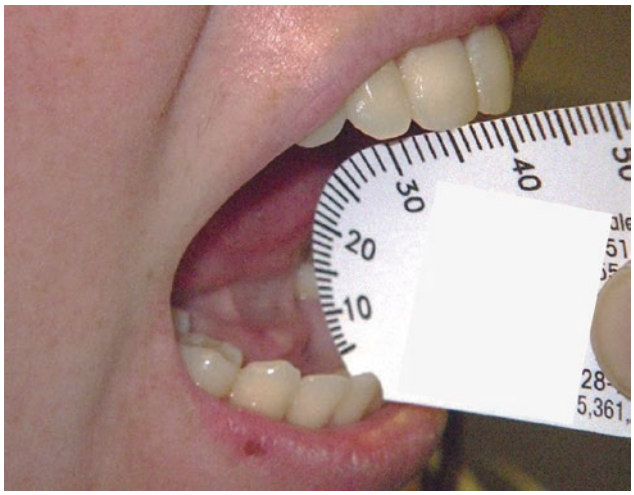

Fig. 34.4 TC 5 years postoperative occlusion and maximum interincisal opening

34.6 Summary

The current literature supports isolated mandibular advancement as an efficacious treatment modality for adult OSA in select patients with mandibular insufficiency [56]. Therefore, based on the evidence cited, alloplastic TMJ replacement appears to provide the most predictable functional and esthetic outcomes for replacement of the TMJ in patients with end-stage disease and pathology resulting in symptoms of OSA.

Disclosure Statement Dr. Mercuri is compensated by TMJ Concepts as a Clinical Consultant and maintains stock in that company.

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Maxillomandibular Advancement Using Total Joint Replacement for the Treatment of Obstructive Sleep Apnea

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Contents

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35.1 Introduction

Obstructive sleep apnea (OSA) is the most common sleep-related breathing disorder in the USA [1, 2]. The prevalence of moderate-to-severe OSA is estimated to be up to 30% in males and 15% in females [3, 4]. This multisystemic disease process can result in an increased risk of acute myocardial infarction, increased odds for stroke, decreased quality of life, and premature death [5–10].

End-stage temporomandibular joint (TMJ) diseases frequently cause resorption and destruction of the temporomandibular joint [11–13]. Connective tissue diseases, autoimmune diseases, idiopathic condylar resorption, condylar fractures, or severe osteoarthritis have been implicated in anteroposterior loss of mandibular projection [14–17]. The subsequent loss of vertical height and mandibular projection from these maladies results in posterior oropharyngeal airway collapse that either exacerbates or causes OSA.

Typically, maxillomandibular advancement (MMA) using conventional intraoral osteotomies is a very effective surgical treatment for obstructive sleep apnea, especially in the hypoplastic or retrognathic mandible [18]. In a patient with airway collapse caused by TMJ degeneration, MMA by conventional orthognathic surgical procedures may not be possible or stable due to their degenerated mandibular condyles. In such cases, MMA can be achieved with insertion of an artificial alloplastic total joint prosthesis (TJP) to advance the mandible. The MMA with TJP corrects the posterior airway collapse, subsequent OSA, and inhibits relapse of the mandible [19–22].

This chapter discusses the preoperative evaluation, treatment planning, and surgical technique used to treat a patient with OSA caused by TMJ degeneration utilizing TMJ total-joint replacement.

35.2 History of MMA

Traditionally, MMA is a surgical technique for advancing both the maxilla and the mandible. The usual osteotomies for this are a Le Fort I maxillary osteotomy and a bilateral sagittal split mandibular osteotomy. The Le Fort I osteotomy owes its origins to Rene Le Fort, who described the natural planes of maxillary skeleton fracture in cadaveric specimens [23]. Axhausen first utilized the Le Fort I osteotomy to completely mobilize the maxilla, but Obwegeser was the first to recognize that complete mobilization of the maxilla was necessary to reduce relapse [24, 25]. Many modifications to the procedure have been trialed and tested since that time to develop the surgical techniques we adhere to today [26–32].

Mandibular advancement was first described as a treatment for obstructive sleep apnea in 1979. Kuo et al reported three cases of mandibular advancement for the successful correction of "hypersomnia sleep apnea," a clinical equivalent of OSA [33]. Bear et al continued the use of mandibular advancement, using bilateral C-osteotomies and iliac corticocancellous grafts for surgical cure of OSA [34]. Later, concomitant maxillary advancement supplemented mandibular advancement to preserve dental occlusion [35]. In the setting of severe temporomandibular joint disease, the bilateral sagittal split osteotomy (BSSO) does not target the primary source of retrognathia and soft tissue collapse. Dysplastic or degenerated temporomandibular joints may require replacement with alloplastic devices for stable correction of upper airway collapse, dentofacial deformities, and dental malocclusion.

TMJ TJPs have had a surge of success in product design, surgical technique, and long-term stability for orthognathic procedures. Previous total joint replacement systems had failure in material durability and product design [36]. Current TJPs have proven invaluable in treating both TMJ pathology and dentofacial deformities with long-term stability and success [37, 38]. The natural evolution of alloplastic TMJ TJPs has been their application in concomitant orthognathic surgery.

The BSSO has some limitations when advancing the mandible compared to a TJP. A TJP, especially a patient-specific TJP, has greater flexibility than an SSRO in the magnitude of mandibular advancement and in correcting asymmetric dentofacial deformities, especially for restoring posterior facial height (◘ Fig. 35.1). Degeneration of the temporomandibular joint as a result of internal disc derangement, reactive arthritis, idiopathic condylar resorption, or other end-stage TMJ pathology can result in impressive dentofacial deformities requiring large mandibular advancements to treat the malocclusion and associated comorbidities [39–41]. Custom TMJ TJPs allows the reconstructive surgeon the ability to create a patient-fitted joint that is specific to each patient's anatomy and treatment goals [42].

If the patient has a dentofacial deformity caused by TMJ degeneration that requires reconstruction, or if the patient has OSA and needs an advancement but the TMJs are not stable enough to withstand a conventional BSSO, then a TJP should be considered. In patients with concomitant TMJ pathology, total joint replacement has evolved into an effective treatment option for obstructive sleep apnea as if by necessity. The advancement of oropharyngeal structures caused by the repositioning of the maxilla and mandible by total joint replacements treats both the redundant oropharyngeal tissue collapse during sleep and the causative TMJ disease.

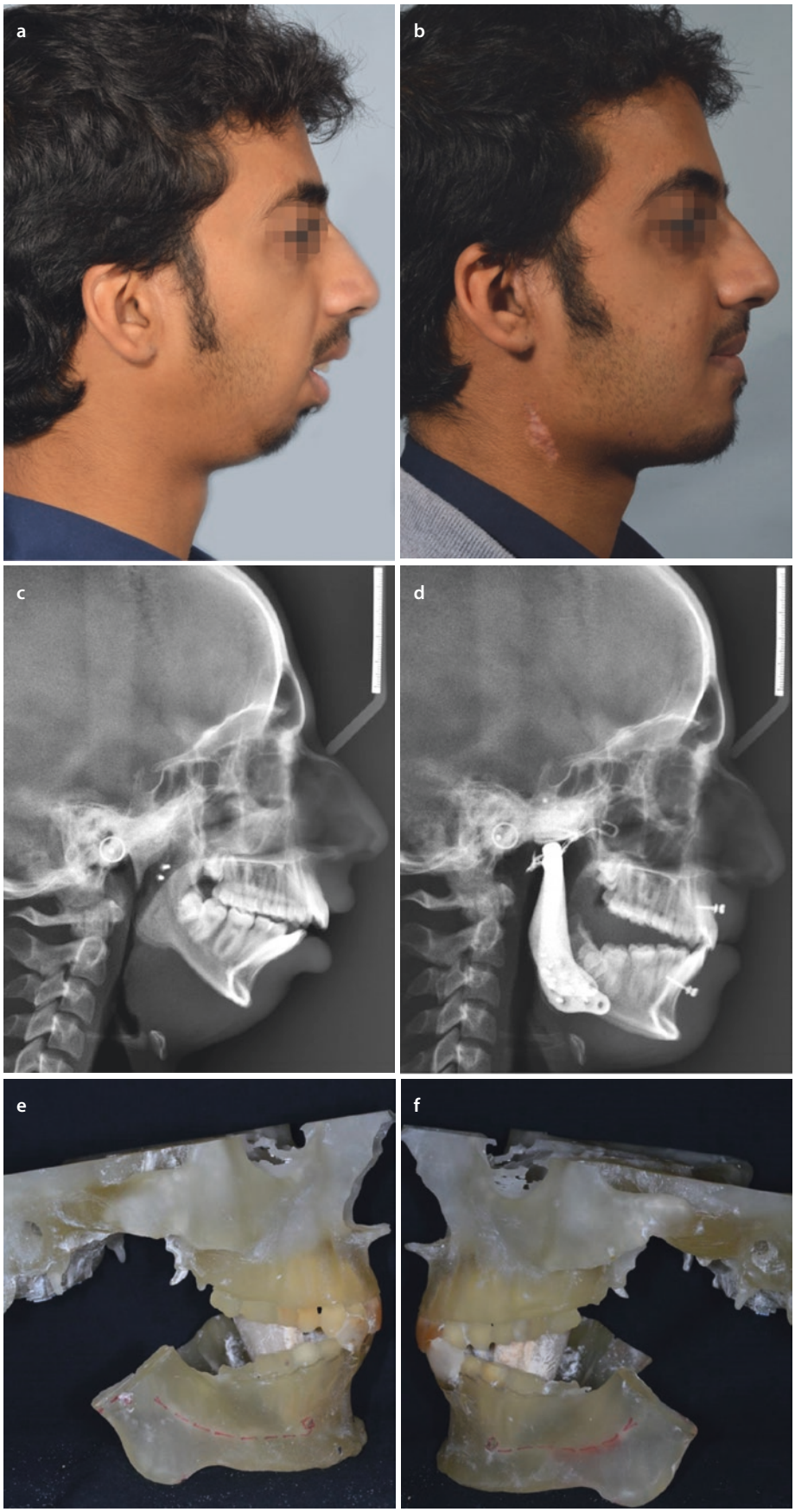


Fig. 35.1 This patient presented with bilateral condylar ankylosis that initiated at an early developmental age. He developed a severely retrognathic mandible, decreased MIO, and difficulty with jaw function. His treatment included a two-step approach. First with bilateral condylectomies, coronoidotomies, and TMJ total joint reconstruction with TMJ Concepts total joint prosthesis. The second stage with Le Fort I osteotomy was completed after orthodontic treatment. **a** Patient at initial presentation. **b** Patient after stage 1 treatment. **c** Preoperative lateral cephalograph. **d** Postoperative lateral cephalography. **e**, **f** Stereolithic model surgery with a large vertical movement after osteotomy to establish an appropriate mandibular vertical height. Large mandibular defects, such as this, illustrate the need for patient-fitted total joint prosthesis in order to provide a functional and esthetic outcome

35.3 Preoperative Evaluation

The treatment planning and surgical objectives of MMA for the treatment of obstructive sleep apnea using TJPs are very similar to classic orthognathic treatment planning. Both are grounded in clinical patient analysis, radiographic assessment, static model acquisition, radiographic interpretation, and treatment objectives based on each patient's dentofacial anatomy and the severity of their OSA.

Historically, MMA as treatment for obstructive sleep apnea has often required a minimum of 1 cm advancement for predictable reduction in postoperative apnea-hypopnea index (AHI) and stability of surgical cure [43–46]. One centimeter of MMA is frequently surpassed in patients with degenerative temporomandibular disease to achieve restoration of their lost facial equilibrium. TMJ patient-fitted TJPs allow for much greater advancements of the mandible and thus large airway corrections (Fig. 35.2) [22, 47–50].

A key difference between OSA treated by MMA versus treatment by TMJ TJPs with a maxillary osteotomy is that all of the patients in the latter category are undergoing advancement of their mandible because of a mandibular destructive process [11, 12, 39, 51]. Patients with posterior oropharyngeal collapse secondary to temporomandibular joint degeneration require not only replacement of the anterior-posterior projection of the mandible but also replacement of the vertical height of the ascending ramus and restoration of a functional occlusal plane [19].

Patients with TMJ disease present with differing dentofacial deformities depending on not only the disease etiology but also the time of onset during facial development. Most cases of early-onset mandibular condyle degeneration will require a Le Fort I osteotomy. These patients' facial development predictably compensate for mandibular retrusion with an increase in the maxillary occlusal plane angle with intrusion of the posterior maxillary dentition. In such cases, mandibular repositioning will rotate the mandible in a counterclockwise (CCW) fashion, creating a posterior open-bite that requires maxillary posterior downgraft (and often advancement) for occlusal restoration. This counter-

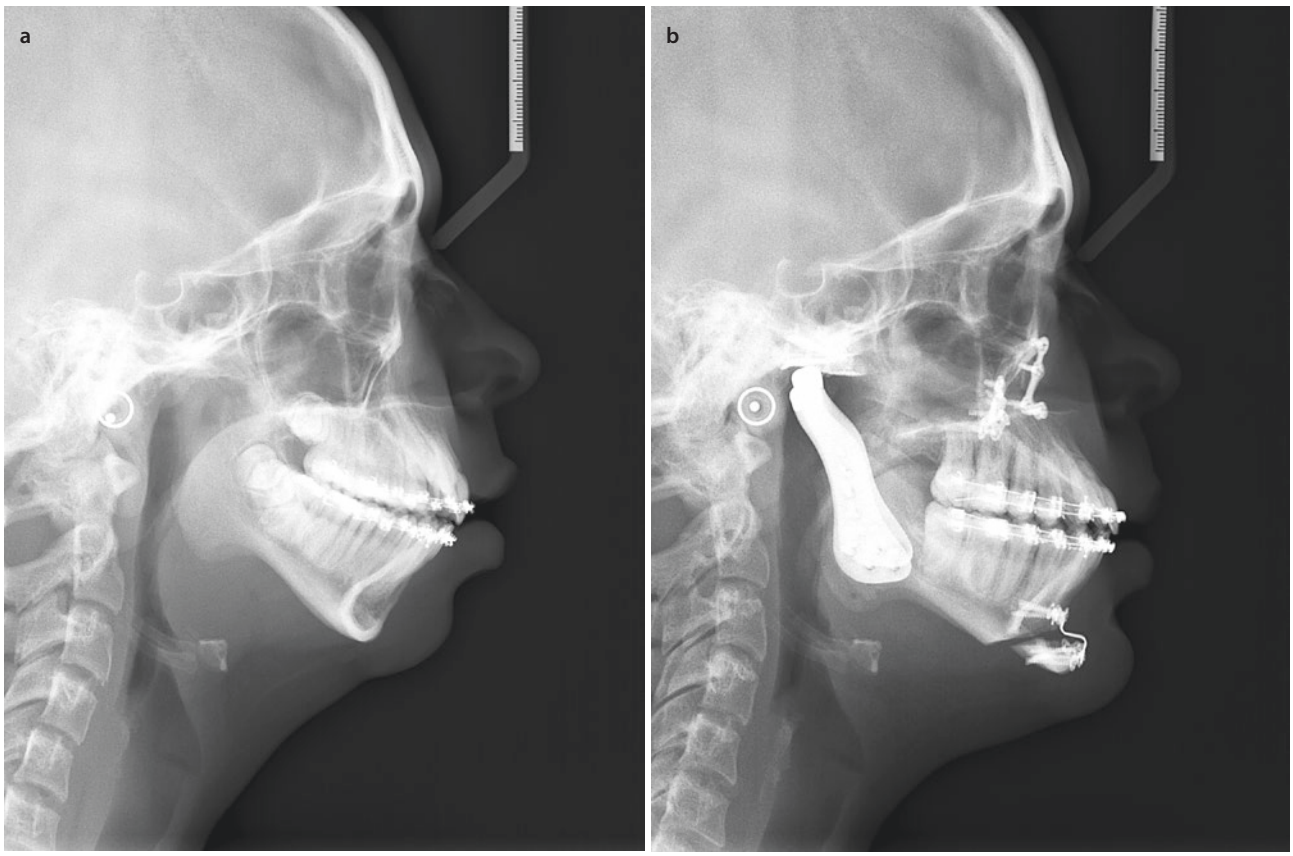


Fig. 35.2 **a** Preoperative lateral cephalograph demonstrating severe mandibular retrusion with a restricted posterior airway space (9.45 mm). **b** Postoperative lateral cephalography demonstrating bilateral TMJ total joint replacements with prosthesis, a Le Fort I osteotomy, and maxillomandibular advancement. Posterior airway space has increased with maxillomandibular advancement with TMJ total joint prosthesis (15.45 mm)

clockwise rotation of the maxillomandibular complex will not only correct the high occlusal plane angle but corrects the needed mandibular projection. The maxillary incisor is the key landmark used to plan and position the maxillomandibular complex in space paying attention to the yaw, pitch, and roll of the complex as it accommodates its final position. Another important factor when planning the maxillary position is preservation of the anterior nasal spine (ANS) in its preoperative position or slightly forward based on esthetic considerations. If the ANS is allowed to move posteriorly, there could be an unappealing decrease in nasal tip support.

Patients with late-onset TMJ degeneration may have a maxilla that is in a stable position compared to the now retrognathic mandible and oropharyngeal space. In these patients, a Le Fort I osteotomy is not always needed. Patient's with an anterior-posterior (A-P) discrepancy with adequate maxillary position may only need repositioning of their mandible for cosmetic and functional cure. This discrepancy must allow a mandibular advancement of at least 8–10 mm to be sufficient to open the posterior airway space.

Many patients will have a preexisting malocclusion mandating orthodontic therapy in conjunction with surgical intervention. This may not be typical of OSA patients where the preoperative occlusion is maintained, requiring only a relatively good occlusion to provide support for arch bars during surgery. As with other orthognathic surgery patients, most patients with preexisting malocclusions would benefit from orthodontic therapy to prepare the dentition for MMA.

In patients with severe mandibular degeneration, CCW rotation can be extremely beneficial in correcting both the high occlusal plane angle and deficient chin projection. The degree of chin projection from a CCW rotation of the maxillomandibular complex far exceeds the advancement possible from pure anterior-posterior advancement [52–57]. CCW rotation must be planned preoperatively with some orthodontic considerations. The postsurgical angulation of the maxillary incisor will be vastly different from the presurgical, orthodontic goals. Typically, the maxillary incisor angulation would increase in CCW rotation. It is important to communicate this to the orthodontist to plan accordingly.

35.3.1 History and Physical Examination

Determining patients that would benefit the most from TMJ TJP with MMA requires a thorough history and physical examination. An adequate history includes the patient's TMJ symptoms, when the symptoms began, how they progressed, and any past causative or contributing factors that may have attributed to their disease process. Usually a destructive symptomatic TMJ problem is easily identifiable in a radiograph. During the clinical exam of a patient who is referred to you for OSA and TMJ resorption, it is important to gather the following information: Has the patient had a history of TMJ orthognathic surgical interventions in the past? Have they had orthodontics? Are there parafunctional habits that could be contributing to their disease process? Does the patient or their family have any autoimmune or connective tissue disorders? [58] Patients require a thorough history to determine the prognosis of their TMJ condition and if MMA or other surgical interventions are a more appropriate treatment for their obstructive sleep apnea. Only when functional problems are found should one consider a TMJ replacement. Pain should never be the sole indication to operate on the TMJ.

The physical examination of a patient with condylar degeneration begins with a macroscopic assessment of facial symmetry and balance where the head is oriented with the Frankfort horizontal line parallel to the floor. Skeletal Class I, II, or III, facial profile, and gross facial asymmetries should be assessed. Palpation of the TMJs for crepitus and attention to clicks or pops may help to identify patients with a TMJ component to their dentofacial deformity. The patient should have a full muscular examination to document muscle pain and function. Maximal interincisal opening (MIO) and excursive movements should also be documented.

Intraoral examination of occlusal plane angle, transverse cant, or crossbites should be evaluated. In evaluating their Angle's classification of molar and canine relation, care must be taken when these patients close into centric occlusion (CO). Premature occlusal contacts may cause a functional shift in occlusion, or they may not physically be capable of creating a reproducible CO secondary to lack of condylar support.

35.3.2 Imaging

Most patients can be screened with a simple panoramic X-ray (Fig. 35.3). Treatment planning then utilizes medical-grade computed tomography (CT) scans or cone-beam computed tomography (CBCT) for a custom TMJ prosthesis.

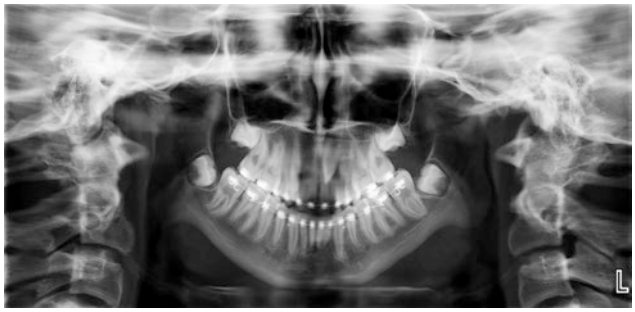
Cephalometric analysis continues to be the cornerstone of radiographic treatment planning in combined TMJ total joint replacement with MMA. Creation of a lateral cephalogram is imperative to treatment planning despite the onset of new imaging modalities such as CBCT. Cephalometric evaluation of patients with condylar degeneration often present with a mixture of features such as a retruded mandible, high occlusal plane

angle, posterior maxillary hypoplasia, Class II skeletal relationship, Class II malocclusion, and an anterior open bite.

Cone-beam computed tomography of the entire maxillofacial complex or a medical-grade CT with cuts that are less than 1 mm are recommended when planning for a patient-specific TMJ TJP (see below).

Patients with suspected early TMJ pathology or unilateral joint destruction will typically require imaging and assessment of their articular disc with a magnetic resonance image in the unaffected joint. (◘ Fig. 35.4). Magnetic resonance image (MRI) continues to be the gold standard to assess the position, shape, mobility, and degeneration of the TMJ disc and associated structures [59]. A patient with OSA due to a degenerated TMJ where bony changes and destruction are present on plane films or CT scans does not routinely require an MRI evaluation due to the advance state of joint destruction. However, when plain films or CT scans do not show severe degeneration, it becomes imperative that the TMJ is thoroughly analyzed with MRI for the health of the temporomandibular disc and to help determine progression of any disease. If the TMJ disc is salvageable, alternative treatment options may be available for correction of the patient's retrognathia (i.e., orthognathic surgery with or without disc repositioning) [40, 41, 60].

An instance where an MRI is helpful during treatment of severe mandibular condyle degeneration would be in unilateral conditions where an exam of the contralateral joint is necessary to decide if the healthy joint needs a procedure. If the patient has a unilateral TMJ condition, it is appropriate to modify the surgical technique to save the unaffected joint [61].



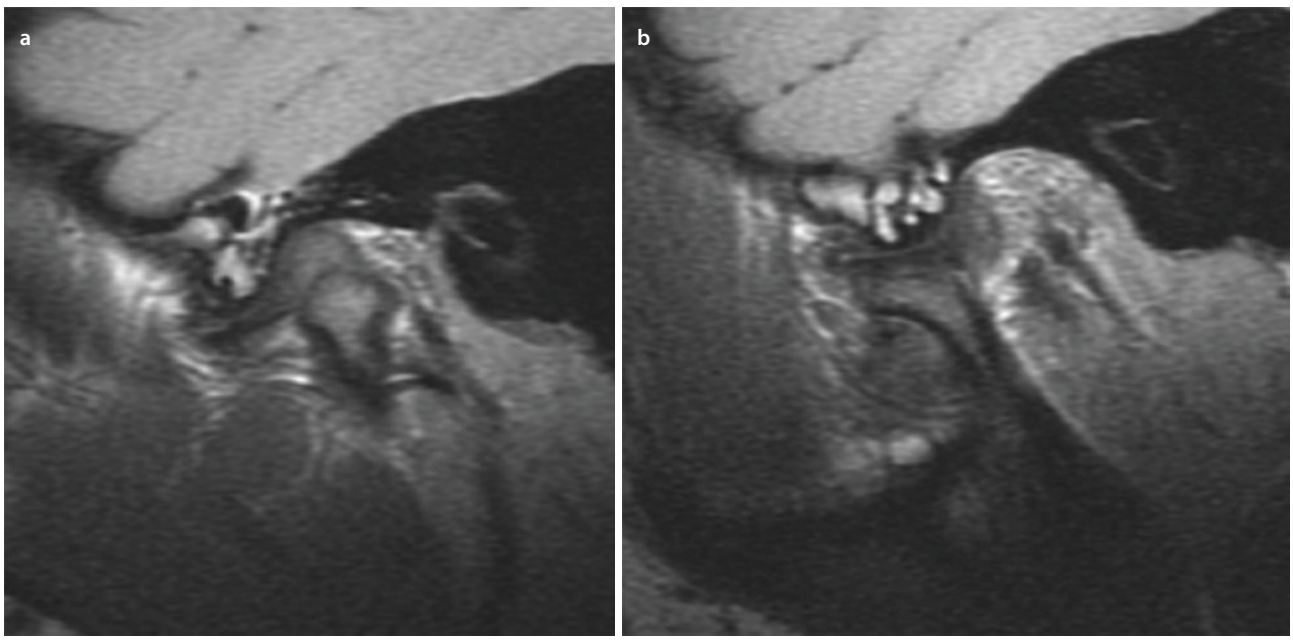
◘ **Fig. 35.3** Panoramic radiograph as screening with severely degenerated mandibular condyle

35.4 Joint Prosthesis Systems

35.4.1 Stock or Patient-Specific Prosthesis

In treatment planning of a patient for MMA with TMJ TJPs, the surgeon has many choices of surgical instrumentation but only two options for alloplastic TJPs: stock or patient-specific (also called custom or patient-fitted). Both systems have a fossa element and a condyle/ramus element of similar component materials but of vastly different designs (◘ Fig. 35.5).

While the efficacy of stock TMJ prostheses is comparable to TMJ patient-specific total joint prostheses



◘ **Fig. 35.4** **a** T1-weighted MRI scan of the left TMJ in a closed mouth position with anterior disc displacement. **b** T1-weighted MRI scan of the left TMJ in an open mouth position with reduction of the disc displacement

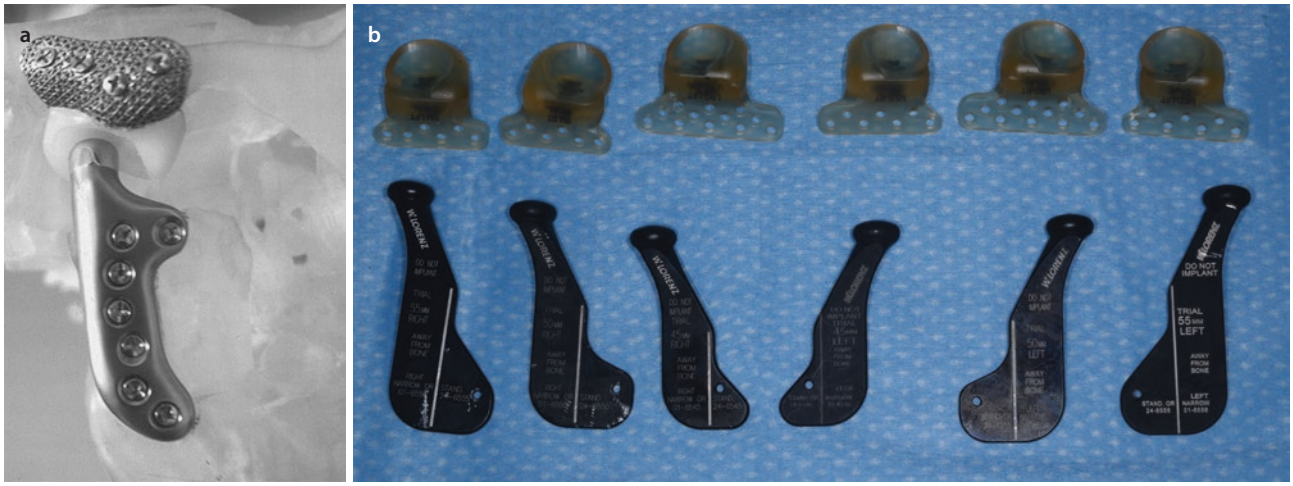


Fig. 35.5 **a** TMJ Concepts custom total joint prosthesis mounted on a stereolithic model. **b** Bilateral Zimmer Biomet stock TMJ replacement templates size 45 mm, 50 mm, and 55 mm

when it comes to restoring joint function, stock TJPs have some specific drawbacks when planning for a major MMA [62]. The prosthetic fossa element of a stock prosthesis has a prefabricated intaglio surface that requires significant bone recontouring in the patient's glenoid fossa in order to develop intimate contact of the prosthesis and bone for stable fixation. Adapting the prosthetic fossa can lead to additional surgery time or material fatigue and may lead to early implant failure. In the event that intimate contact is unattainable, a failure of osseointegration may occur [63]. Currently, the condyle/ramus component of stock TJPs available in the USA has lengths of 45 mm, 50 mm, and 55 mm [64]. The main drawback of stock prostheses for large anterior-posterior movements and/or CCW mandibular rotations is that the desired magnitude of mandibular repositioning often exceeds 55 mm. To achieve the desired mandibular advancement in the setting of diminished bone stock from condylar degeneration, restoration of the angle of the mandible is sacrificed with a stock prosthesis [65]. Further, the stock prosthesis available in the USA does not have a posterior stop to the fossa component, making the MMA more prone to posterior dislocation of the condyle.

The patient-specific TJP is created with each patient's anatomy integrated into the separate components. The prosthetic fossa element is designed to adapt intimately to the patient's fossa with no reduction required and with both an anterior and posterior stop. Not only does this reduce the amount of time required to place it, but it also ensures that, once fitted, the fossa component is where it was planned to be preoperatively. The main advantage of a patient-specific prosthesis is that in large mandibular advancements and/or CCW rotations, the condyle/ramus element can be tailored to accommodate the needs of the surgery and can also restore a deficient gonial angle (Fig. 35.6).

The long-term efficacy of a stock TMJ TJP versus a patient-specific TMJ total joint prosthesis has largely been reported in the success of each individual system rather than in a comparison between the two. Gerbino et al. reported on their experience over 14 years using both stock TMJ and patient-specific TMJ prostheses. Thirty-eight patients (55 joints) with appropriate recorded data were included in the study. Twenty-five patients had TMJ replacement with stock prostheses and 12 patients had TMJ replacement with custom-made prostheses. Each group had a minimum follow-up of 12 months. In both groups, quality of life and MIO improved in all cases [66].

The choice between patient-specific and stock prostheses is solely up to the surgeon. It is the authors' preference to use patient-fitted TMJ for MMA when possible.

35.5 Presurgical Steps for MMA with Total Joint Prostheses Using Computer-Assisted Surgical Simulation

Presurgical planning for MMA surgery with TJPs begins with clinical evaluation of the patient's TMJ and likely associated dentofacial deformities, acquiring dental models, recording centric relation (CR) in a bite registration, and obtaining images to include at minimum a CBCT or medical-grade CT scan. Traditional model surgery and stereolithic model manipulation were done in the past to fabricate splints and place the mandible into the new position for prosthesis fabrication. This

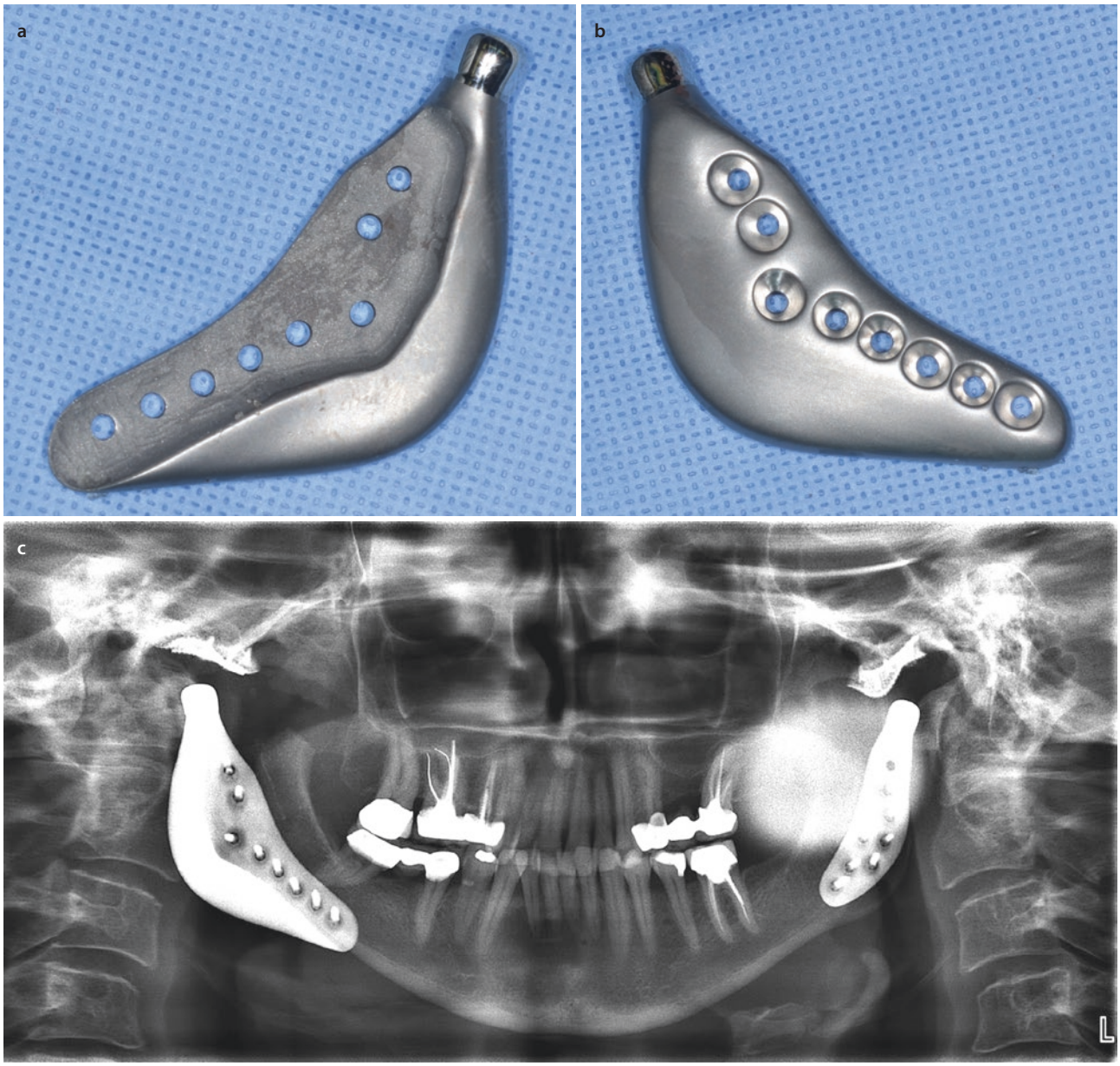


Fig. 35.6 Patient is a 56-year-old female with significant TMJ surgical history to include proplast total joint reconstruction of the right TMJ. The patient had failure of the implant system with a significant foreign body giant-cell reaction. A patient-fitted TMJ total joint prosthesis was designed in order to replace the destroyed mandibular osseous tissue. **a**, **b** Preoperative patient-fitted TMJ Concepts total joint prosthesis. **c** Postoperative panoramic radiograph

technique is not used routinely anymore and has been replaced by virtual surgical planning.

A stereolithic model for the construction of the TJP will be fabricated based on the CT. Special attention in obtaining the full TMJ, maxilla, and mandible in <1 mm cuts is paramount to an accurate prosthesis. A DICOM CT data set is uploaded to the TMJ prosthesis company to create a three-dimensional computer model. Dental models are also sent to the TMJ prosthesis company in order to input occlusion into the three-dimensional computer modeling. Dental models are not only necessary for treatment planning but also an adjunct in capturing dental occlusion for orthognathic surgical planning and splint fabrication. Current CT scanners cannot adequately capture the occlusal surfaces for splint fabrication.

The lateral cephalogram is traced according to the patient's surgical treatment objectives, and a prediction tracing is generated. This can be acquired by manipulation of the CBCT or medical-grade CT through imaging software. The patient's surgical treatment objectives and prediction tracing on the lateral cephalogram are

used to correct the dentofacial deformity and severe retrognathia with computer-simulated surgical MMA. The TMJ prosthesis company fabricates a stereolithic model with the maxilla and mandible in their final position. This model is sent to the surgeon for TMJ fossa recontouring, condylectomy, and recontouring of the lateral ascending ramus as needed (Fig. 35.7). Classically, 15–18 mm of clearance between the glenoid fossa and the superior portion of the ascending ramus is required for adequate space of the TJP fossa and condyle

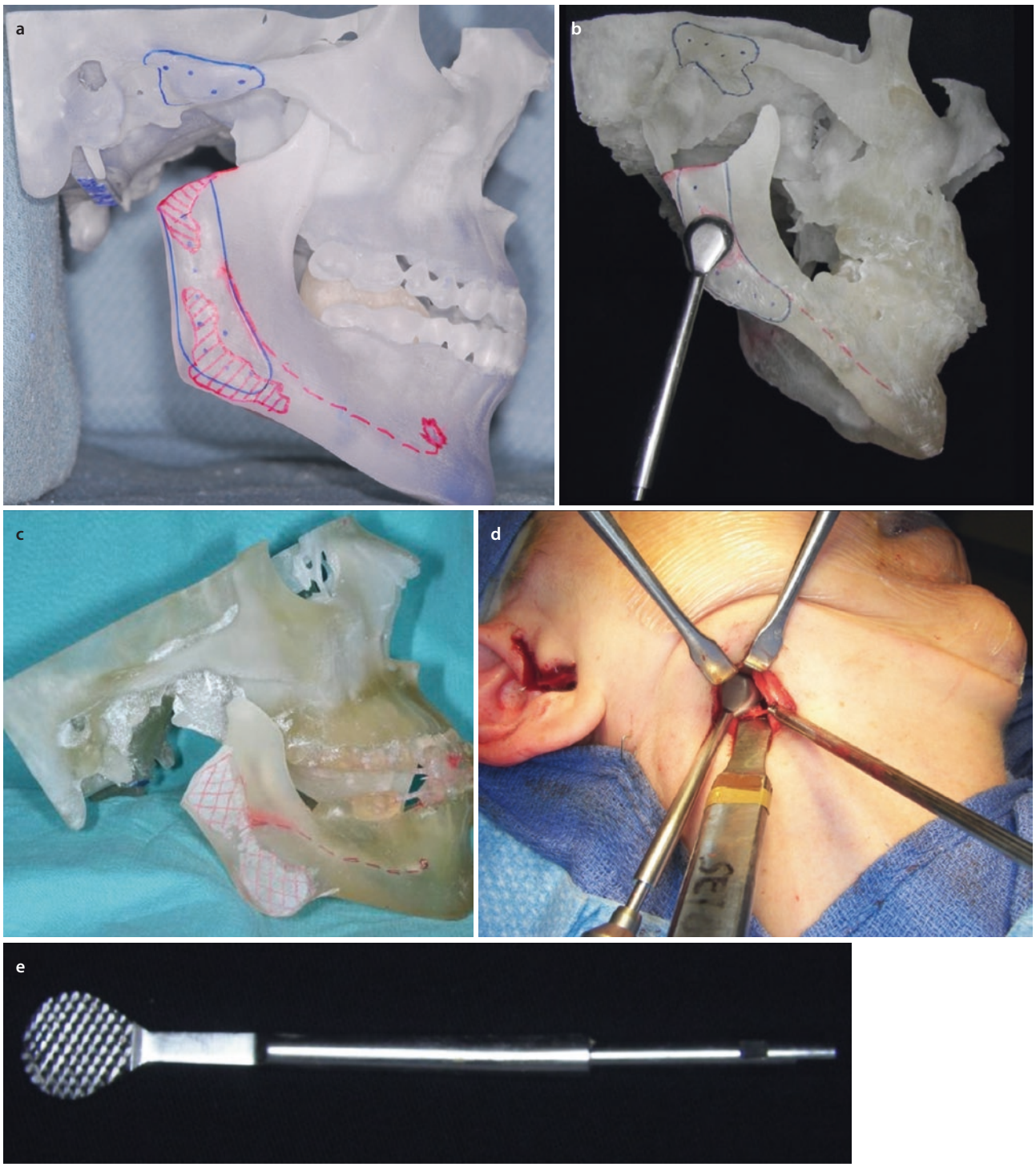


Fig. 35.7 Flattening of the lateral ramus as illustrated on a stereolithic model. The red marked areas on the lateral ramus require reduction for passive contact of the condylar component of the TMJ total joint prosthesis, as well as allow for anterior-posterior or superior-inferior positioning of the custom joint intraoperatively if required. This can be accomplished with a reciprocating rasp intraoperatively to recreate the flattened lateral ramus

components. However, patients with severe condylar resorption typically have exceedingly large mandibular advancements, so this is rarely a problem. All surgical modifications are completed on the stereolithic model and sent back to the TMJ prosthesis company for prosthesis design and fabrication. Images of the prosthesis wax-up and blueprints are sent to the surgeons for their approval before the appliances are fabricated. After approval, the TMJ prosthesis is manufactured and sent to the hospital.

The manufacturing process takes an average of 4 months in the USA. During prosthesis fabrication, the patient's occlusion may change, creating a splint that no longer fits the patient. In order to compensate for the possible occlusal changes that have occurred during the fabrication period, new impressions and dental models are again sent to the TMJ prosthesis company 2 weeks prior to surgery. The TMJ prosthesis medical modeling department incorporates the patient's current occlusion into the computer-simulated surgical plan for creation of both intermediate and final splints.

All materials (models, splints, printouts of surgical movements from the computers-simulated surgery) are sent to the surgeon (Table 35.1).

Table 35.1 Sequence of contemporary TMJ total joint prosthesis fabrication. Process takes approximately 4 months in the USA

Sequence of TMJ total joint prosthesis fabrication using computer-assisted surgical simulation	
1	CBCT or medical-grade CT of maxillomandibular complex to include mandible, maxilla, and TMJs
2	DICOM data used to create a computer-assisted surgical simulation
3	Modification of the computer model to correct the patient's dentofacial deformity while placing the maxilla and mandible into their final position
4	TMJ total joint prosthesis company creates a stereolithic model that is sent to the surgeon for condylectomy and lateral ramus recontouring
5	Modified stereolithic model then sent back to TMJ total joint prosthesis company for prosthesis wax-up and design
6	Design and blueprint of final prosthesis approved by surgeon
7	TMJ prosthesis manufactured by TMJ total joint prosthesis company. Prosthesis is sent to surgeon's hospital
8	Two weeks prior to surgery, two sets of dental models are acquired and sent to the TMJ total joint prosthesis company
9	Dental models are integrated with the stereolithic models for intermediate and final splint construction
10	Splints (intermediate and final), dental models, and all blueprints are sent to the surgeon for TMJ surgery

35.6 Surgical Techniques

35.6.1 General Perioperative Considerations

Patients with obstructive sleep apnea typically have an assortment of comorbidities that complicate their perioperative management. Comorbidities such as obesity, hypertension, coronary artery disease, pulmonary hypertension, and a propensity for desaturation due to minimal respiratory reserve predisposes this patient population to increased perioperative risks. Patients with significant oropharyngeal collapse due to TMJ degeneration should be treated in a surgical facility with the appropriate personnel and equipment required to manage their possible complications. The American Association of Anesthesiologist recommendations for perioperative management of OSA find the literature to be insufficient to require inpatient admission for all patients; however, patients with MMA with TMJ total joint prostheses require a minimum of an overnight observational period after surgery [67].

The anesthesiologist and surgeon rely on an open communication to elucidate any future complications to a safe anesthetic before they enter the operating room. Commonly, TMJ patients will have anatomical restrictions or deformities that create a difficult environment for nasal endotracheal intubation. Nasal endotracheal intubation typically requires awake fiberoptic intubation in patients with severe malocclusion and mandibular retrognathia. Early recognition of limitations in range of motion and early discussion with the institution's anesthesiology department are crucial in minimizing adverse events during intubation of these difficult airways.

Standard preoperative intravenous antibiotics that cover aerobic cocci, such as cefazolin, are administered between 1 hour and 15 minutes prior to incision. Antibiotics should be readministered according to each institutions' perioperative antibiotic guidelines [68–70].

Typically, cefazolin at 3–4 hour intervals is used. For patients with a penicillin allergy clindamycin can alternatively be used.

Patients without contraindication to corticosteroids may benefit from pre- and immediately postoperative dosing. Postoperative airway edema is a significant concern for the OSA patients as they have significant postoperative desaturations and upper airway edema [71]. Patients within 24–48 hours after traditional MMA have been found on fiberoptic nasopharyngolaryngos-

copy to have diffuse lateral pharyngeal wall edema, ecchymosis, and edema involving the pyriform sinus and aryepiglottic fold. A decrease in postoperative swelling may help to reduce length of stay and reintubation of this at-risk population [72].

Preoperative CPAP use has been suggested to improve surgical outcomes; however, the literature supporting this is insufficient. Patients undergoing general anesthesia with CPAP adherence prior to surgery have been suggested to have lower frequencies of severe postoperative complications compared to those without CPAP adherence [73]. No such studies have thoroughly investigated preoperative CPAP effects on postoperative complications in MMA or in MMA with TJP. However, postoperative CPAP should not be used for a minimum of 2 weeks due to the risk of subcutaneous emphysema.

Immediately preoperatively and immediately postoperatively, the external auditory canals and tympanic membranes must be inspected with an otoscope. Abnormal anatomy, membrane perforation or bulge, and signs of preoperative infection should be adequately documented. Active infection is a contraindication for surgery.

35.6.2 Mandible First Versus Maxilla First

When the maxilla must also be operated, there are two sequencing options for concomitant TMJ and maxillary orthognathic surgery: maxillary surgery first or mandibular surgery first. With maxillary surgery first, the maxilla is repositioned and stabilized first, then the TMJ surgery is performed. This option requires two sets of instruments to avoid contamination of the clean and sterile TMJ. The second option is performing the TMJ surgery first, followed by the maxillary osteotomy. This sequence allows one set of instruments.

If the TMJ replacement is performed first, one must take care during maxillary bone plate application because the prosthetic condyles could slip out of the prosthetic fossa. The final maxillary and mandibular position would be fixated in an inappropriate position if the surgery continues without recognition of the malposition condyle.

A major concern for many surgeons during concomitant TMJ and orthognathic surgery is condylar position and control. Some of the recommendations to manage the factors affecting condylar seating during concomitant surgery are as follows: (1) Achieve good hemostasis at surgery. (2) Place gentle but firm upward pressure at the angle of the mandible to seat the artificial condyle vertically into the fossa while applying maxillary fixation. (3) Do not create posterior open bites, but instead maximize intercuspation of the occlusion at the completion of surgery. (4) Perform careful and accurate surgery. (5) Attention must be paid to any potential interferences in the maxilla, especially in the area of the pterygoid plates because this can dislodge the condyles out of the fossa. (6) Side-to-side mobility of the artificial mandibular condyle is also of concern, and the planned position should be verified relatively to the piriform rims or other bony landmarks.

35.6.3 Single Stage Versus Two Stage

Some temporomandibular joint pathology can create the concern for continued disease after total joint reconstruction. Patients may benefit from a two-staged TMJ reconstruction in cases with TMJ ankylosis, previously operated TMJs with a failed implant system and foreign body giant-cell reaction, septic TMJ arthritis, or in tumors of the TMJ where there is concern for seeding of disease [74]. In such cases, an arthroplasty to remove the ankylosis can be performed first with an acrylic spacer to maintain space for the future TJP. Then a second surgery is performed to place the prostheses and complete any maxillary surgery required [75, 76].

Without specific TMJ pathology, there has been little need for staged reconstruction as TMJ surgery with concomitant orthognathic surgery has been well established as stable in recent literature. We recommend one-stage MMA where the maxillary osteotomy is done immediately after the TJPs have been placed except in the cases where TMJ pathology mandates delayed reconstruction.

35.6.4 Patient Preparation and Drape

Nasal endotracheal intubation is performed, and the tube is sutured to the nasal septum above the columella with 2-0 silk suture. The patient is placed in a supine position with the neck in a slightly extended position to allow access for the submandibular approach.

After intubation is complete, the sideburn region is trimmed of any hair. Long hair in the lower temporal region or around the insertion of the helix is pulled back with silk tape to isolate it from the operative field. The head is then wrapped, eyes are protected against corneal abrasion, and a throat pack is inserted.

Arch bars must be placed prior to prepping the patient with betadine if no orthodontic appliances are present. The choice between arch bars and IMF screws depends on the number of teeth present and the movement to be done. The patient is not wired into intermaxillary fixation until after the condylectomy because the surgeon needs to be able to manipulate the mandible

during surgery, especially in case bleeding occurs. Finally, the patient is prepped intraorally and extraorally and a large Tegaderm barrier is placed over the mouth and nares.

35.6.5 Endaural Incision

Exposure of the temporomandibular joint, disc, and ascending ramus is achieved through an endaural approach (Fig. 35.8a). The superficial temporal vessels and the auriculotemporal nerve lie just anterior to or within your incision line and may be retracted anteriorly within the flap [77]. The tragus is grasped with forceps and posterior tension is applied so that tenotomy scissors can dissect the anterior tragal cartilage to the full depth of the tragal cartilage (Fig. 35.8b). Supraperichondral dissection can be assured with a white color of the underlying cartilaginous tissue while dissection anteriorly into soft tissue will have a slight pink hue.

The anterior flap, which now contains the temporal vessels, auriculotemporal nerve, and the zygomatic branch of the facial nerve, is retracted 1.5–2 cm anteriorly [78]. Upon entering through the superficial temporal fat pad through the superficial temporalis fascia, the Dean scissors can be opened completely, spreading the fat and allowing access to the periosteum of the arch (Fig. 35.8c). Continued dissection 1 cm inferior to the level of the arch will expose the underlying joint capsule. The capsule is then injected with 1–2 cc of a lidocaine with epinephrine solution. Appropriate placement is confirmed by visualization of the chin moving forward upon injection.

35.6.6 Discectomy

The mandible is distracted anterioroinferiorly to increase the joint space and facilitate entrance into the capsule. The joint capsule is incised with a 15 blade at a medial-superior angle to prevent damage to the disc or the cartilage of the glenoid fossa. The incision is curvilinear on the lateral rim of the glenoid fossa. The capsule and any remaining periosteum are incised with Dean scissors anteriorly, around the articular eminence. The inferior joint space is entered by making a vertical mid-incision release extending 1–2 cm onto the lateral portion of the condyle between the lateral pole and disc. The disc is released and the bilaminar tissue is incised with Dean scissors at the junction of the posterior band. Now the lateral pterygoid muscle is its only remaining attachment. At this point, the surgeon can decide to remove the disc prior to the condylectomy or leave it attached and remove it while performing the condylectomy.

35.6.7 Condylectomy

The dissection of the condyle begins at the T-shaped incision in the superior lateral region of the condyle. The condyle is dissected circumferentially to 1 cm beyond the planned site of the condylectomy to include the coronoid notch and coronoid. Maintaining a subperiosteal plane of dissection is paramount to prevent bleeding from the internal maxillary artery or one of its multiple branches. At the level of condylar osteotomy, condylar neck retractors are placed at a 90-degree angle, and with the planned osteotomy site exposed a reciprocating saw or piezolelectric handpiece is used to complete the osteotomy (Fig. 35.8d–f). Once the osteotomy is complete, the condylar neck is grasped and retracted laterally to allow visualization of the attachment of the lateral pterygoid. The lateral pterygoid is incised at the insertion point with a Bovie cautery. If the disk was not removed prior to the condylectomy, the remaining dissection of the disc is performed, and it is removed with the condyle. Once the condyle and disc have been removed, all remaining soft tissue is thoroughly debrided. The fossa component of the TJP must be in intimate contact with remaining glenoid fossa, and any residual tissue predisposes the prosthesis to possible malunion and the formation of heterotopic bone.

35.6.8 Coronoidotomy

Attachment of the coronoid to the temporalis muscle restricts the advancement of the mandibular complex and reduces stability of MMA. After both condyles have been removed, the mandible can be significantly displaced posteriorly and superiorly so that the coronoids can be dissected circumferentially and removed. The coronoidotomy is performed with a reciprocating saw at the level illustrated on the stereolithic mode. The temporalis muscle is left attached to the coronoid and retracts it superiorly once the osteotomy is performed (Fig. 35.8g). At this time, the surgeon can decide to remove the coronoid process, but this is only necessary in cases when there is a risk of ankylosis.

35.6.9 Submandibular Incision

The submandibular incision allows access to the inferior-lateral portion of the ascending ramus for placement of the condylar component of the TJP. If local anesthetics with vasoconstrictors are to be used, they must be deposited superficial to the platysma to prevent false-negative nerve conduction of the marginal mandibular nerve. The 1.5- to 2-cm-long incision is placed 2 cm infe-

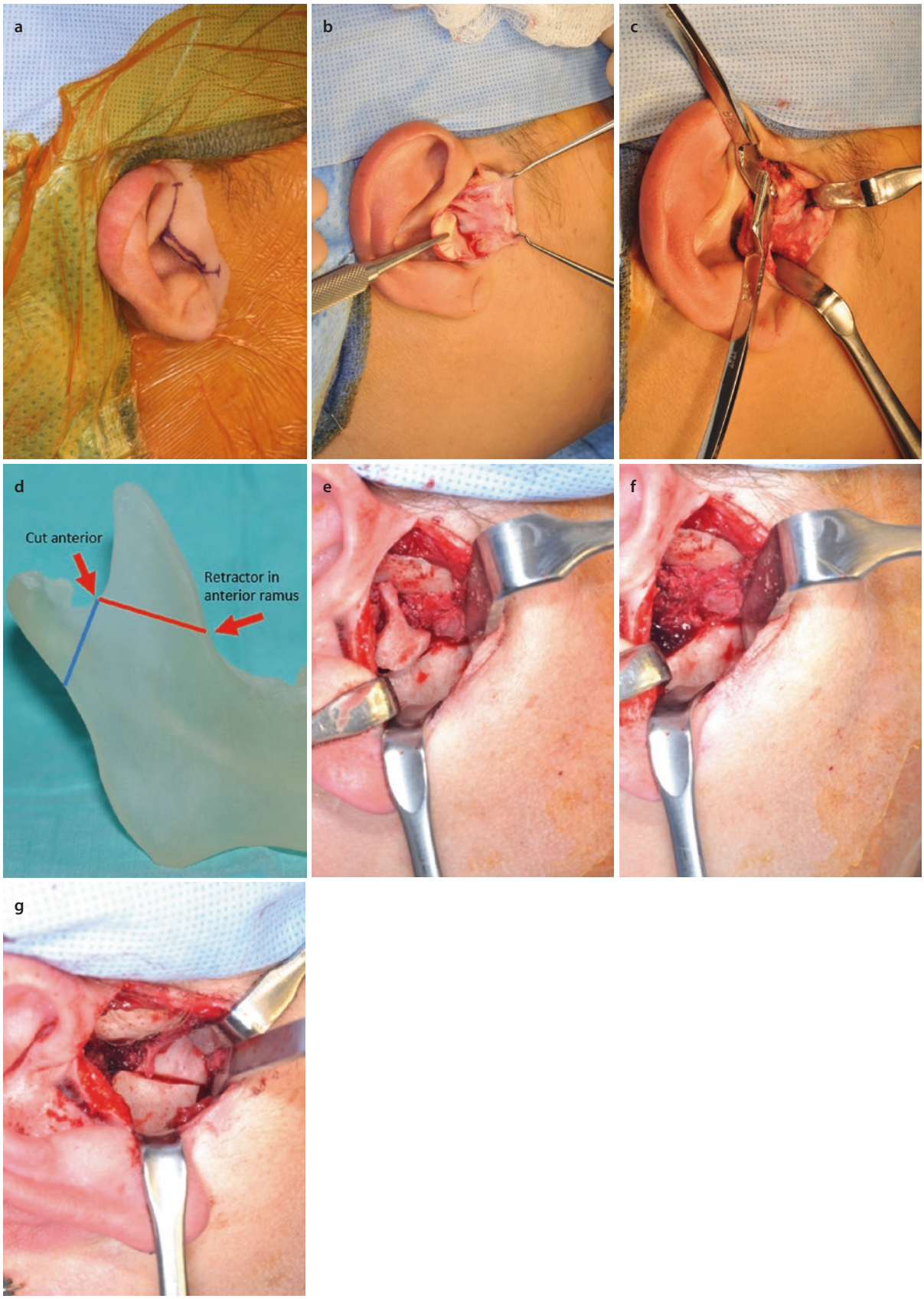


Fig. 35.8 **a** Endaural incision site marked. **b** Dissection anterior to the tragus to the tragal root. **c** Entering the superficial temporal space prior to dissection into the superior joint space. **d** Condylectomy and coronoidotomy illustrated on a stereolithic model. **e**, **f** Condylectomy. **g** Coronoidotomy exposure and osteotomy after posterior pressure applied to the mandible

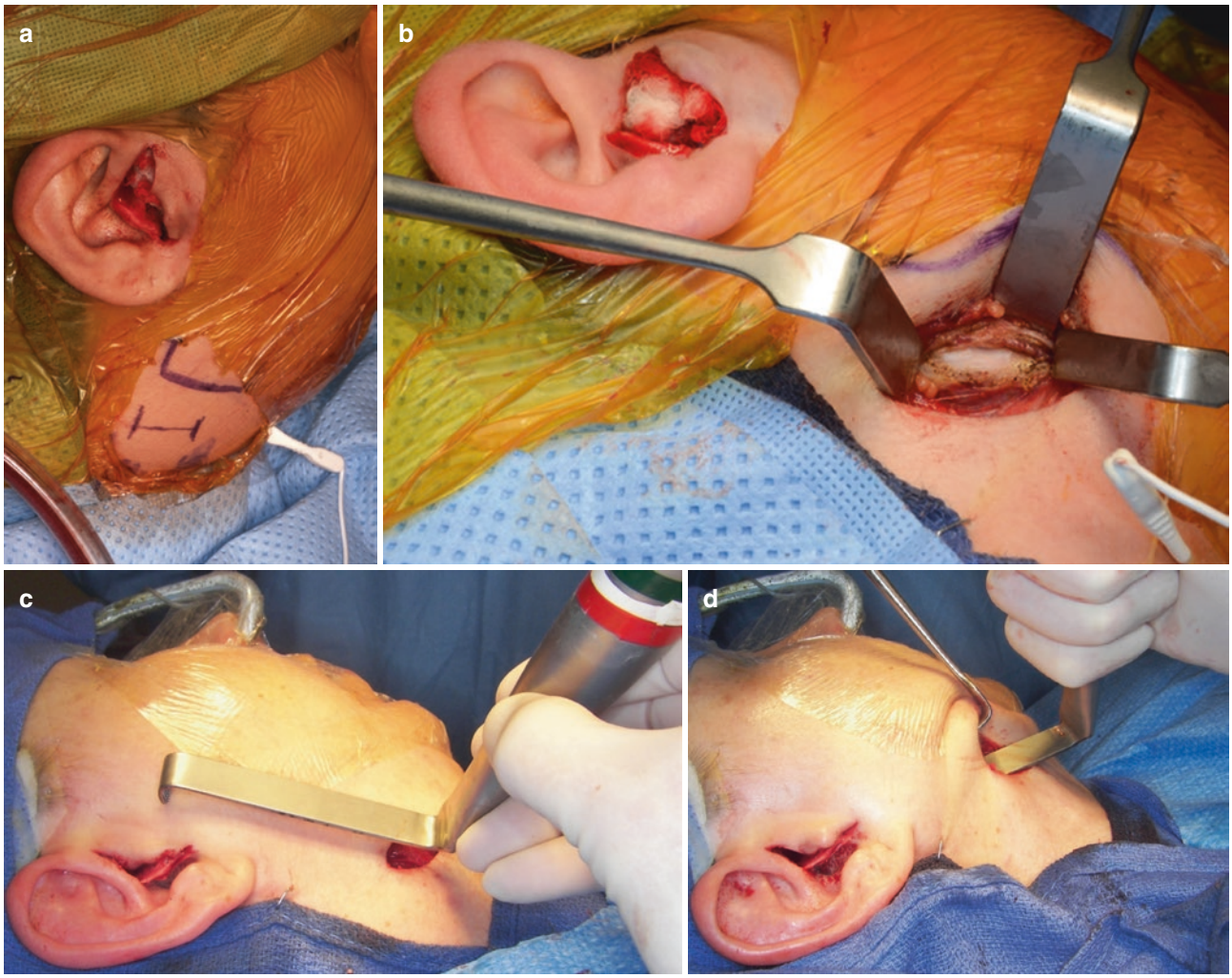


Fig. 35.9 **a** Submandibular incision site marked. **b** Transection of the pterygomasseteric sling with exposure of the inferior border of the mandible. **c**, **d** Anterior-inferior mandible mobilization prior to implant placement

rior to the inferior border of the mandibular to avoid injury to the marginal mandibular branch of the facial nerve (Fig. 35.9a) [79, 80]. Careful dissection through the platysma with scissors and monopolar electrocautery is carried out with the help of nerve monitoring. In exposing the pterygomasseteric sling, the facial vessels may be sacrificed if they are unable to be retracted. Once the pterygomasseteric sling is incised along inferior border of the mandible, the entire lateral ramus and posterior mandibular border is dissected to join with the superior dissection (Fig. 35.9b). After thorough debridement of periosteum, a large reciprocating rasp can be used to recontour the lateral ramus according to the stereolithic model (Fig. 35.7). If the mandible will be relocated (advanced, counterclockwise rotation, etc.) in a fashion that increases the forces on the pterygomasseteric sling, then the medial pterygoid is dissected from the medial aspect of the mandible. When performing this dissection, it is important to maintain a subperiosteal plane and to not lacerate or traumatize the IAN as it enters into the lingula. After dissection is complete bilaterally, the mandible is mobilized to stretch the sphenomandibular and stylomandibular ligaments (Fig. 35.9c, d). In large CCW rotations, the IAN may suffer, and the possibility of permanent paresthesia needs to be discussed with the patient (Table 35.2).

35.6.10 Intermediate Intermaxillary Fixation

It is imperative that the surgical field is isolated from oral contents while placing the patient into intermaxillary fixation. Towels and new drapes can be used to this end. The back table must not be touched during this non-sterile portion of the procedure, or a new set of instruments must be opened. The Tegaderm placed over the mouth is incised to gain entry to the oral cavity. The

Table 35.2 General considerations for troubleshooting a condylar component malalignment during placement and fixation

Considerations for different unsatisfactory condyle/ramus positions		
Ramus component	**Condyle**	**Alteration required**
Flush	Lateral	1. Reduction of the superior lateral ramus 2. Grafting of inferior lateral ramus
Flush	Medial	1. Reduction of inferior lateral ramus 2. Grafting of superior lateral ramus
Inferior	Unseated	1. Condylectomy osteotomy inferior to predicted osteotomy

patient is placed into intermaxillary fixation with the intermediate splint provided by the TJP manufacturer. It is paramount that the intermaxillary fixation does not loosen during the placement of the TMJ prostheses. Once finished, a Tegaderm is placed over the oral cavity, and the field is again deemed sterile.

35

35.6.11 Component Fixation

The titanium portion of the glenoid fossa prosthesis is grasped with a hemostat and placed into the patient's glenoid fossa. A fossa seating tool is then placed against the inferior aspect of the glenoid fossa to place the fossa component into its correct position (Fig. 35.10a, b). The position is correct when the fit is tight, there is no rocking, and the prosthesis sits clinically as it does anatomically on the stereolithic model. The distance between the anterior tympanic plate and the posterior wall of the prosthesis is a reasonable point of reference. The component is fixated with usually four screws of predetermined length as per the customized screw length prescription by the TMJ TJP manufacturer.

The mandibular component is placed through the Risdon incision and is inserted so that the condyle can be visualized in the prosthetic fossa. The condyle is placed at the most posterior-superior aspect of the prosthetic fossa and centered mediolaterally (Fig. 35.10c–e). Once satisfied with the location and verified by stereolithic model, a gauze is packed anteriorly to maintain the appropriate prosthetic condyle to fossa contact during fixation. The ramus component should lie flush against the lateral ramus and have the same distance from the posterior and inferior borders as depicted on the model. If this is not the case, then further reduction of bone of the lateral ramus is performed.

Once the mandibular component is satisfactorily positioned, it is stabilized with custom length screws per the prescription provided by the TMJ TJP manufacturer. The first screw placed is the most superior screw possible and the hole should be drilled superiorly in the hole to maintain the vertical position of the mandibular component. After this, the surgeon places anterosuperior pressure in the most inferior hole to sit the prosthesis posteriorly. A screw next to this hole is drilled anteriorly to maintain the posterior position of the head of the prosthesis (Fig. 35.11). We recommend evaluating positioning after placing the first two screws; if satisfied, place the remaining screws. Keep in mind that the superior most fixation site may not have sufficient underlying bone if condylectomy was made inferior to the site on the model. The lack of fixation in one screw hole is not problematic as the ramus component is retained by 7–9 screws [81].

35.6.12 Abdominal Fat Harvest and Placement

Fat has been shown to reduce the risk of heterotopic bone formation. If fat is to be harvest, the abdomen is usually the easiest place to obtain it. The abdomen should be draped from umbilicus to suprapubic region at the time of TMJ skin preparation. A 4- to 5-cm-long periumbilical or suprapubic incision can be used to gain access to the subcutaneous fat of the abdomen. The autogenous fat graft loses both graft volume and viability if harvested >4 hours prior to placement, thus harvest should be directly after the TJP components are fixated [82]. Two surgical teams can work in concert to harvest the fat while fixating the TJP [83, 84].

Approximately 30–40 cc of abdominal fat is harvested from the midline abdominal fat pad in this fashion. After en bloc removal of the fat graft, attention is turned to adequate hemostasis with the monopolar electrocautery. Fifteen to twenty cc of harvested adipose tissue is immediately packed around each prosthetic condyle and fossa (Fig. 35.12).

If there is concern for continued accumulation of hematogenous or serous fluid, a Jackson-Pratt drain is placed to bulb suction. The abdominal incision is closed in layers using 2-0 polydioxanone (PDS) or Vicryl to close the subcutaneous layer to obliterate any dead space. The skin incision is closed with 5-0 Prolene in a subcuticular fashion. A Tegaderm dressing or Steri-Strips are placed over the incision. An abdominal pressure dressing is placed at the conclusion of the procedure and maintained for 3 days after surgery to minimize risk of hematoma or seroma formation.

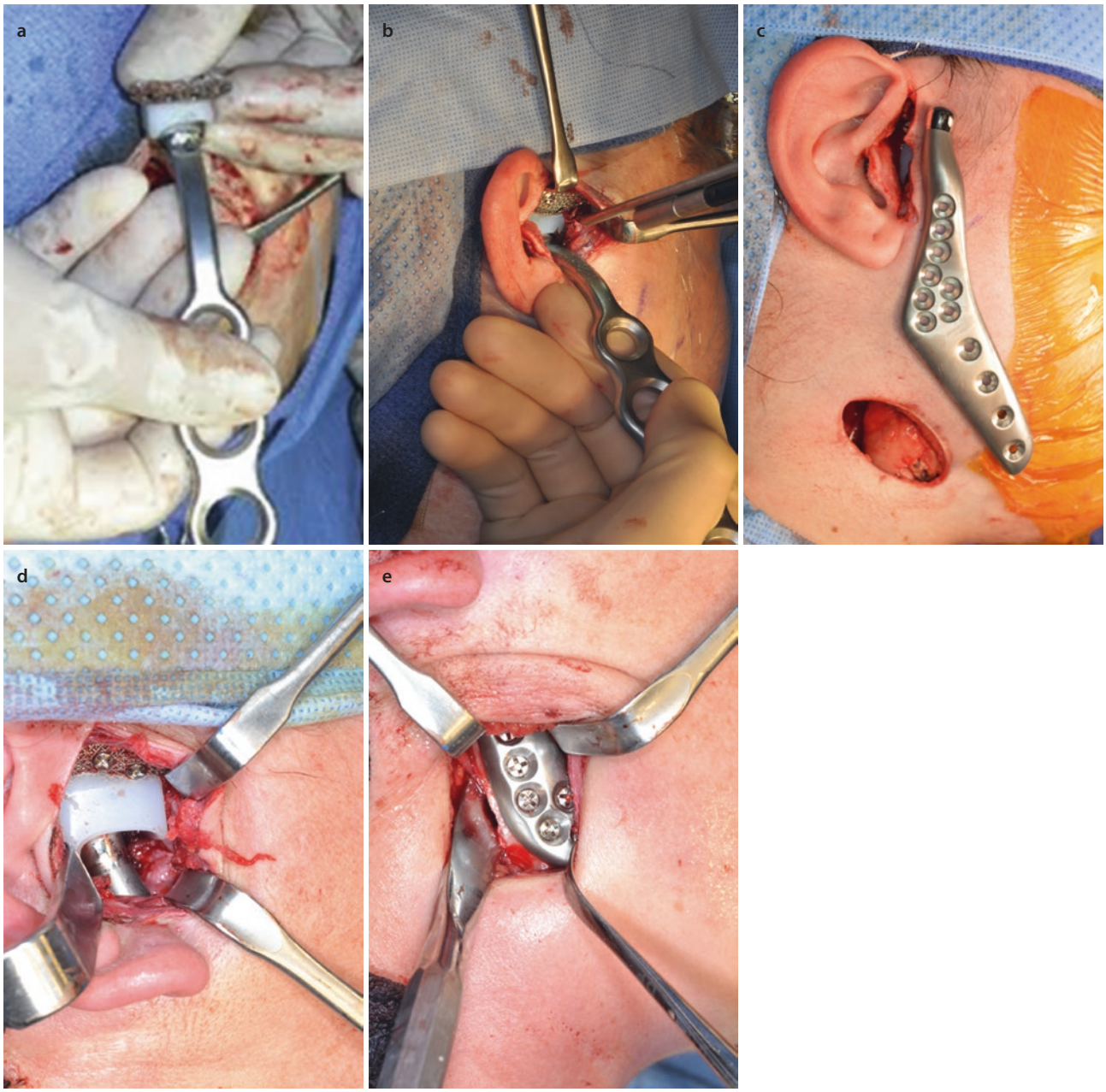


Fig. 35.10 **a**, **b** Insertion of the fossa component of the TMJ prosthesis with fossa seating tool. The ultrahigh-molecular-weight polyethylene of the fossa component can be damaged with other surgical instrumentation. **c**–**e** Insertion of the mandibular component of the TMJ prosthesis

35.6.13 Skin Closure

Two holes are drilled at the inferior border of the mandible, and a 3-0 Vicryl or PDS is then passed through the masseter, the drill holes, and finally the medial pterygoid to re-suture the pterygomasseteric sling (Fig. 35.13). The submandibular incision is then closed in layers.

The endaural incision is closed in layers with a superficial running subcuticular prolene suture that is removed in 5–7 days. Steri-Strips are placed over the incision sites. At the conclusion of the procedure, Kerlix fluffs are placed over the ears and mandible, and a compressive dressing is fitted to the patient.

35.6.14 Le Fort I Osteotomy

At the conclusion of TJP skin closure, the oral field may be opened for the Le Fort I osteotomy. The maxillary osteotomy can then proceed without concern for con-

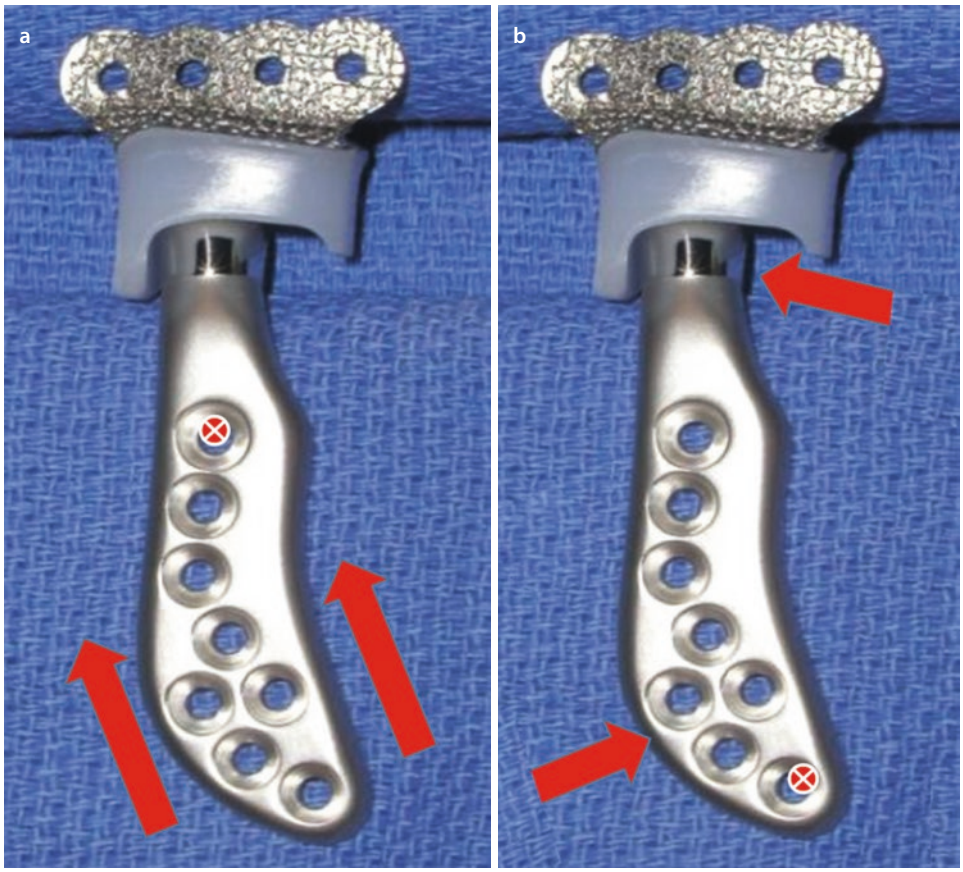


Fig. 35.11 **a** Placement of the first screw into the TMJ mandibular component and the trajectory of force on the mandibular component prior to placement. **b** Placement of the second screw into the TMJ mandibular component and the trajectory of force on the mandibular component prior to placement

tamination of the TJP. The Le Fort I osteotomy begins with removal of the intermediate intermaxillary fixation and intermediate splint. In order to correctly position the maxilla in the preplanned vertical position, a stable point of reference is required for measurement. A Kirschner-wire (K-wire) can be placed at nasion for a stable and reliable vertical point of reference. After placement, the K-wire is trimmed to 1 cm from the skin, and a measurement from teeth #8 and #9 is recorded [85–87].

A standard Le Fort I osteotomy is completed bilaterally using a reciprocating saw with care not to damage the maxillary root tips (typically 4 mm apical to the suspected root tips is sufficient). A double safe-sided osteotome is then used to remove the septum from the nasal crest of the maxilla. Using a single safe-sided osteotome, the lateral nasal walls are fractured to the perpendicular plate of the palatine bone. Next, a curved osteotome is used bilaterally to fracture the pterygoid plates. A curved Freer is placed behind the piriform rims bilaterally, and maxillary downfracture is completed. The descending palatine vessels are visualized and sacrificed via electrocauterization if necessary (Fig. 35.14).

Care must be taken to not dislocate the new prosthetic joint during the down fracture. The entire maxillomandibular complex must be carefully rotated using the artificial TMJs as a hinge with constant pressure to verify the artificial condyles are always seated. Once the maxilla is fully mobilized, it is placed into final occlusion with a final occlusal splint and fixated in place.

An alar cinch suture is placed using 2-0 Vicryl, and a running V-Y closure is used to close the midline incision with 3-0 chromic gut suture [88–90]. The Kirschner wire is then removed from the nasal bridge.

35.6.15 Elastics

TMJ motion is limited in the immediate postoperative period. The dissection of the pterygomasseteric sling on both the medial and lateral aspects of the ramus, as well as the coronoidectomy, required to achieve adequate advancement of the mandible contributes greatly to the possibility of TJP dislocation and occlusal disharmony. Risk of dislocation is greatest in the first week after surgery. However, with the appropriate use of elastics postoperatively, TJP dislocation rates drop to less than 1% [91].

In the first 24 hours to 7 days, heavier elastics are recommended to promote healing of the TJP in the appropriate position. Intraoral elastics with a minimum tensile strength of 6 oz or 170 g are used for this purpose. The

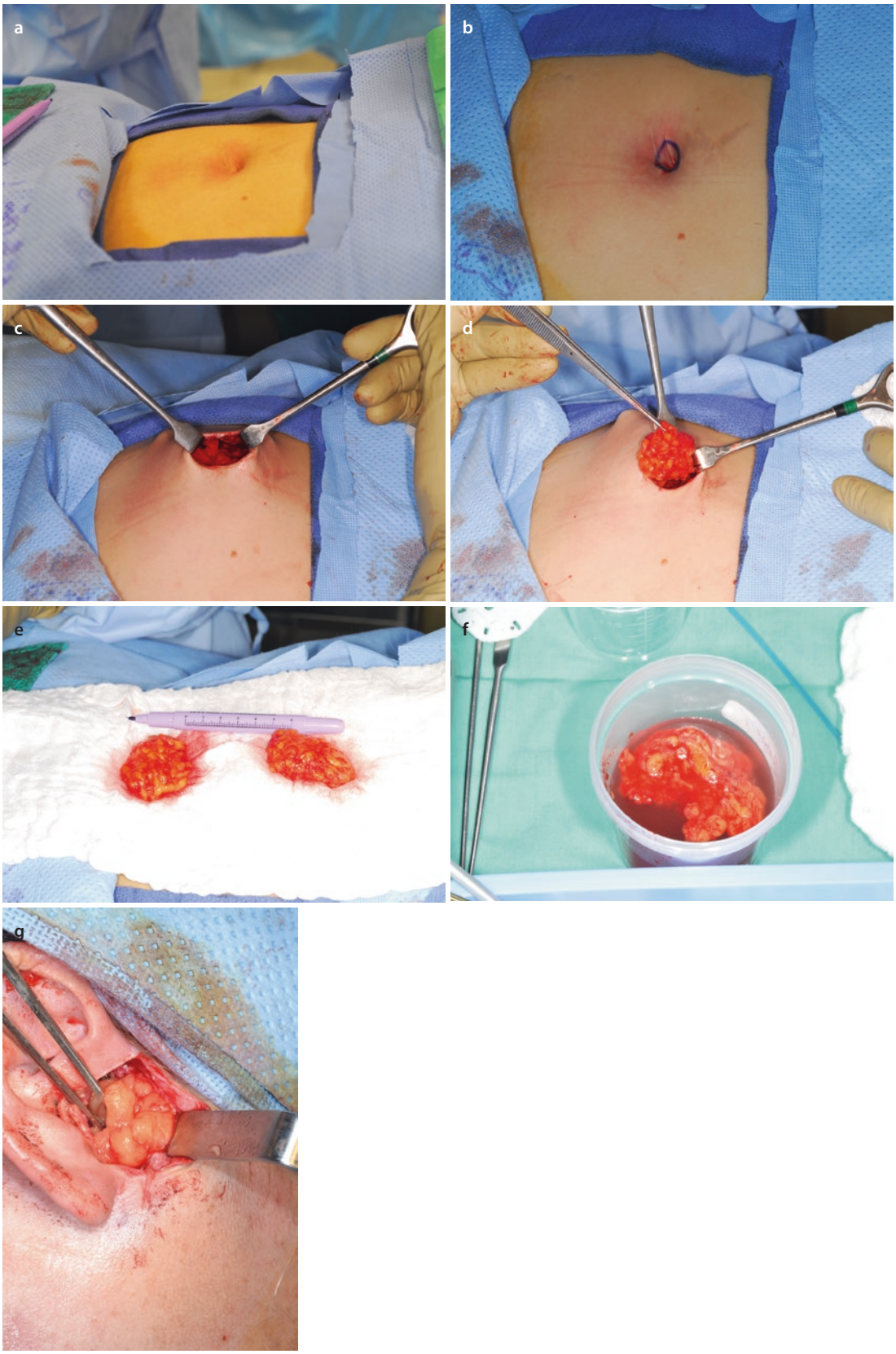


Fig. 35.12 Autogenous fat graft harvest technique. **a** Drape for abdominal fat graft. **b** Periumbilical incision marking. **c** Retraction for fat graft harvest during undermining and procurement. **d** En bloc fat graft removal. **e** Amount of fat graft required for adequate coverage of TMJ total joint prosthesis. **f** If site preparation is not complete, fat graft may be placed in sterile saline. **g** Placement of autogenous abdominal fat graft

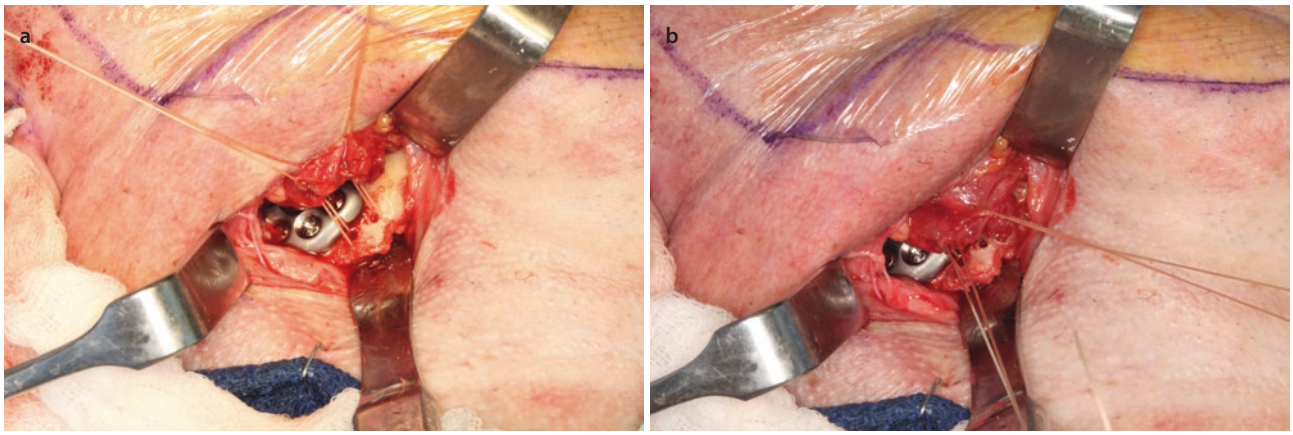


Fig. 35.13 Anchoring of the pterygomasseteric sling

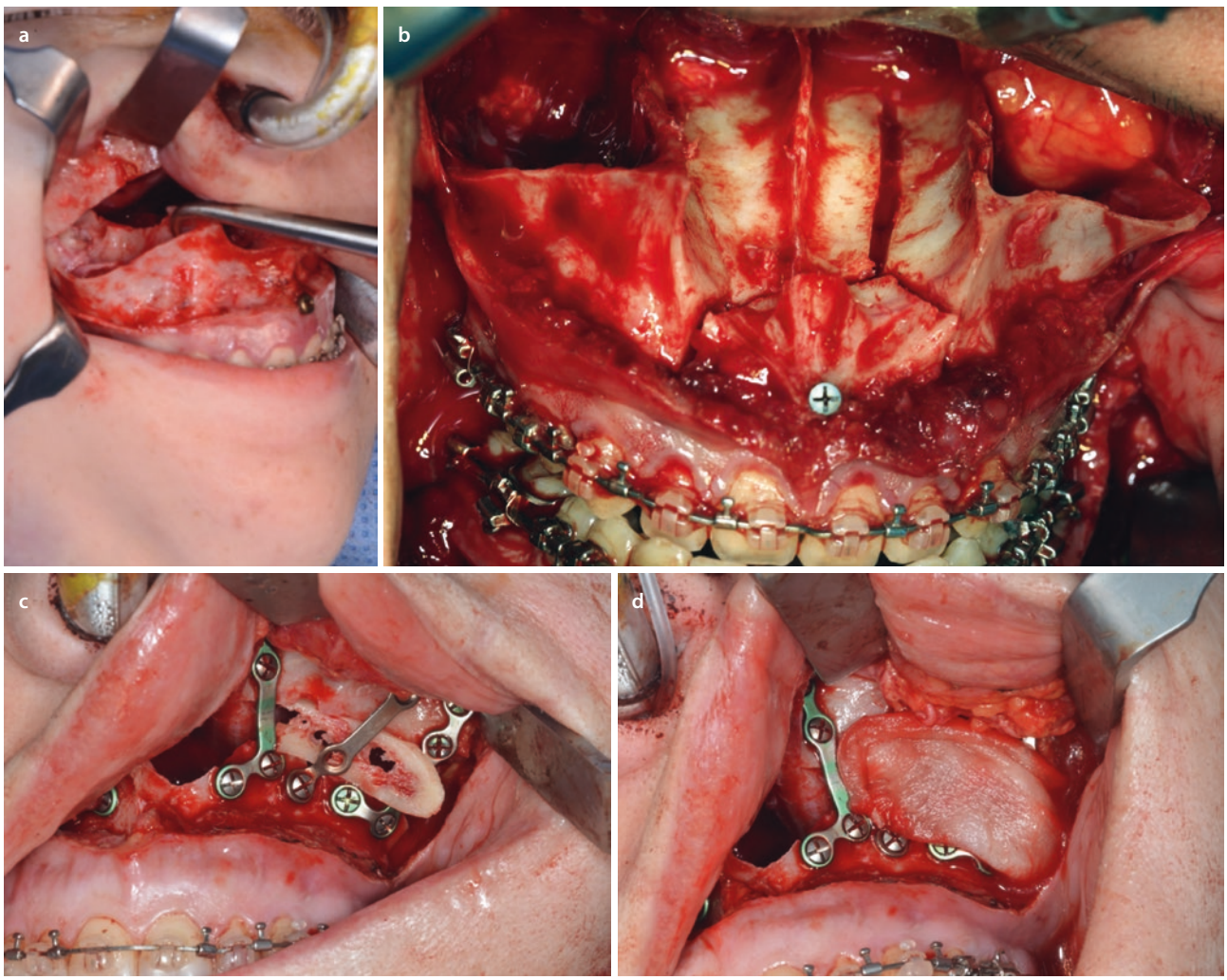


Fig. 35.14 **a** Le Fort I osteotomy during down fracture. **b** Le Fort I after segmental osteotomy. **c**, **d** Fixation of segmental Le Fort I osteotomy with bone graft material

elastic configuration is in a neutral position to maintain centric occlusion. Placement of elastics for correction of minor malocclusions must be monitored closely in order to assess the desired dentoalveolar change in occlusion. After the first week of heavy intraoral elastics, patients may use elastics as would be done in a classical orthognathic case to guide postoperative occlusion into centric relation. Elastics may be discontinued after simple malocclusions are no longer apparent.

35.6.16 Dressing

Compression dressing remains in place for a minimum of 24 hours in order to reduce postoperative swelling.

35.7 Postoperative Patient Management

35.7.1 Pain Management

Multimodal pain management has become the standard of care in patients following MMA with TMJ total joint prostheses. Many factors contribute to the complexity of pain management in this patient population. First, patients requiring MMA for treatment of obstructive sleep apnea have a baseline higher analgesic requirement when compared to patients requiring orthognathic surgery for dentofacial deformities [92]. To compound issues further, patients with TMJ disease have a baseline higher level of pain and pain reporting than the general public [93]. The etiology of the patient's TMJ pain, their preoperative pain level and tolerance, their presurgical analgesic requirements, and the number of previous TMJ operations all contribute to the patient's ability to achieve successful postoperative analgesia [94].

Postoperative management in the immediate and early stages after surgery should specifically target opiate usage in an effort to decrease postoperative oxygen desaturation and other severe comorbidities [71]. In order to reduce postoperative opiate requirements, medications such as liposomal bupivacaine, nonsteroidal anti-inflammatory drugs (NSAIDS), acetaminophen, muscle relaxants, anti-histamine, and CNS neurotransmitter modulators (i.e., Gabapentin, ketamine, etc.) are used in conjunction with smaller doses of common opiate medications [95, 96]. Analgesic regiments should take into consideration the medications that the patient used for pain management preoperatively. It is common that one or more non-opiate medications have provided adequate pain relief for preoperative pain. It is effectuations to listen to the patient's past experiences with non-opiate pain regiments to find the appropriate balance in the postoperative setting.

Local infiltration of liposomal bupivacaine has become a topic of increased research due to its promise in providing long-term (24–72 hours) analgesia with similar side effect profiles to other available local anesthetics [97, 98]. This new formulation provides a longer duration of action with slow release of bupivacaine into local and systemic circulation [99]. The use of regional liposomal bupivacaine has been reported in other arthroplasty surgical procedures with decreased opiate requirements, increased time to first opiate rescue, and improvement in postoperative pain [100]. Adequately powered, multicenter research on the efficacy of liposomal bupivacaine will continue to provide evidence for the use in the TMJ region; however, there is lack of data in the literature today. The authors have used liposomal bupivacaine to reduce both postoperative pain and opiate requirements to good effect.

Patients should be educated preoperatively that postoperative pain after MMA with TMJ TJP can last 1 month or longer depending on preoperative pain levels. In the first days after surgery, rapid transition from opiate patient-controlled anesthesia (PCA) to a multimodal oral pain regiment is advised. A combination of ibuprofen, acetaminophen, gabapentin, and tramadol in conjunction with long-acting local infiltration has provided significant relief to the majority of the author's patient population. In the end, patient satisfaction with analgesia will come down to patient expectations, and tempering patient expectations of a painless surgery is paramount to excellent analgesia.

35.7.2 Antibiotics

MMA with TMJ TJP has two wound classifications: clean and clean-contaminated. As previously stated, sterility of the instrumentation is of upmost importance when operating on the prosthesis surgical field due to possible contamination from the oral cavity. Surgical site infection (SSI) can be a devastating complication of TMJ total joint reconstruction.

A standard preoperative intravenous antibiotic dosage that covers aerobic cocci is administered between 1 hour and 15 minutes prior to incision [101]. Antibiotic regiments include 1 gram cefazolin for patients less than 80 kg and 2 grams for patients greater than 80 kg. Clindamycin 600 mg is an acceptable preoperative alternative for those allergic to penicillin. Perioperative antibiotics should be administered according to institutional guidelines based on the half-life of the antibiotic admin-

istered. Antibiotic duration of at least 5 days postoperatively has been reported to lower SSIs to as low as <1% in classic orthognathic surgery [68, 69].

In a questionnaire study by Mercuri et al., 26 TMJ surgeons from across the world were asked about duration of antibiotic treatment after TMJ total joint reconstruction. The mean postoperative antibiotic duration was 7 days (range 5–14). Forty-six percent (12/26) reported that they soaked their TJP in either an antibiotic solution or antiseptic solution during surgery. Also, 61.5% (16/26) reported that they irrigated their implant site after fixation with either an antibiotic or antiseptic solution. Reported SSI rates at a mean of 6 months are comparable to classical orthognathic surgery with 51 joints (1.51%) infection of the TMJ [102, 103].

The rate of surgical site infections of TMJ total joint reconstruction within 1 year from surgery ranges from 1.5% to 4.5% [104]. Five days of postoperative antibiotics has been reported as adequate for orthognathic surgery; however, the vast majority of TMJ surgeons use 7 days of postoperative oral antibiotics (cephalexin or clindamycin). The authors are among the majority of surgeons that use 7 days oral antibiotics after surgery.

35.7.3 Swelling

In the immediate postoperative period, edema can be quite significant after patients undergo MMA with TMJ TJP. Swelling will usually reach a maximum at 3–5 days after surgery. The extent of postoperative edema can be impressive and concerning to patients immediately after surgery; however, swelling rapidly dissipates after the first month of surgery. There may be residual edema for up to 12 months after surgery but most patients will not have a noticeable reduction in swelling after 6 months [105].

Interventions to decrease postoperative edema include head of bed elevation (HOB) to 30 degrees and perioperative corticosteroids. HOB elevation is maintained immediately after surgery and for up to 2 weeks. Multiple studies have reported on the efficacy of corticosteroid administration in reducing postoperative edema [106–109]. There is significant variability in the evaluation of pre- and postoperative facial edema, as well as significant variability in dosing regiments. Most regiments adhere to one dose of corticosteroid (e.g., dexamethasone, betamethasone, methylprednisolone) pre- or intraoperatively and two to three doses of postoperative corticosteroids. A typical and effective regiment for corticosteroids is 8 mg dexamethasone at induction of anesthesia and 8 mg every 8 hours for the first 24 hours after surgery.

35.7.4 Diet and Dysfunction

Patients with severe retrognathia and joint degeneration have decreased jaw function, diet restrictions, and limited range of motion prior to TMJ total joint replacement [110]. Immediately after surgery, patients will have significant swelling and a sore throat causing difficulty eating, which often predisposes patients to malnourishment and dehydration. The onset of rigid fixation has diminished the need for intermaxillary fixation postoperatively; therefore, patients can function and tolerate a diet immediately after surgery. Patients can start with a clear liquid diet and advance to a full liquid diet as early as the day of surgery. Patients may then progress to a dental soft diet (i.e., pureed/crushed foods) with nutritional supplementation. A soft diet should be implemented for the next 4 months, particularly in patients that required a maxillary osteotomy.

Patients often have significant improvement to jaw function and food quality after MMA with TJP. Patients will report resolution of their restricted diet with chewing, and mandibular function significantly improved in 6–8 weeks from TMJ replacement. On a visual analog scale from 0 to 10 (0 = no diet restriction, 10 = liquid diet), Pinto et al. reported a significant reduction in dietary restrictions at the patient's longest follow-up: 5.6–3.4 [54]. This decrease in dietary restriction has been reported across the literature with increased ability to tolerate a robust diet, increase in jaw function, and decrease in subjective impairment [111, 112].

35.7.5 Activity

Early ambulation and out of bed to chair is recommended after surgery for the immediate postoperative period. Patients often ask when they are able to return to exercise or sports activities. In patients with MMA, the Le Fort I osteotomy site has the most concern for malunion or maxillary displacement after a traumatic blow to the face. It is also the site of significant postoperative bleeding with an increase in blood pressure or heart rate. Patients are recommended to avoid activities that elevator their heart rate or make them sweat for at least 4 weeks to ensure a reduced risk of postoperative bleeding from the maxilla. Patients are recommended to avoid contact sports for at least 4 months due to the risk of displacement of the maxillomandibular complex, hardware failure (typically maxillary hardware), and malunion.

35.7.6 Physical Therapy

Internal fixation has allowed for immediate functioning and physical therapy (PT) after orthognathic surgery, and MMA with TJP prosthesis is no different. Physical therapy after traditional TMJ surgeries has been shown to aide in return of jaw function, increase range of motion, and decrease pain after surgery [113–115]. TMJ physical therapy should be initiated as early as the day after surgery. Patients that require a maxillary osteotomy can participate in physical therapy exercise but should delay use of adjunctive PT devices until the maxilla has fully healed, usually 6–8 weeks. Elastics may be removed in order to participate in physical therapy 7–10 times a day.

Goals of physical therapy are to increase MIO up to 20 mm at 4 weeks after surgery, 30 mm at 6–8 weeks after surgery, and 35–40 mm at 12 weeks. Adjunct physical therapy devices such as the TheraBite can be used to assist in increasing MIO through stretch exercise. Patients simply use the device rather than using traditional techniques. Outcomes between traditional physical therapy and stretch devices have not shown to be more or less effective in increasing mandibular range of motion.

35.8 Complications

35.8.1 Nerve Damage

Permanent or transient damage to cranial nerve V and VII are possible during dissection; however, with careful management of soft tissues and adherence to the techniques listed above, permanent damage is uncommon. Depending on the etiology of the damage (i.e., complete transection, stretch, thermal, etc), there is variable prognosis. Neuropraxia caused by stretching the nerve during liberal retraction is by far the most common nerve injury seen. The majority of these nerve injuries resolve over the course of the first few weeks, but may take up to 12 months to fully recover.

Patients with persistent weakness of the facial nerve should have frequent follow-up to assess for improved function. If there is persistent weakness or lagophthalmos, then a brow lift or gold weight for upper eyelid loading, respectively, may be required. Cranial nerve V damage can be caused either during the placement of the TJP or during fixation with a misplaced screw. During large counterclockwise rotations, care must be taken to prevent stretching of the IAN, which may cause temporary or permanent damage.

35.8.2 Bleeding

Multiple large vessels are located near or within the surgical field of both the TMJ total joint replacement and the Le Fort I osteotomy [116, 117]. The superficial temporal, maxillary, masseteric, and facial vessels all pose a source of significant bleeding. Strict surgical technique with appropriate isolation of blood vessels is vital to avoid damage to vascular structures. If bleeding occurs, ligation or embolization of large vessels must be done to stop bleeding into the surgical field.

Intra- and postoperative bleeding from the Le Fort I osteotomy is expected. Posterior nasopharyngeal oozing and drainage often subside within 24 hours after surgery; however, blood clots and blood-tinged drainage may persist for up to 7–10 days [118]. The reported incidence of severe postoperative bleeding after Le Fort I osteotomy is relatively low, however, at about 1% [119].

35.8.3 Infections of the TMJ

Surgical site infections of the TMJ TJP can result in multiple surgical debridement or loss of the prosthesis itself. The reported rate of SSI, as previously stated, is relatively low: 1.5–4.5% [102, 104]. Treatment begins with prevention of the infection with sterile surgical technique and postoperative antibiotic therapy; however, should an infection arise prompt management is guided by onset of infection. Patients may present with acute or chronic infection in TJP, and each is managed with specific tenants in mind.

Acute TMJ TJP infections are defined as 0–24 days after surgery. Infections within this time frame are less likely to have an organized biofilm seeded onto the UHMWPE fossa component, the most likely site of biofilm development, or the condylar component [120]. Patients may be treated with a combination of inpatient IV antibiotics, incision, and drainage with debridement around the TMJ TJP irrigation catheter placement for 4–5 days after debridement, and a regiment of 4–6 weeks of outpatient IV antibiotics [121]. The irrigation drains are flushed every 4–6 hours with a mixture of neomycin and polymyxin B. The irrigation catheters are removed after 5 days. Patients treated in this manner have about 40–80% chance of successful retention of their TMJ prosthesis without the need to remove the prosthesis.

Chronic TMJ TJP infections are defined as 25 days and greater after surgery. The mean reported onset of first infection for all TMJ TJP SSIs consistently exceeds 1 month. In chronic TMJ infections, there has been sufficient time to establish a mature biofilm. Successful

treatment of these resistant, complex colonies requires a more extensive treatment regimen. Patients require broad-spectrum antibiotics and multiple-staged surgeries. Stage I surgery consists of incision and drainage with debridement of the active infection, prosthesis removal, placement of an acrylic spacer, and placement of irrigation catheters for 4–5 days. The irrigation drains are also flushed every 4–6 hours with a mixture of neomycin and polymyxin B, and the catheters are removed at day 5. IV antibiotics should be administered for 4–6 weeks. After a period of 8–10 weeks, Stage II surgery is completed to encompass TMJ total joint reconstruction with placement of autogenous fat graft around the prosthesis [121].

35.8.4 Biomaterial Sensitivity and Foreign Body Reactions

Failure of TMJ TJPs due to biomaterial reaction has been reported in past iterations of TMJ TJP. Proplast-Teflon and metal-on-metal prosthesis systems have been found to cause foreign body giant cell reactions and subsequent implant failure [122–125]. Metal allergy may also play a key role in development of foreign body giant cell reactions (FBGCR) to total joint prosthesis [126]. The pathophysiology of FBGCR in joint arthroplasty revolves around the release of foreign nanoparticles that stimulate a delayed-type hypersensitivity reaction with subsequent infiltration of T- and B-lymphocytes [127]. Under histological examination, macrophages with metal debris can be seen in the periprosthetic tissue [128]. These reactions cause loosening of hardware and inevitably failure of the TJP.

Dense fibrous connective tissue develops an envelope around current temporomandibular joint prosthesis systems without any evidence of foreign body reaction [129]. The combination of UHMWPE in the fossa component and Co-Cr-Mb alloy in the condylar component do not seem to cause the same micro debris that previous systems have failed to prevent [130, 131].

35.8.5 Recurrent Bone Formation

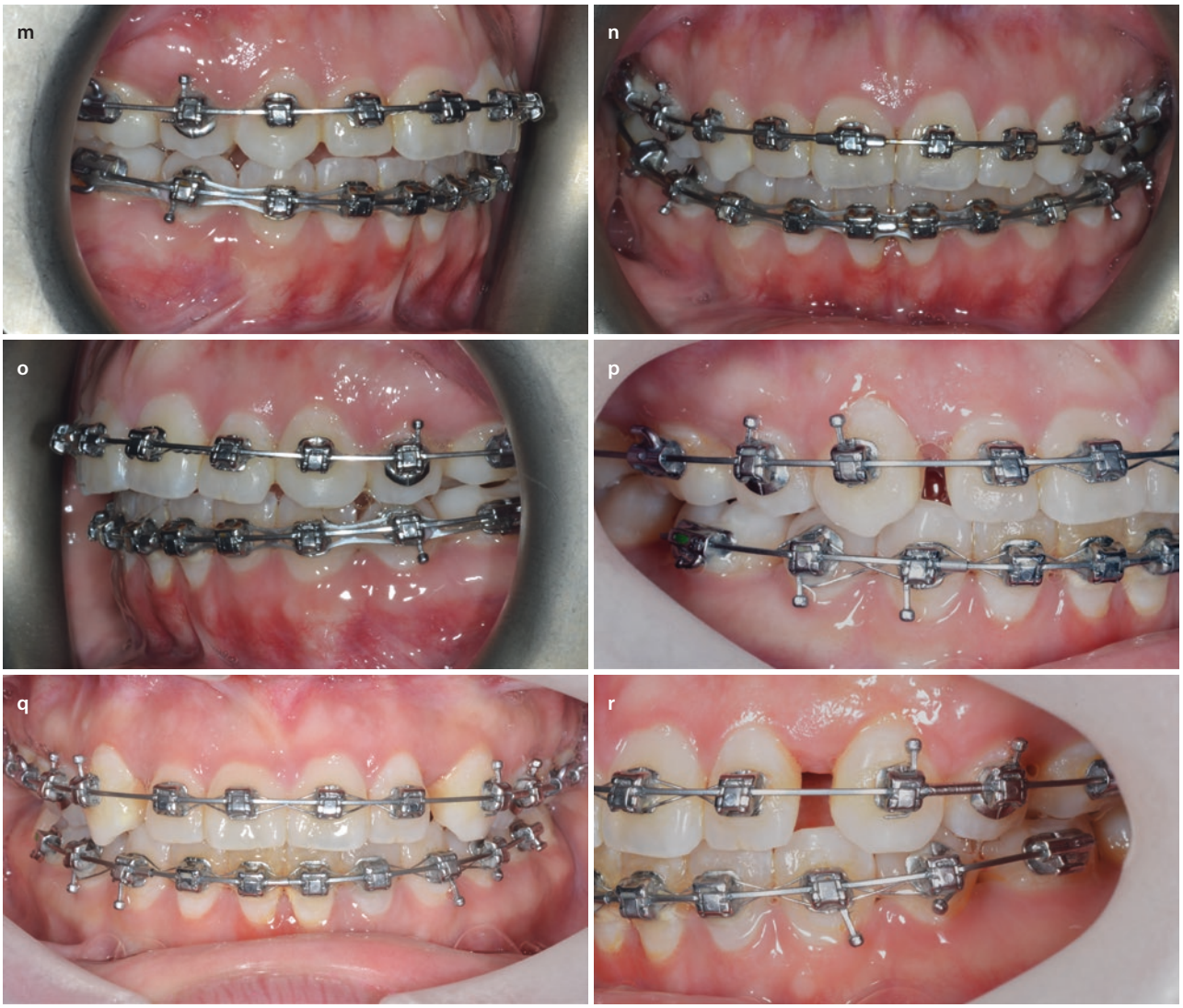
Heterotopic bone formation and fibrosis have been reported in the literature as complications of TMJ TJPs [132]. Abdominal fat graft harvesting is a simple, inexpensive, and a relatively low risk procedure that has been shown to increase MIO, increase patient perception of jaw function, and clinically reduce the risk of heterotopic bone formation [82, 84, 133, 134]. In both animal [135] and human [136] histological studies, abdominal fat grafting has been shown to prevent migration of bone and fibroses within the TMJ joint space after condylectomy [84].

The complications of autogenous abdominal fat graft harvest include infection, ileus, hematoma, seroma, and possible iatrogenic injury to surrounding structures. The incidence of complications has been between 8% and 10%. However, with appropriate sterile technique, hemostasis, and use of an abdominal binder, the complication rate can be significantly decreased.

Please see ◘ Cases 35.1, 35.2, 35.3, and 35.4 below:

Case 35.1 Patient is a 24-year-old female referred by her orthodontist for comprehensive orthodontic and TMJ evaluation. She had a severely retrognathic mandible due to idiopathic condylar resorption. At the age of 13, she noticed clicking and popping of her joints. She began orthodontic treatment at age 13 for her Class II malocclusion and had extraction of her 4s and 8s. She presented after completion of an additional 4 years of failed orthodontic treatment with progressive worsening occlusion, retruded mandible, large overjet, and upper airway compromise due to her idiopathic condylar resorption. The patient's surgery consisted of bilateral condylectomy, bilateral coronoidotomy, bilateral total joint replacements with TMJ Concepts total joint prosthesis, Le Fort I osteotomy (3-piece), a genioplasty, and a septoplasty. **a**–**c** Frontal view in repose, frontal view in function, and right lateral view at the initial presentation of the patient. **d**–**f** Frontal view in repose, frontal view in function, and right lateral view after orthodontic treatment prior to surgery. **g**–**i** Frontal view in repose, frontal view in function, and right lateral view after surgery. **j**–**l** Occlusal views prior to treatment. **m**–**o** Occlusal views after beginning repeat orthodontic treatment. **p**–**q** Occlusal views after treatment. **s**–**t** Lateral cephalogram before and after surgery

Case. 35.1 (continued)



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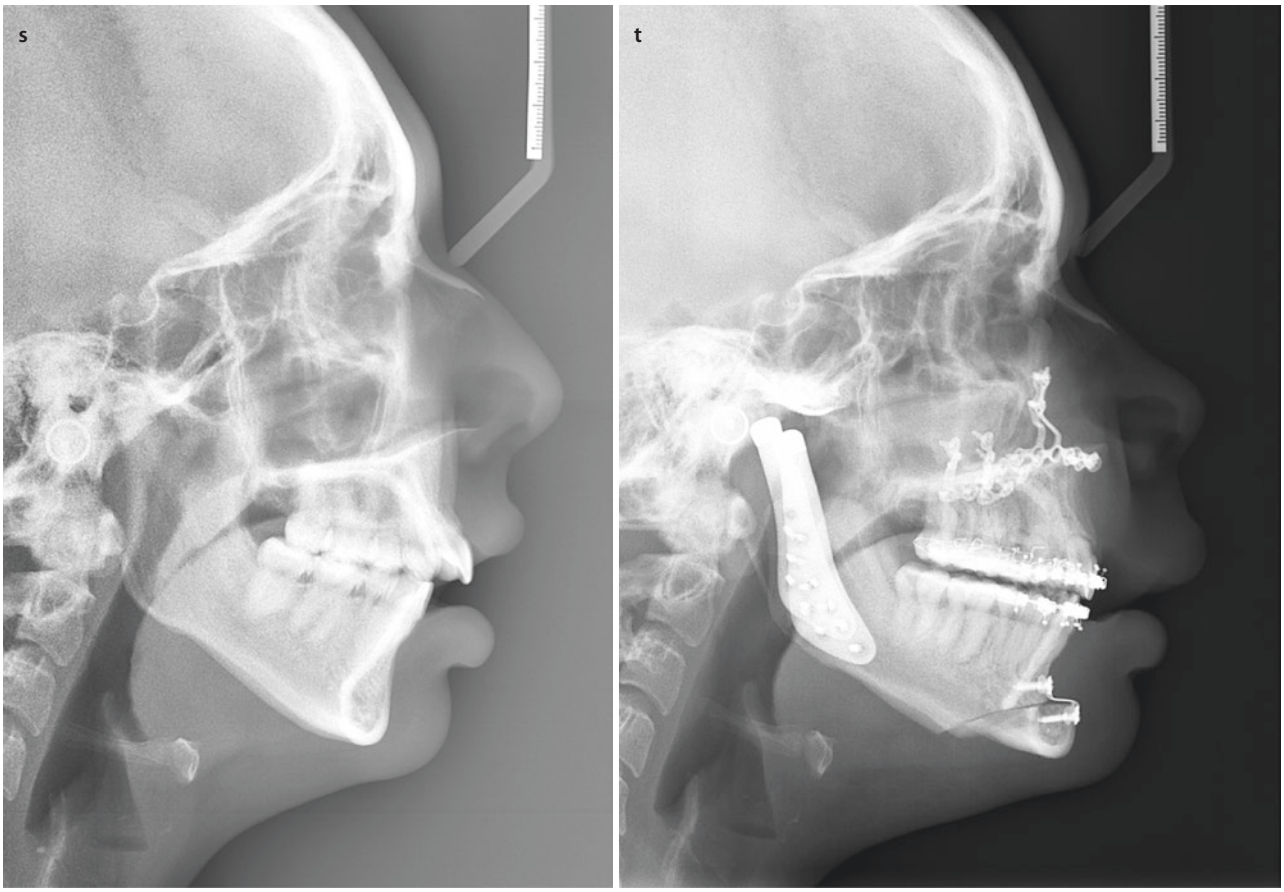

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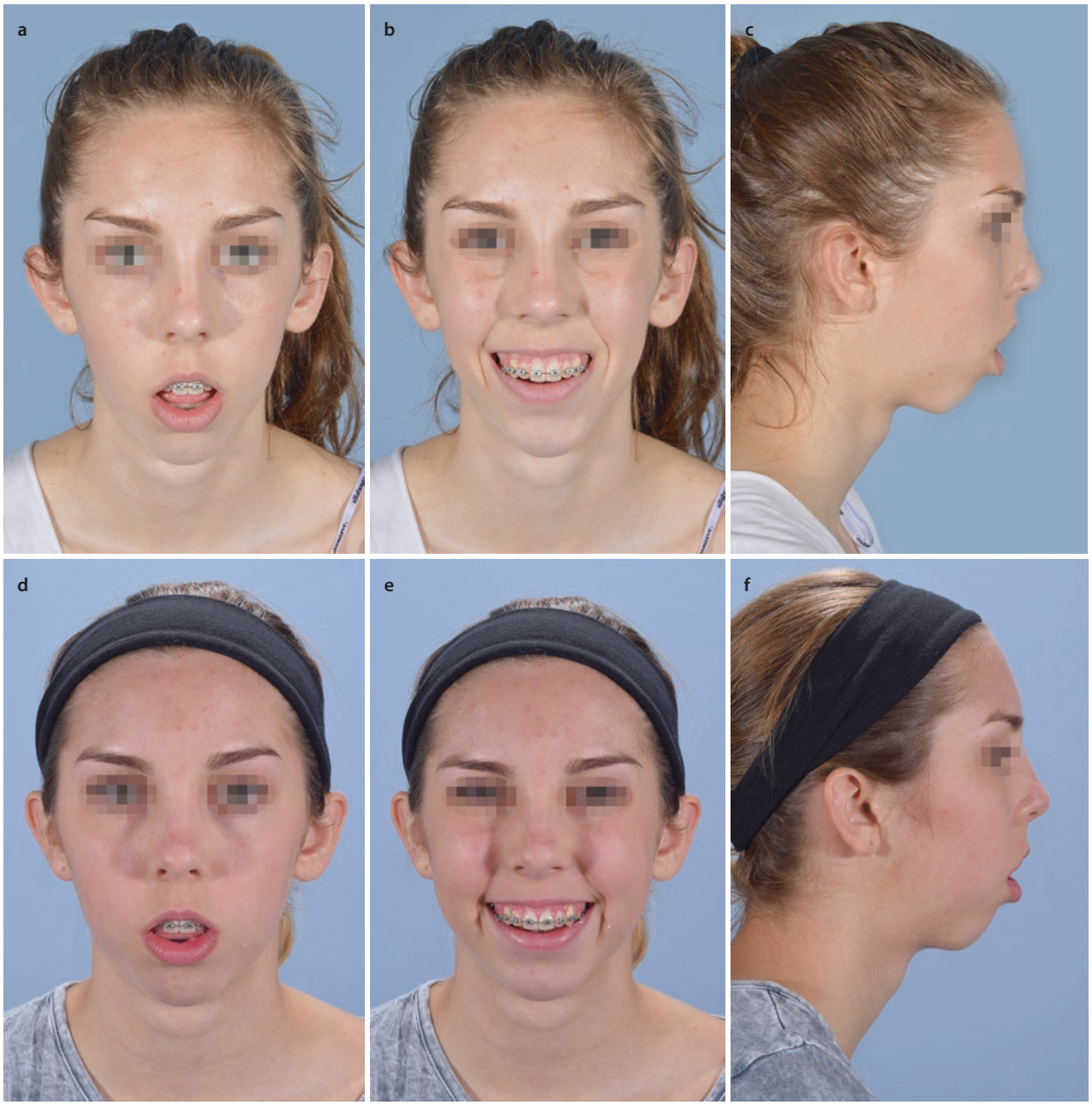
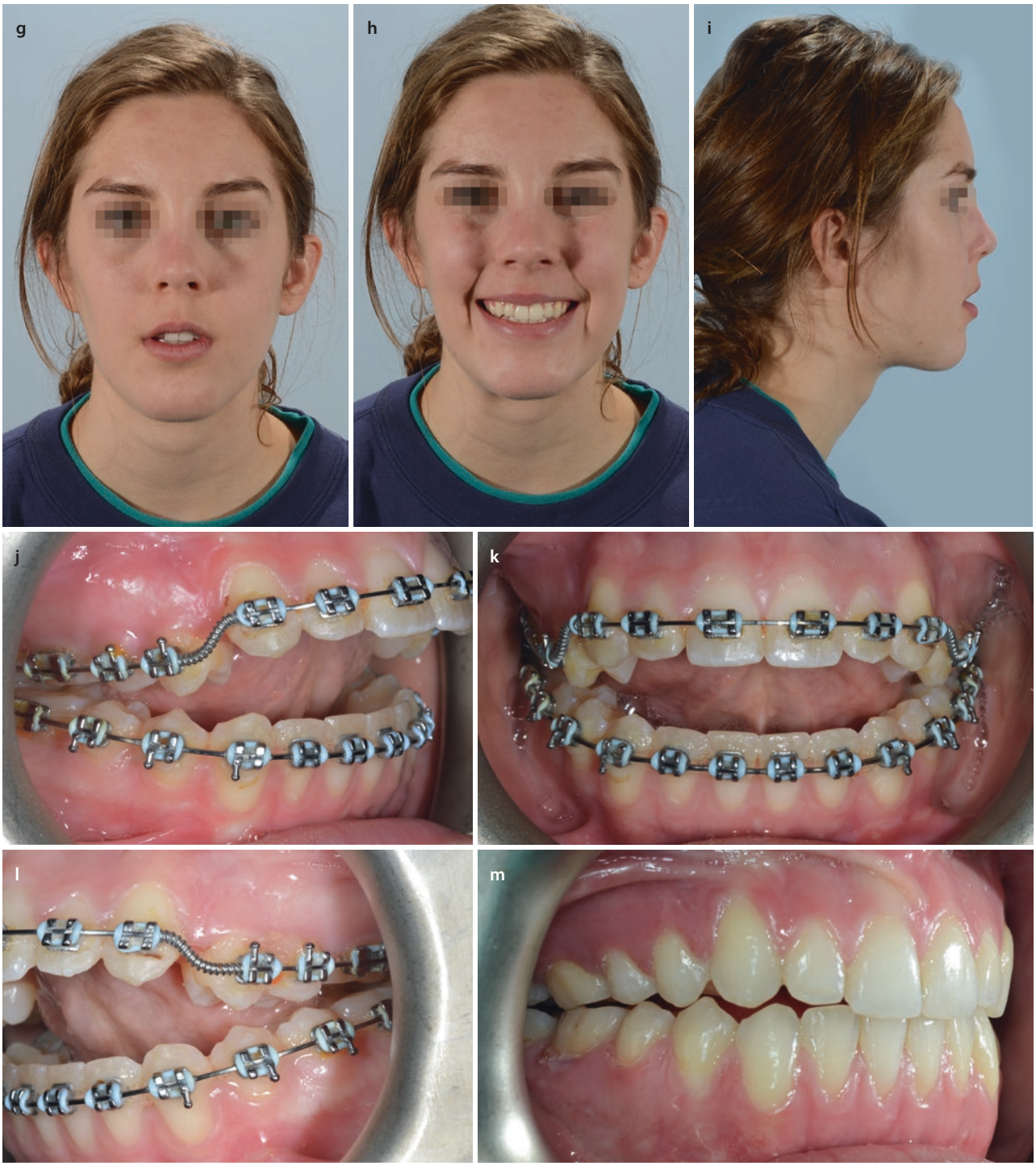


Case 35.2 Patient is a 19-year-old female referred by her orthodontist for comprehensive orthodontic and TMJ evaluation. She had previous orthodontic treatment at 13 years old with progressive worsening of her retrognathia, diet dysfunction, TMJ pain, and upper airway compromise. She presented with Class II malocclusion with a severely retruded mandible due to her degenerated condyles. The patient's surgery consisted of bilateral condylectomy, bilateral coronoidotomy, bilateral total joint replacements with TMJ Concepts total joint prosthesis, Le Fort I osteotomy (3-piece), and a genioplasty. **a**–**c** Frontal view in repose, frontal view in function, and right lateral view at the initial presentation of the patient. **d**–**f** Frontal view in repose, frontal view in function, and right lateral view after orthodontic treatment prior to surgery. **g**–**i** Frontal view in repose, frontal view in function, and right lateral view after surgery. **j**–**l** Occlusal views prior to treatment. **m**–**o** Occlusal views after treatment. **p**–**q** Lateral cephalogram before and after surgery



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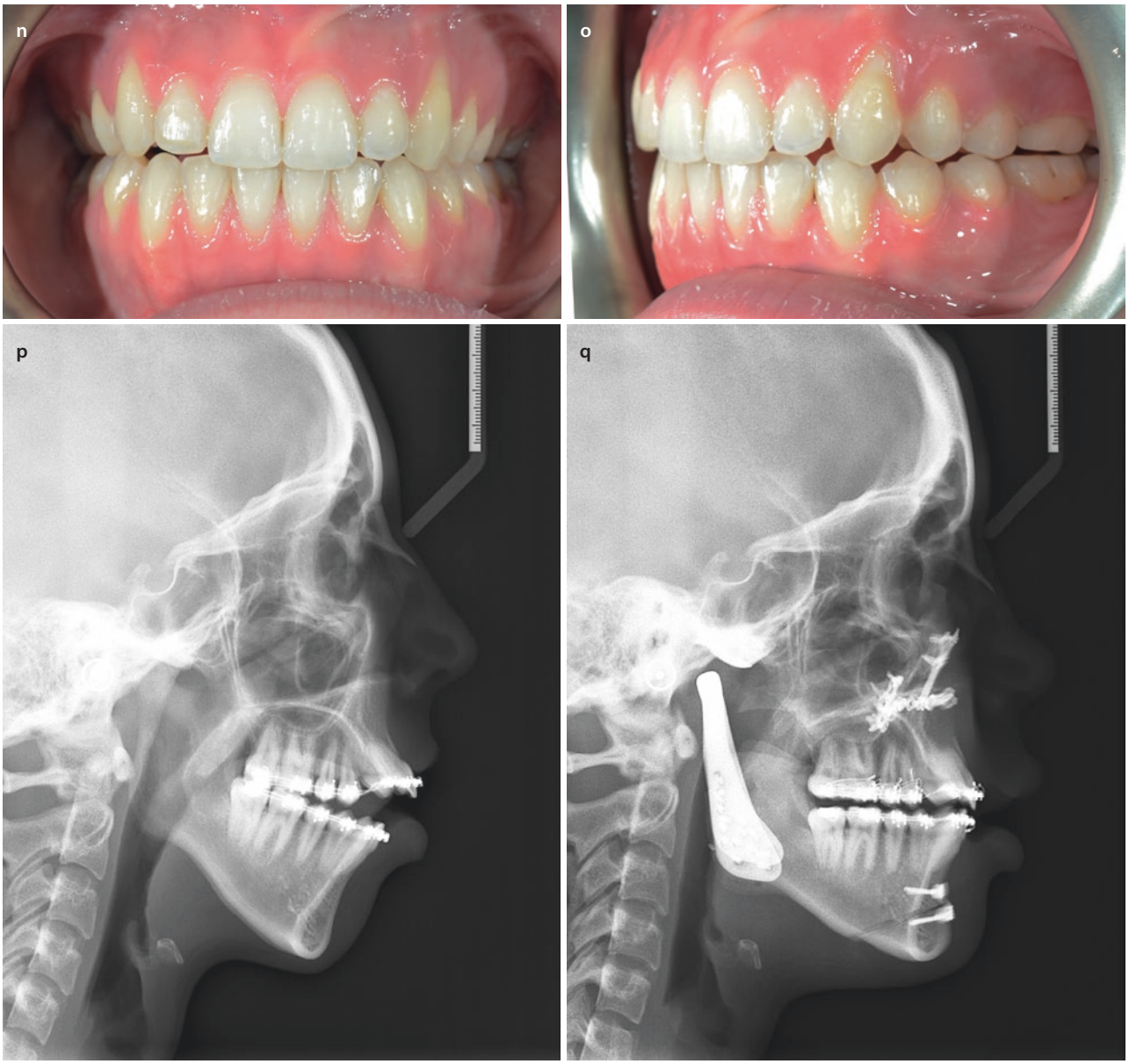
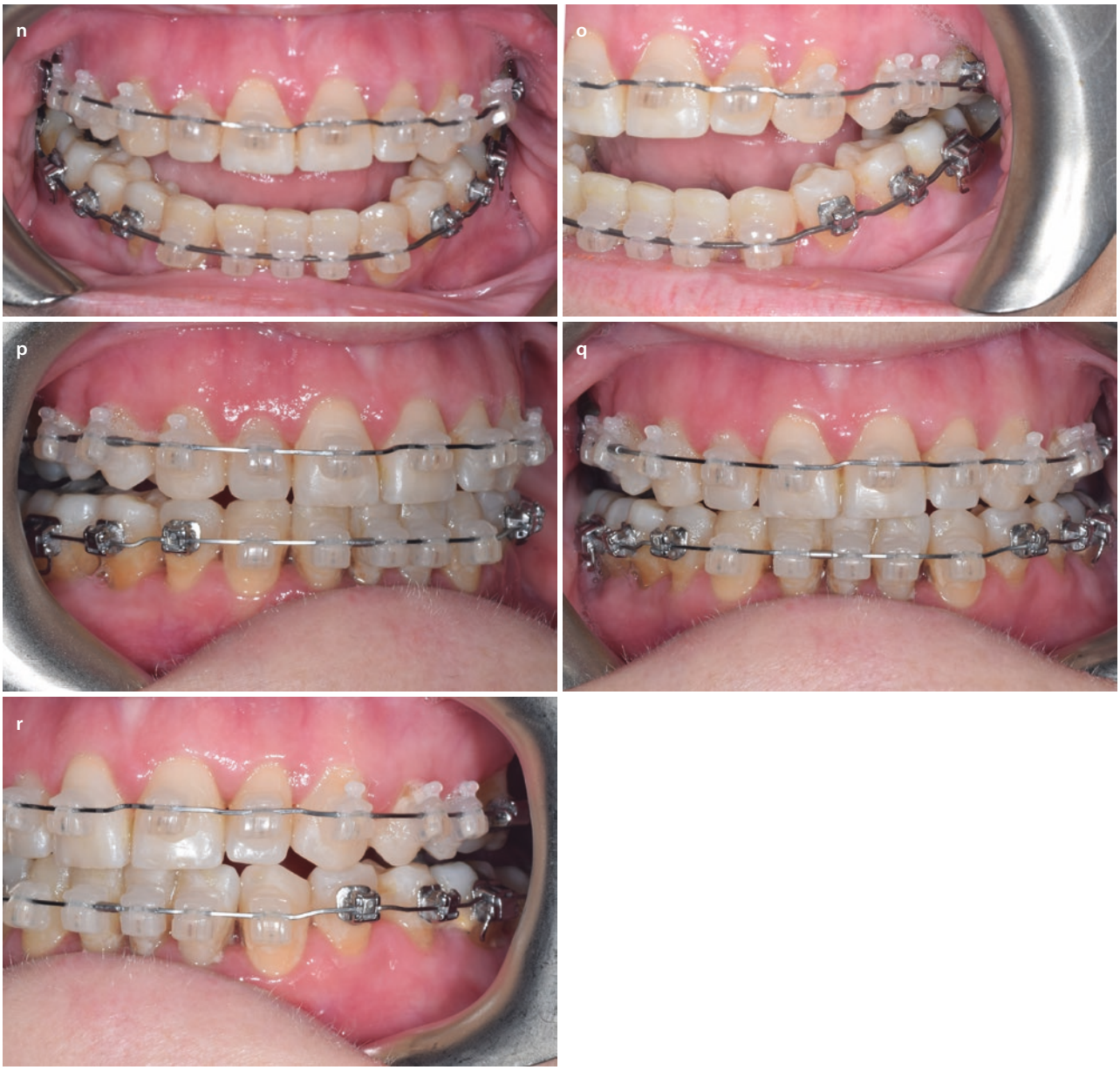


Case. 35.2 (continued)

Case 35.3 Patient is a 55-year-old female referred by her orthodontist for comprehensive orthodontic and TMJ evaluation. She was diagnosed with rheumatoid arthritis (RA) at the age of 22 resulting in bilateral hip and knee replacements, and chronic joint pain. Her chief complaint at the time of presentation was not TMJ pain but dysfunction with a progressively increasing anterior open bite, Class II malocclusion causing speech changes, and OSA. The patient's surgery consisted of bilateral condylectomy, bilateral coronoidotomy, bilateral total joint replacements with TMJ Concepts total joint prosthesis, Le Fort I osteotomy (3-piece), and a genioplasty. **a**–**c** Frontal view in repose, frontal view in function, and right lateral view at the initial presentation of the patient. **d**–**f** Frontal view in repose, frontal view in function, and right lateral view after orthodontic treatment prior to surgery. **g**–**i** Frontal view in repose, frontal view in function, and right lateral view after surgery. **j**–**l** Occlusal views prior to treatment. **m**–**o** Occlusal views after beginning repeat orthodontic treatment. **p**–**q** Occlusal views after treatment. **s**–**t** Lateral cephalogram before and after surgery

Case. 35.3 (continued)



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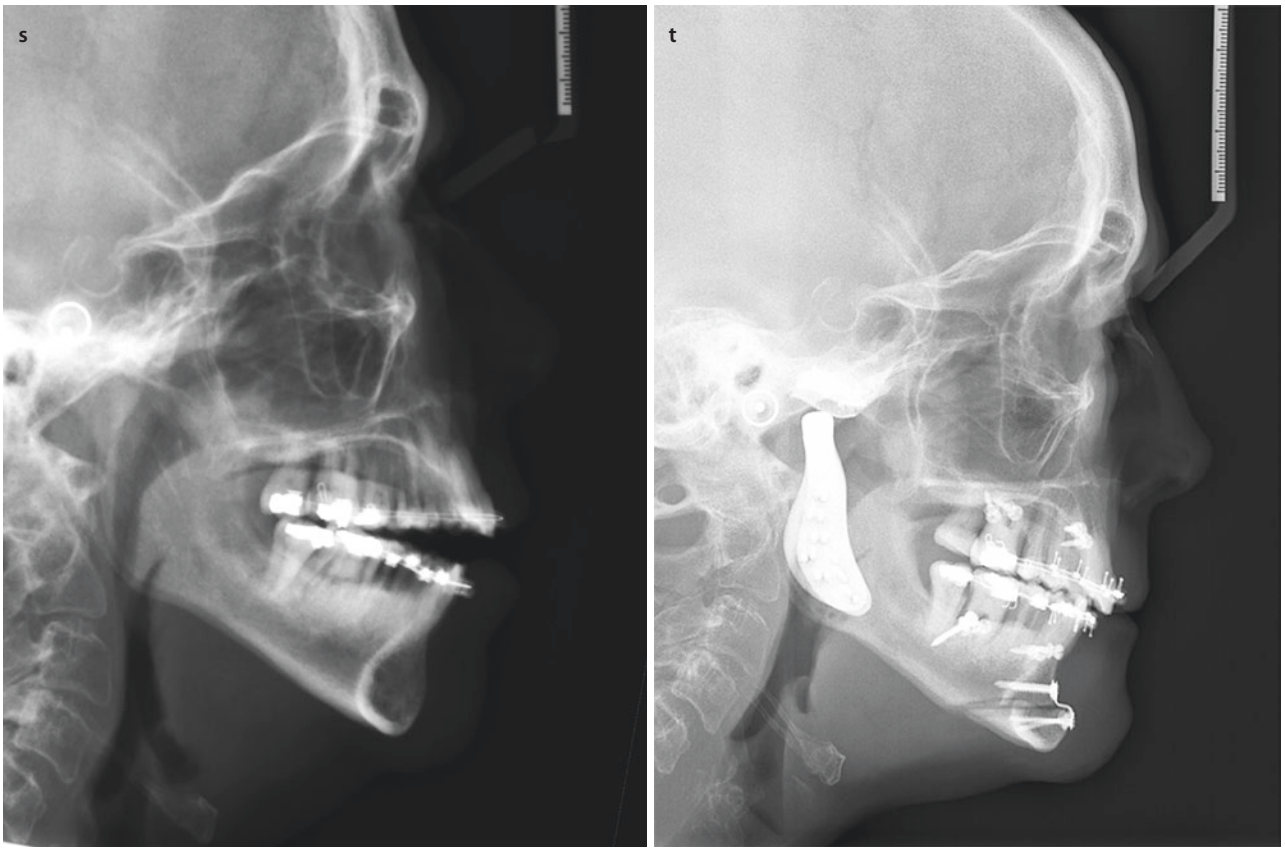
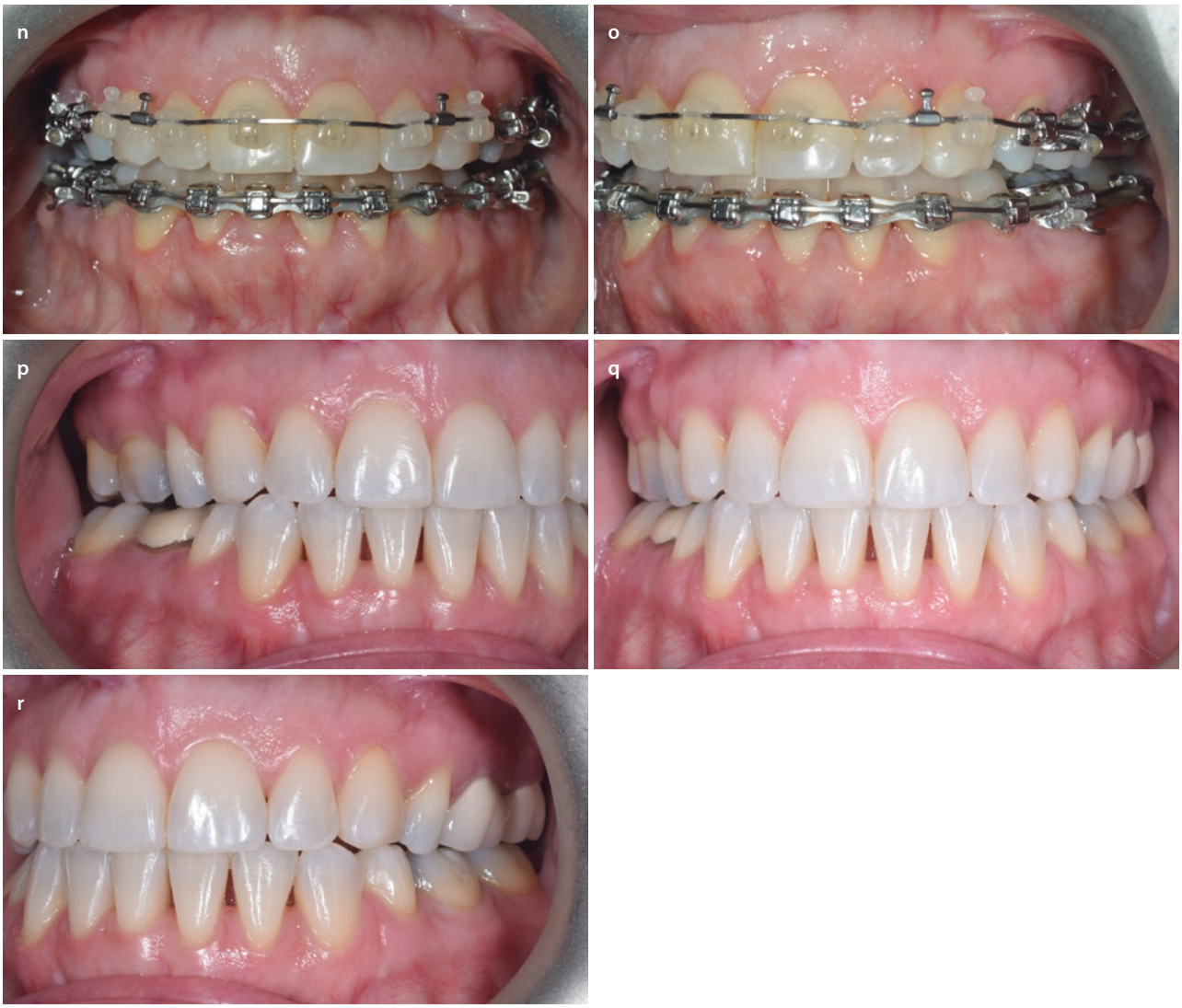


Case. 35.3 (continued)

Case 35.4 Patient is a 58-year-old female referred by her orthodontist for comprehensive orthodontic and TMJ evaluation with her primary concerns of TMJ pain, muscle achiness, Class II malocclusion, anterior open bite, and OSA. She presented without prior orthodontic treatment and multiple attempts at non-operative management for her TMJ symptoms without significant improvement. Treatment was initiated with preoperative orthodontics and extraction of her 4s. The patient's surgery consisted of bilateral condylectomy, bilateral coronoidotomy, bilateral total joint replacements with TMJ Concepts total joint prosthesis, Le Fort I osteotomy, and a genioplasty. **a**–**c** Frontal view in repose, frontal view in function, and right lateral view at the initial presentation of the patient. **d**–**f** Frontal view in repose, frontal view in function, and right lateral view after orthodontic treatment prior to surgery. **g**–**i** Frontal view in repose, frontal view in function, and right lateral view after surgery. **j**–**l** Occlusal views prior to treatment. **m**–**o** Occlusal views after beginning repeat orthodontic treatment. **p**–**q** Occlusal views after treatment. **s**–**t** Lateral cephalogram before and after surgery

Case. 35.4 (continued)



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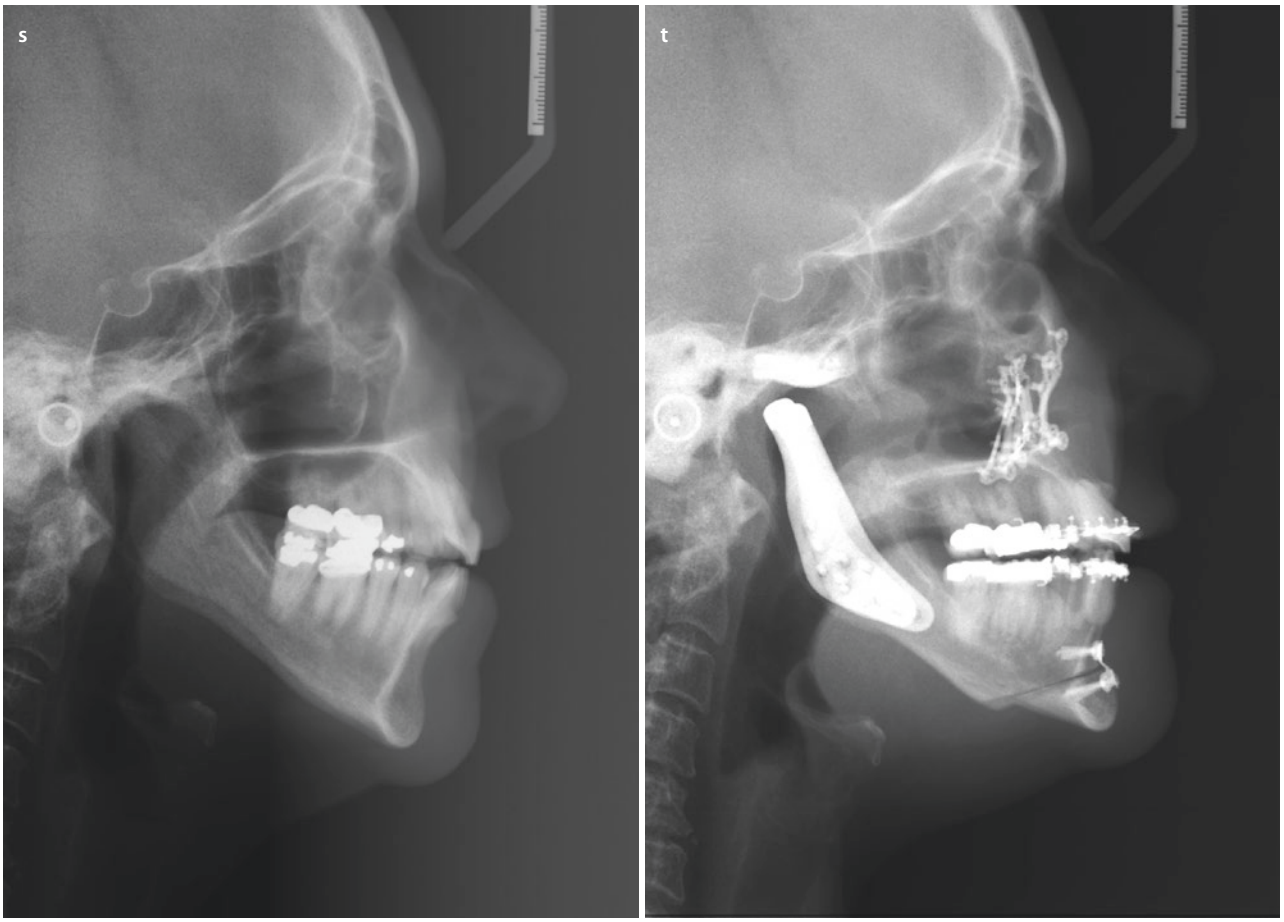

Case. 35.4 (continued)

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Computational Fluid Dynamics and Morphometric Changes in OSA

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Contents

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Computational fluid dynamics, or CFD, is a modern analysis method that uses computers to solve fluid dynamics equations for a provided geometry and set of boundary conditions. The first methods were developed in the 1920s and 1930s to predict atmospheric weather patterns and suffered from inefficient solution algorithms and inaccuracy. While these early computations were accomplished using pencil, paper, and a slide rule, general use of CFD methods among engineers did not occur until computer technology was further developed around the 1960s. Today, high-speed computers and modern solution algorithms allow CFD to be applied by many scientists and physicians of varying skill level to study a wide range of fluid flow problems in all areas of engineering, science, and medicine, including the nasal and pharyngeal airways of interest for sleep apnea studies. This chapter starts by introducing basic fluid dynamics concepts to help the reader gain insight into fluid flow behaviors. Next, the fundamentals of the CFD process are described to help the reader understand how CFD works and includes some general tips for effective use of modern programs. Thereafter, several examples are presented which use CFD tools to study the fluid dynamic impacts of various morphological changes to nasal and pharyngeal anatomies which can make breathing easier and alleviate sleep apnea conditions. This chapter concludes with an outlook for what may be possible with continued application of computational tools in the field of dentistry.

36.1 Basic Fluid Dynamics Concepts

This section contains descriptions of fluid dynamics concepts to improve the reader's insight into the relationships between geometry, volumetric flow rate, and required breathing force. Engineers use the word geometry to mean the fluid and solid domains to be studied; this is the anatomy for patient-specific geometries. The force required to move a given volumetric flow rate of air through a geometry will be described as the pressure difference (or pressure drop) between the inlet and exit boundaries of the geometry, and this pressure difference can be thought of as the breathing force necessary for a given volumetric flow rate to pass through a nasal or pharyngeal airway (since pressure is defined as a force distributed about the cross-sectional area of the airway). Just enough mathematics is explained in this chapter to help the reader gain the physical intuition that only comes through understanding the meaning of the terms in a small set of fluid dynamics equations.

36.1.1 Conservation of Mass and the Bernoulli Equation

Two of the most widely used fluid dynamics equations are the conservation of mass, also known as the continuity equation, and the Bernoulli equation. The conservation of mass states that the amount of mass flow entering a volume of space over a given amount of time must equal the amount of mass flow exiting the volume plus the amount of mass flow that accumulates inside the volume over the same amount of time. It is easy to visualize this by considering a common bathtub. Water enters the bathtub from the spout and exits through the drain. If more water is coming in from the spout than that exiting through the drain, water accumulates inside the bathtub. Likewise, if more water exits than that enters, the bathtub water level decreases. The conservation of mass can also explain why the lungs and chest must expand when we breathe air in and contract when we breathe air out, as the difference between the mass flow of air entering and exiting the lungs is related to the lungs expanding for storage of air during inhalation and contracting during exhalation. The idea of the conservation of mass can be understood more formally by considering Fig. 36.1 which shows a two-dimensional volume of space having two inlets at boundaries 1 and 2 and one exit at boundary 3, where boundaries are indicated with dashed lines; this represents a simplified nasal and pharyngeal airway where the blue arrows indicate flow direction for inhalation. Mass flow rate, $\dot{m}$, is defined by the product of fluid density, ρ, average velocity, V, and cross-sectional area, A, so that $\dot{m} = \rho VA$. To apply the conservation of mass equation, we sum the mass flow rates at all inlets and equate that to the sum of mass flow rates at all exits plus the change in mass within the volume over the period of time, as shown in Eq. 36.1:

$$\sum \dot{m}_{\text{in}} = \sum \dot{m}_{\text{out}} + \frac{dm}{dt} \tag{36.1}$$

The second term on the right-hand side of the equation contains a fraction, where dm in the numerator represents the change in mass and dt in the denominator represents a change in time. This fraction notation is more generally called a derivative, where dm/dt mathematically represents how the numerator changes as the denominator changes, or for this example how mass in the volume changes with a change in time. In nasal and pharyngeal airways, for example, there is no storage of air mass over time within the volume so the second term on the right-hand side of the equation can be neglected, and the conservation of mass relating flow rates at boundaries 1, 2, and 3 then becomes:

$$(\rho VA)_1 + (\rho VA)_2 = (\rho VA)_3 \tag{36.2}$$

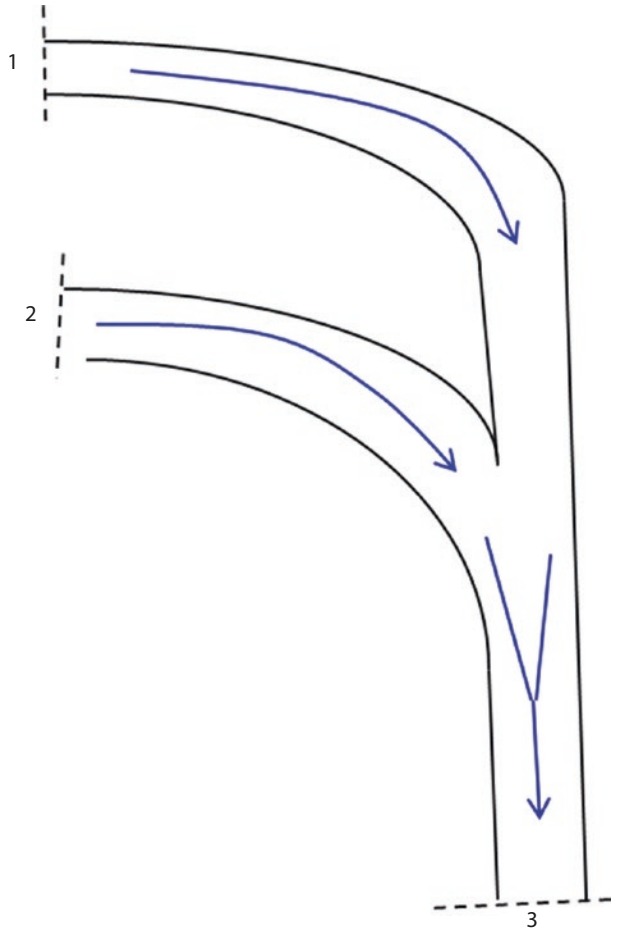

Fig. 36.1 Simple two-dimensional volume representing a nasal and pharyngeal airway

Assuming steady flow could also lead to the above equation, which assumes there is no change with time of any mass flow rate or mass flow storage (or any other fluid property at a given location). We can also assume incompressible flow for nasal and pharyngeal airway studies, meaning the density of the fluid does not change ($\rho_1 = \rho_2 = \rho_3$) and we can therefore divide by the constant density to further simplify the conservation of mass equation, as shown in Eq. 36.3:

$$(VA)_1 + (VA)_2 = (VA)_3 \tag{36.3}$$

The above equation shows how changes in anatomy, or cross-sectional areas, can affect changes in velocities across those areas.

The other most widely used fluid dynamics equation, and possibly the most incorrectly applied, is the Bernoulli equation which relates the mechanical energy between two locations in a moving fluid and includes internal energy due to pressure, p, kinetic energy due to velocity, and potential energy due to elevation, h, as shown in Eq. 36.4 where the g symbol is the gravity constant.

$$\left(p + \frac{1}{2}\rho V^2 + \rho gh\right)_1 = \left(p + \frac{1}{2}\rho V^2 + \rho gh\right)_2 \tag{36.4}$$

There are some very strict rules that are required to properly apply this equation:

1. Steady flow – assumes no fluid properties change with time
2. Incompressible flow – assumes fluid density does not change throughout the domain
3. Flow along a single streamline – assumes the fluid flows directly from location 1 to location 2
4. Frictionless flow – also called inviscid flow, this assumes viscosity does not affect the fluid as it travels along the streamline

When the Bernoulli equation is applied incorrectly, usually rules 3 or 4 are violated. Another useful simplification to the Bernoulli equation occurs with the assumption of negligible elevation change, meaning the contributions of the ρgh terms are neglected and the Bernoulli equation becomes:

$$\left(p + \frac{1}{2}\rho V^2\right)_1 = \left(p + \frac{1}{2}\rho V^2\right)_2 \tag{36.5}$$

Figure 36.2 shows a simple schematic that can be used to understand the relationships between cross-sectional area, velocity, and pressure using the simplified conservation of mass and Bernoulli equations. Figure 36.2a shows an area increase from boundary 1 to boundary 2, where flow direction is left to right. Application of the appropriate conservation of mass equation ($(VA)_1 = (VA)_2$) shows the velocity must decrease between locations 1 and 2. Application of the simplified Bernoulli equation (Eq. 36.5) with the understanding gained from the conservation of mass shows the fluid pressure must increase between locations 3 and 4 since the density is constant and the velocity at location 4 is lower than the velocity at location 3. Thus, for a constant mass flow rate, cross-sectional area increases cause a decrease in fluid velocity and an increase in fluid pressure. Note the comparison of properties using information at locations 3 and 4 along the same streamline is a valid application of Bernoulli's equation, while application of the Bernoulli equation to relate properties between locations 3 and 5 would not be appropriate since those locations are not on the same streamline. Figure 36.2b shows an area decrease, where for a constant mass flow rate, the same type of analysis reveals that the velocity must increase and the fluid pressure must decrease due to the decrease in cross-sectional area.

Figure 36.2c compares two different types of area increases, one that is sharp and abrupt overlaid onto one that is smooth and gradual. Application of the above

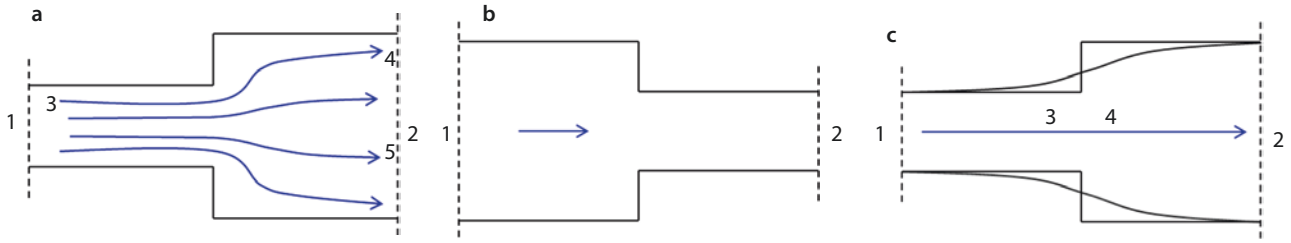

Fig. 36.2 Schematic of three example cross-sectional area changes: **a** area increase, **b** area decrease, and **c** abrupt vs gradual area increase. Flow direction is left to right

logic to these two anatomies shows the overall pressure increase from boundary 1 to boundary 2 occurs more gradually for the smoother anatomy than for the sharp increase in area. This means the localized pressure rise between locations 3 and 4 (on the same streamline) occurs more abruptly for the sharp increase in area than it does for the smooth gradual increase in area, meaning more severe adverse pressure gradients (where pressure increases with downstream distance) exist for anatomies having more abrupt cross-sectional area increases. The opposite is true for more gradual area decreases as compared to abrupt area decreases. For patients having significant enough anatomic constriction (reduction in flow area) in their pharyngeal airway during breathing, the significant area decreases have correspondingly significant decreases in local pressure which can cause the compliant airway walls to collapse inwards and exacerbate the problem, as more constricted airways require higher pressure drops to maintain constant airflow. The resistance to airflow, R, can be calculated for a given geometry by dividing the pressure drop between the inlet and exit boundaries of the flow domain, Δp, by the mass flow rate, as shown in Eq. 36.6:

$$R = \frac{\Delta p}{\dot{m}} \tag{36.6}$$

This flow resistance parameter may be a better representative measure of breathing difficulty for comparing anatomies than pressure drop alone since it also takes into account the amount of mass flow going through the passage that requires the pressure difference. Physiologically speaking, more constricted airways with higher pressure drops and higher flow resistances require the respiratory system to work harder to breathe and the patient may instead respond by receiving less air than if the airway included more gradual area changes or less constricted airways. Patients with severe enough sleep apnea use continuous positive airway pressure (CPAP) devices to maintain a positive airway pressure in the constricted zones, thus countering the decrease in pressure which could cause a collapse in airway cross-sectional area that in turn exacerbates a breathing problem. For the most severe cases of sleep apnea or if a patient cannot tolerate a CPAP device, surgical intervention may be required.

36.1.2 Viscosity, the Boundary Layer, and the Navier-Stokes Equations

The viscosity of a fluid can be considered as the stickiness of a fluid and is caused by adhesion of fluid molecules to the surface of the airway and cohesion of fluid molecules to themselves away from the wall. As a moving fluid travels over a surface, the first layer of molecules adjacent to the wall adhere to the surface and stop their motion; this is known as the no-slip wall boundary condition, where the velocity of the fluid is equal to the velocity of the surface (i.e., zero for a stationary surface). Figure 36.3 presents a schematic of three velocity profiles at different streamwise locations in a simple boundary layer on a flat wall indicated by the horizontal line with ' / ' characters below it; a boundary layer is the region of flow near a surface with reduced velocity due to viscous effects. The fluid approaches the wall from the left with a uniform inlet velocity, U, where uniform indicates the velocity is constant across the inlet plane. As the fluid interacts with the surface, the first layer of molecules adheres to the surface and each adjacent layer of fluid away from the wall is successively slowed due to cohesive viscous stresses generating the velocity gradient. The blue dotted line represents the growth of the boundary layer, where the velocity in the boundary layer is a function of surface-normal distance, y, at each streamwise location, x. The boundary layer height, δ, is defined as the surface-normal distance where the local velocity equals 99% of the unaffected velocity stream called the freestream. This boundary layer height grows with downstream distance as more and more of the fluid is slowed due to cohesive viscous stresses at the edge of the boundary layer interacting with the freestream. If the surface includes curvature, pressure forces may also speed up or slow down the fluid in the freestream and in the boundary layer following the conservation of mass and Bernoulli equation logic presented earlier.

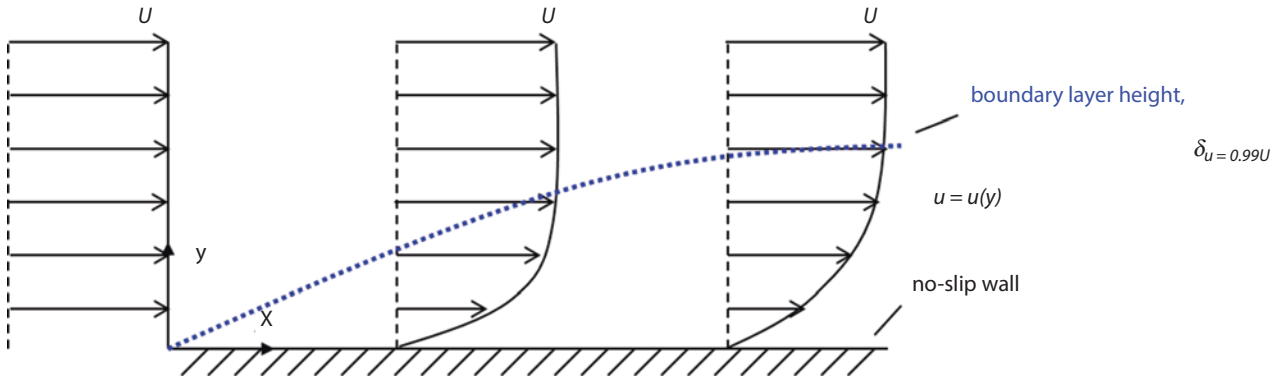

Fig. 36.3 Example boundary layer behavior of a moving fluid near a solid surface including velocity profiles at three streamwise locations

Since viscosity is important near a surface, the Bernoulli equation no longer strictly applies in boundary layer regions and a different set of equations that conserve total energy and momentum in each direction are used. The governing momentum equations for most fluid flows are the Navier-Stokes equations, which were developed from considerations of Newton's second Law of Motion for a moving fluid. Newton's second Law of Motion says the total rate of change of momentum in a linear direction is equal to the sum of the forces acting upon it in that same linear direction. The x-direction momentum equation for steady flow in the two-dimensional coordinate system depicted in Fig. 36.3 using the x- and y-directions is provided in Eq. 36.7.

$$u\frac{\partial(\rho u)}{\partial x}+v\frac{\partial(\rho u)}{\partial y}=-\frac{\partial p}{\partial x}+\mu\left(\frac{\partial^2 u}{\partial x^2}+\frac{\partial^2 u}{\partial y^2}\right) \tag{36.7}$$

In this equation, u is the streamwise velocity in the x-direction, v is the surface-normal velocity in the y-direction, μ is the coefficient of viscosity, and the del operator ∂ in the numerators and denominators indicate a partial derivative which still represents how the variable(s) in the numerator change with a change in the variable(s) in the denominator. The del operator in the partial derivatives is different than the d operator in a standard derivative as described earlier in Eq. 36.1 and indicates the numerator can change due to changes in other variables in addition to the one(s) listed in the denominator. For example, as seen in Eq. 36.7 the combination of ρu has two partial derivatives since it can change with respect to both directions x and y. Similarly, the pressure can change with respect to the x-direction as listed in this equation, but it can also change with respect to the y- and z-directions for two and three-dimensional problems, respectively; however, only the partial derivative with respect to the x-direction is included in the x-direction momentum equation as it represents the pressure gradient force in that direction. The second-order partial derivatives indicated by $\partial^2/\partial x^2$ and $\partial^2/\partial y^2$ should be interpreted as rates of change of the change in the numerator variable(s) with respect to the variable(s) in the denominator, where for example $\partial^2 u/\partial x^2 = \partial(\partial u/\partial x)/\partial x$ which describes how $\partial u/\partial x$ changes with a change in ∂x. It can be easier to understand this logic with familiar arguments of position, velocity, and acceleration. A first-order derivative of position with respect to time, or how position changes with a change in time, is the velocity where $u = dx/dt$. The first-order derivative of velocity with respect to time is then the acceleration, a, where $a = du/dt$. Acceleration could also be written as the first-order derivative of the first-order derivative of position with respect to a change in time, or the second-order derivative of the position with respect to a change in time, or $\partial^2 x/\partial t^2 = \partial(\partial x/\partial t)/\partial t = \partial(u)/\partial t = a$.

Now consider the meaning of the terms in the x-direction conservation of momentum equation presented in Eq. 36.7. The first term on the left-hand side relates how the x-direction momentum (ρu) changes in the x-direction due to the x-direction velocity (u), while the second term on the left-hand side of the equation describes how the x-direction momentum (ρu) changes in the y-direction due to the y-direction motion (v). These two left-hand side terms collectively describe how the x-direction momentum changes due to the forces which cause those motions listed on the right-hand side of the equation, including the pressure gradient force term and the viscous stress force term. We have assumed the flow to be steady, or else there would also be an unsteady term $\partial(\rho u)/\partial t$ on the left-hand side that relates how the x-direction momentum changes in time. These physical mechanisms described by terms in the equation compete to determine where the fluid moves, where pressure gradient forces are due to area or anatomy changes and viscous forces are due to proximity to a surface and the no-slip condition. There are also conservation of momentum equations for the y- and z-directions for two- and three-dimensional problems, respectively, where the y-momentum equation operates

on v instead of u and includes $\partial p/\partial y$ instead of $\partial p/\partial x$; the z-momentum equation includes the same changes for w and $\partial p/\partial z$. A typical CFD program solves this collection of momentum equations and the conservation of mass equation for a wide range of geometries and flow conditions; solution of an energy equation is required for compressible flows caused by high speed or heat transfer.

Another flow phenomenon which can appear in boundary layer regions with a strong enough pressure increase with downstream distance (i.e., an adverse pressure gradient) is called flow separation, where the near-wall fluid can actually travel upstream while the bulk fluid flow away from the surface travels downstream. If we apply the x-direction momentum equation at the wall, where velocities equal zero due to the no-slip condition, we have:

$$\left(\frac{\partial^2 u}{\partial y^2}\right)_{\text{at } y=0} = \frac{1}{\mu}\frac{\partial p}{\partial x} \tag{36.8}$$

This equation says the velocity profile curvature (second derivative) has the sign of the pressure gradient at the wall. Figure 36.4a shows three different velocity profiles under the influence of an adverse pressure gradient ($dp/dx > 0$), a zero pressure gradient ($dp/dx = 0$), and a favorable pressure gradient ($dp/dx < 0$); the straight line with slanted ticks underneath represents a flat wall surface. All three profiles have negative curvature as they meet the freestream away from the wall, but only the adverse pressure gradient produces a point of inflection (PI) due to the requirement of positive curvature at the wall, as noted in the figure with a blue circle; this inflection point makes the profile much more unstable and receptive to the influence of velocity fluctuations. Figure 36.4b shows velocity profiles approaching and passing the point of separation onset, indicated with a blue circle on the wall. If viscosity has slowed the near-wall fluid enough and the adverse pressure gradient is strong enough (such as occurring with the sharp corner from Fig. 36.2c), the near-wall fluid may not have enough streamwise momentum to overcome the adverse pressure gradient pushing backwards on the flow and the near-wall flow will eventually succumb to the pressure gradient and travel upstream. Flow separations cause displacement of the main bulk fluid as shown by the two dashed lines representing streamlines near and away from the wall. This shows how flow separations fluid dynamically create a blockage or constriction in the flow that either: (1) limits the mass flow that can pass through an airway, or (2) requires more pressure force to push the same amount of mass flow through the smaller constricted area. The separated flow also causes a separation bubble to appear, as the near-wall upstream traveling fluid recirculates with the downstream traveling fluid in the top of the recirculating region; one example streamline in the bubble region is indicated by the solid circular line. Separation bubbles oscillate in streamwise and surface-normal extent, creating oscillatory fluctuations in the fluid and on the surface. The wall shear stress follows the adjacent fluid direction, with a zero value at separation points and reattachment points and a negative value under the separated flow to indicate an upstream direction. The magnitude of the adverse pressure gradient required to cause flow separation is dependent on the streamwise flow momentum (or lack thereof) in the boundary layer near the wall, where the pressure gradient can be understood by considering the cross-sectional area changes as described earlier in ▶ Sect. 36.1.1. Even though the left- and right-hand sides of the Bernoulli equation will only exactly equal each other if the four rules are strictly followed, application of the general relationship philosophy among cross-sectional area and velocity using the conservation of mass and with pressure using the Bernoulli equation can still provide useful insight into fluid flow behavior without more involved calculation.

One last feature of boundary layer flows is important to understand for general fluid dynamics knowledge of the relationship between cross-sectional area, velocity, and pressure for viscous flows. As the boundary layer height grows in the surface-normal direction with downstream distance, the effective flow area decreases due to growth of the displacement thickness, which is a small region of flow next to the wall that displaces the main bulk fluid flow similar to but at a much smaller magni-

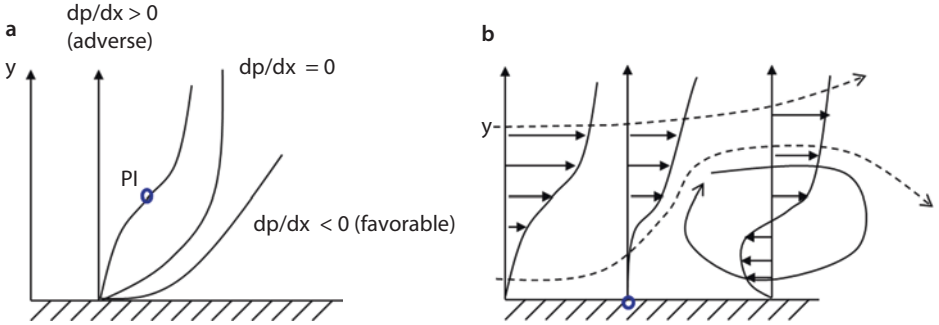

Fig. 36.4 Schematics showing **a** boundary layer velocity profiles as functions of pressure gradient and **b** progression of velocity profiles with streamwise distance under the influence of a strong adverse pressure gradient; flow left to right

tude than separated flow as shown in Fig. 36.4b. The formal definition of displacement thickness, δ^*, is presented as Eq. 36.9. This is called an integral equation, and is a calculus way of expressing

$$\delta^* = \int_{y=0}^{y=\delta} \left(1 - \frac{u}{U}\right) dy \tag{36.9}$$

additions. The meaning behind the integral is to provide a total sum amount of velocity deficit ($1 - u/U$) at a single streamwise location where viscous stresses are present, from the wall ($y = 0$) to the boundary layer height ($y = \delta$). This number increases with downstream distance due to the growth of the boundary layer and represents a fluid dynamic blockage to available cross-sectional flow area due to the growth of the boundary layer itself. Velocity and pressure respond accordingly to the shrinkage of effective flow area by increasing average speed and decreasing the average pressure with downstream distance. This effect is minimal in most situations but has a larger importance for smaller airways due to a larger percentage of total flow area being blocked and for longer airways where more boundary layer growth occurs.

36.1.3 Laminar, Transitional, and Turbulent Flows

Another nuance of fluid dynamics relevant for nasal and pharyngeal airways is how laminar flows can transition into turbulent flows. Laminar flows are characterized by minimal to negligible mixing between streamlines, where the amount of mixing is related to the level of velocity fluctuations in the fluid flow. On the contrary, turbulent flows are characterized by a high level of mixing due to rapid and strong velocity fluctuations in all three directions. Anything that can induce enough velocity fluctuations can also induce the transition to turbulence. Such mechanisms can include surface roughness, surface curvature variations, or the junction of two streams such as in Fig. 36.1. Turbulent fluctuations also take their energy from the bulk fluid's momentum, which in turn can increase the total pressure drop required to maintain constant mass flow. In general, anything that causes mixing also requires more energy and force to sustain mass flow because of the loss of energy due to the fluid shearing involved in the mixing process, and the increased energy and force result in the larger pressure drop requirement (i.e., more forceful response from the respiratory system) to maintain mass flow. An exception to this general rule is how a turbulent flow mixes near-wall fluid with fluid higher away from the wall, with the result of enlargening the boundary layer region and energizing the near-wall region. This boundary layer mixing allows turbulent flows to navigate through more severe adverse pressure gradients without separating as soon as laminar flows would under the same pressure gradients.

Turbulent velocity fluctuations produce turbulent stress forces which can also be reduced or amplified by viscous stresses in the boundary layer, depending on the relative magnitudes of flow momentums and viscous stresses. The Reynolds number, Re, is the most important non-dimensional parameter in fluid dynamics since it compares the magnitudes of the two forces always present in any fluid flow: momentum and viscous stresses, as shown in Eq. 36.10 where the length scale L is representative of the problem. For flow over aircraft wings, this length scale is usually chosen as the

$$Re = \frac{\rho V L}{\mu} \tag{36.10}$$

streamwise distance of the wing, while for flow inside circular pipes it is usually the diameter of the pipe. A hydraulic diameter, D_h, defined by Eq. 36.11 is used for non-circular duct cross-sections, where A is

$$D_h = \frac{4A}{P} \tag{36.11}$$

the cross-sectional area and P is the perimeter around that cross-sectional area. This is useful for defining Reynolds number for nasal and pharyngeal airways since those cross-sections are rarely if at all circular. In general, smaller Reynolds numbers indicate laminar flows while higher Reynolds numbers indicate turbulent flows. The range of Reynolds number values for each category depends on the geometry and length scale chosen. For example, it is common knowledge among engineers that pipe flows start to transition at a Reynolds number above 2300 or so, depending on inlet flow conditions and whether or not there are any disturbances that could cause sufficient velocity fluctuations to induce a transition to turbulence such as inner pipe surface roughness due to pipe material selection or scale growth over its service lifetime; vibration in the pipe structural network could also cause an earlier transition. Even though nasal and pharyngeal airway cross-sections are mostly not circular, the idea of transition to turbulent flow around a similar Reynolds number value (based on hydraulic diameter) as circular pipe flows can be a useful predictive indicator. As noted above, turbulent flows require more pressure drop to maintain mass flow in order to counteract the energy-stealing characteristics of the fluctuating velocity mixing processes, which also induce higher shear stresses within the fluid and on wetted surfaces than laminar flows.

36.1.4 Characteristics of Flows Through Circular and Rectangular Cross-Sections

Pharyngeal airways contain varying levels of anatomy shape morphing from the pharynx towards the esophagus. Some anatomies are more circular while others are more rectangular, but all typically include decreasing cross-sectional area towards the esophagus. Figure 36.5 presents four sets of patient-specific inlet and exit shapes where each set has the large inlet and smaller exit paired together; shapes are scaled properly for each set but scale is not the same among the four sets. These images illustrate typical variations in cross-sectional anatomic shape for the pharyngeal passage. In order to help the reader understand the fundamental fluid dynamic impacts due to these types of cross-section changes, this section includes results from CFD simulations on circular, square, and rectangular cross-sections using the commercially available CFD software SC/Tetra version 13 from MSC. Figure 36.6a shows the cross-sectional shapes of the simple models in solid lines having equal areas of 5cm^2 at the inlet and exit, with the circles and rectangles including blended variations with exit areas reduced by 10% (dashed), 25% (dash-dot), and 50% (dotted); the exit areas in cm^2 are given in the figure legend. The inlet area value was chosen, as it is similar to typical pharyngeal inlets, while rectangular shapes were determined by first calculating the square side length for a given area and then setting the rectangle height as 2/3 of the square side length for that area. The length of the rectangle is then determined by dividing 5 cm^2 by the rectangle height. Full circle and rectangle models were blended to those area reductions of the same cross-section shape, and the full circle model was also blended to the reduced rectangular cross-sections for a total of 13 different models, all of which are 10 cm long; Fig. 36.6b shows the circle-to-circle reduction and Fig. 36.6c shows the circle-to-rectangle reduction. These simple models were chosen to give the reader an understanding of the various fluid dynamic effects that can occur with the types of anatomical variations seen in the pharynx region including constrictions and airway morphing from one shape to another. Blended models were created using the Creo 2.0 solid modeling software, where the full area inlets are uniformly blended to the various full and reduced area exits. Changing the cross-section to more slim rectangular shapes also decreases the hydraulic diameter due to the area reducing faster than the reduction in perimeter; therefore, the local Reynolds number based on hydraulic diameter decreases as the shape changes towards slimmer rectangles. In addition, reducing the area increases the average velocity in the passage (for a constant mass

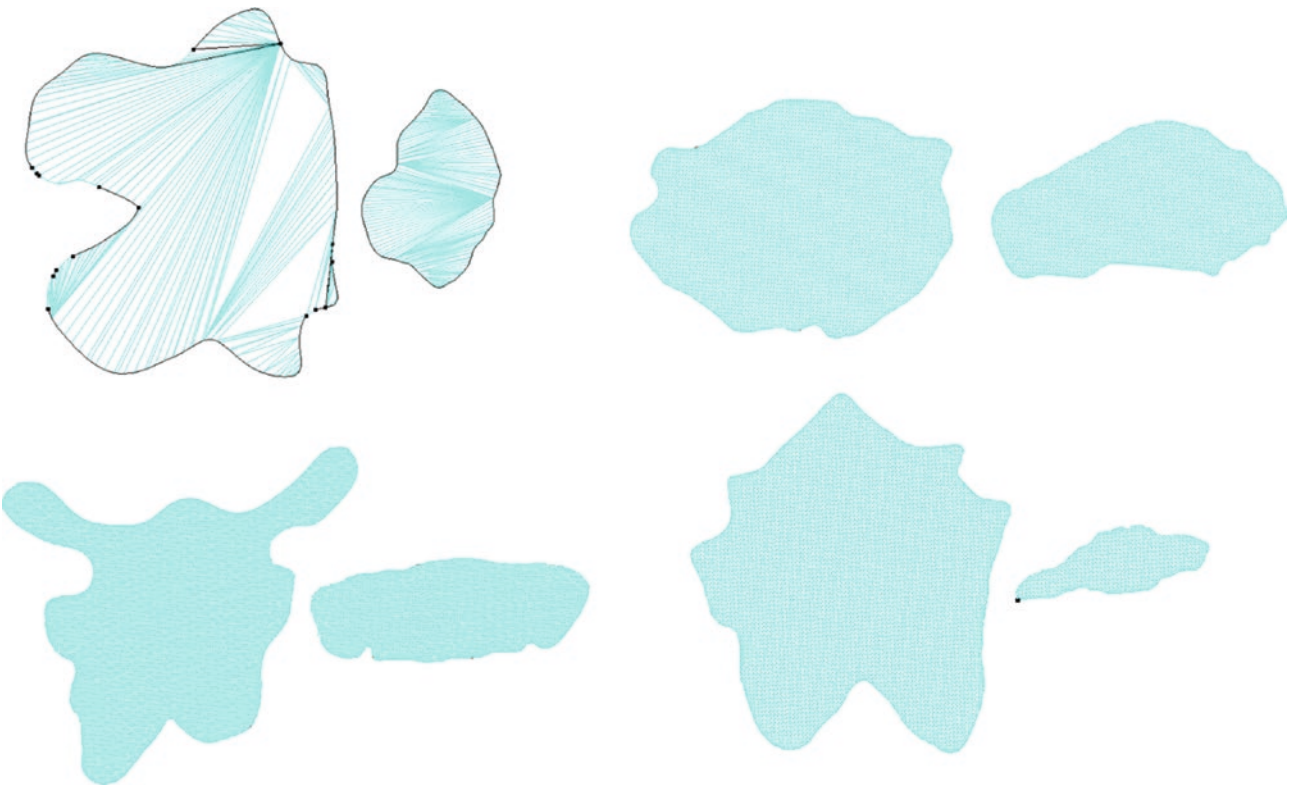

Fig. 36.5 Four patient-specific inlet and exit shapes of the pharyngeal region; scale is retained among each set but not among all four sets

flow rate) which increases the Reynolds number, and these two mechanisms compete to determine the level of velocity fluctuations as indicated by the Reynolds number's magnitude. The Reynolds numbers for all simulations were below 2300, so transition to turbulent flow does not occur due to Reynolds number effects alone for these simulations. All CFD simulations included inlet volumetric flow rates of air equal to typical breathing rates of 350 and 450 ml/s, no-slip walls for the inner surfaces, and zero relative static pressure at the exits. These inputs are the typical boundary condition settings for incompressible flows, where the actual value of pressure throughout the simulation domain is relative to zero at the exit and the CFD solver computes the magnitude of inlet pressure to create the necessary pressure drop. For example, if the exit pressure is actually 14.7 psia and the computed inlet pressure with a zero relative static pressure exit boundary condition leads to an inlet calculation of 10 psi, then the actual pressure level at the inlet is the simulated 10 psi relative to (0 + 14.7psia) at the exit, or 24.7 psia. For incompressible flows, this method of setting the pressure exit boundary condition has minimal to negligible effect on velocity and pressure drop results and is standard practice. The RNG low-*Re* turbulence model was used to close the Reynolds-averaged Navier-Stokes equations.

Table 36.1 presents the shape, defining exit dimensions in centimeters, pressure gradient result in Pascals from the inlet to the exit, and the percent changes in pressure drop over the constant circle and full rectangle exit results. Of the constant cross-section models, the circle requires the lowest force (per unit area) to push the mass flow through the tube. This is due to the effects of boundary layer merging in corners, as shown in the velocity magnitude contours of Fig. 36.7 for the exit area planes of the three constant cross-section models. In corners, boundary layers from two perpendicular walls merge and create larger regions of additional friction as compared to boundary layer growth away from the corner, and this additional friction requires additional pressure force to maintain a constant mass flow rate. The rectangular geometry requires slightly higher

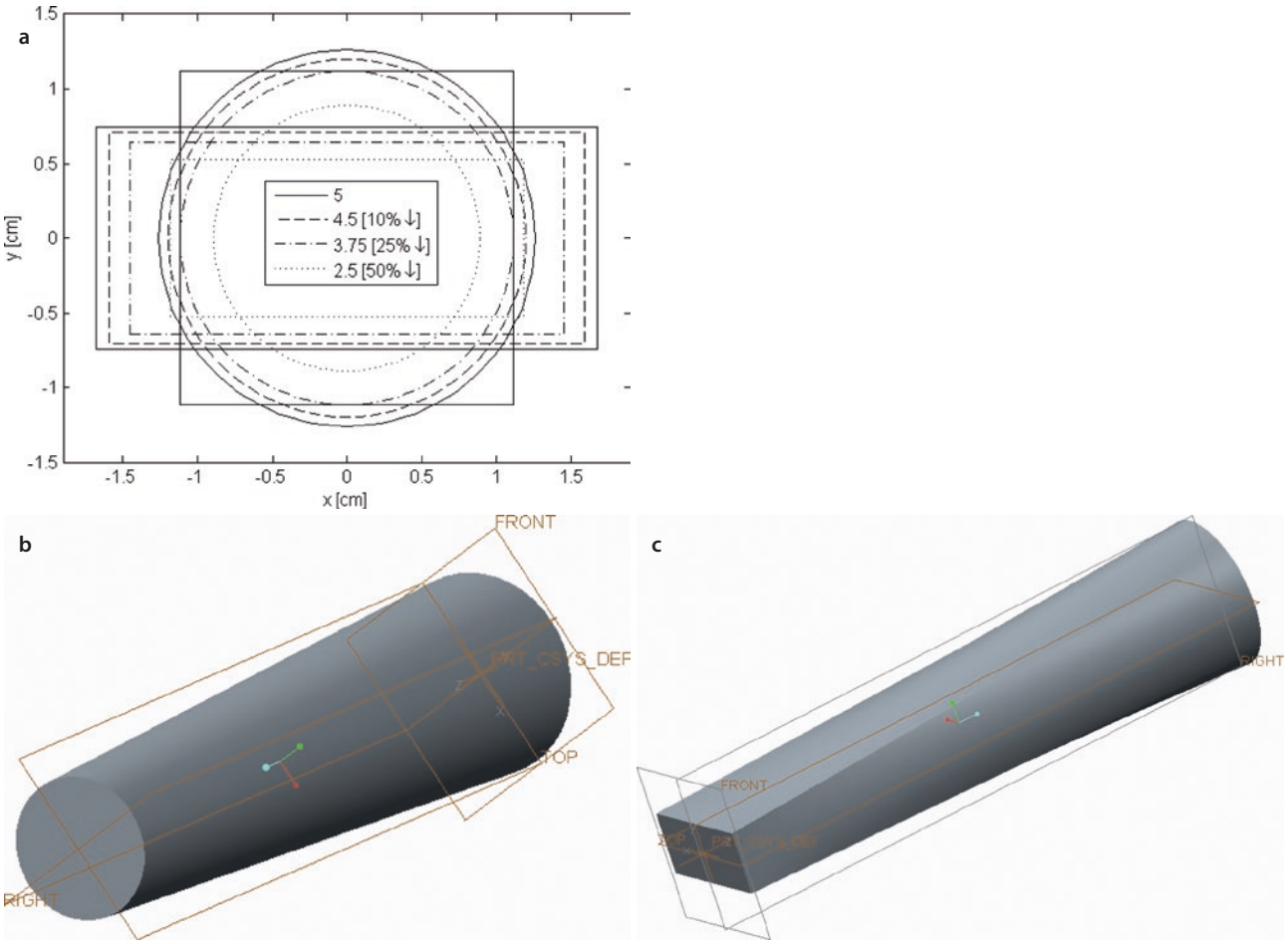

Fig. 36.6 Cross-sections for CFD simulations: **a** shapes, **b** circle to 50% circle, and **c** circle to 50% rectangle; areas in cm^2 given in figure legend

Table 36.1 Shape, exit dimensions, pressure drop, and percent changes for all 13 models at 350 and 450 ml/s

Shape	Exit[a] [cm]	350 ml/s		450 ml/s	
		Δp [Pa]	% constant	Δp [Pa]	% constant
Circ to circ	2.523 D	0.262	–	0.387	–
Sq to sq	2.236 W × 2.236 H	0.302	–	0.442	–
Rect to rect	3.353 W × 1.491 H	0.333	–	0.487	–
Circ to rect	3.353 W × 1.491 H	0.267	–	0.394	–
Circ to circ (10% ↓)	2.257 D	0.497	90	0.760	96
Circ to circ (25% ↓)	2.185 D	0.586	124	0.901	133
Circ to circ (50% ↓)	1.784 D	1.470	461	2.314	498
Rect to rect (10% ↓)	3.182 W × 1.414 H	0.440	32	0.656	35
Rect to rect (25% ↓)	2.905 W × 1.291 H	0.679	104	1.034	112
Rect to rect (50% ↓)	2.372 W × 1.054 H	1.628	389	2.539	421
Circ to rect (10% ↓)	3.182 W × 1.414 H	0.368	38	0.552	40
Circ to rect (25% ↓)	2.905 W × 1.291 H	0.598	124	0.917	133
Circ to rect (50% ↓)	2.372 W × 1.054 H	1.505	464	2.363	500

[a]*D* diameter, *W* width, *H* height

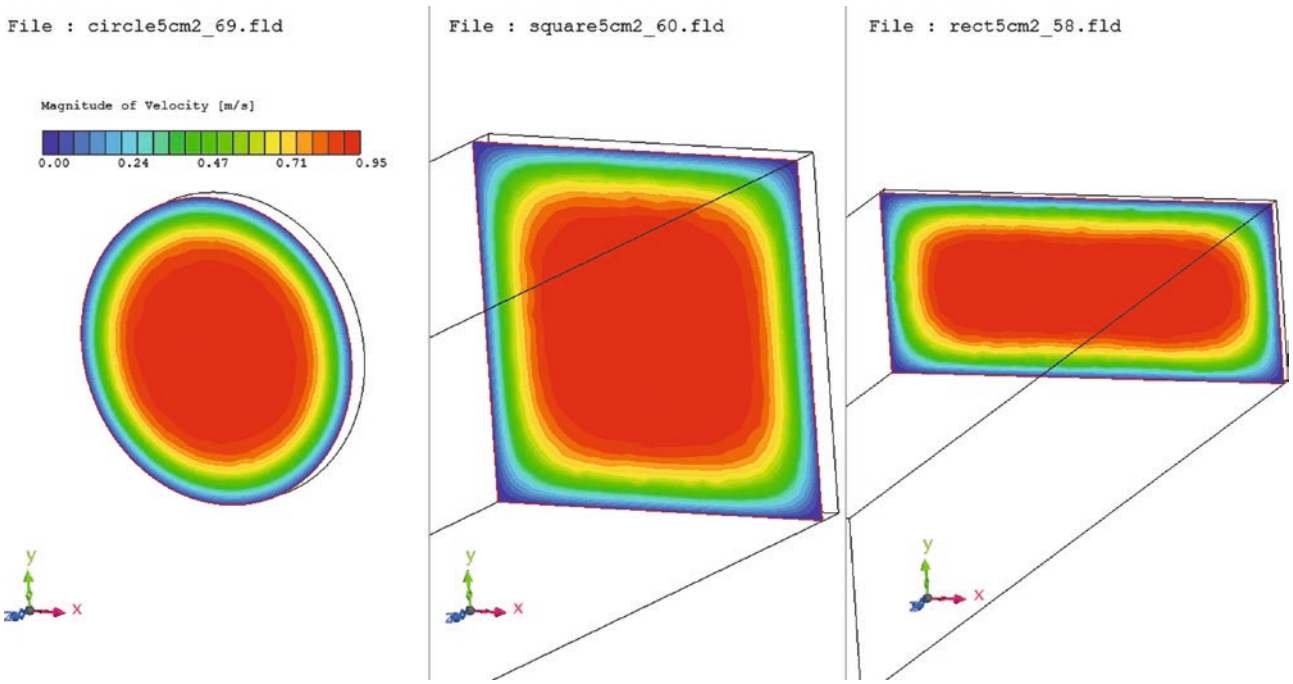

Fig. 36.7 Contours of velocity magnitude at the exit planes of the three constant cross-sections

pressure drop than the circle or square. It is interesting to note the constant area circle to rectangle geometry requires nearly the same pressure as the constant area circle, showing the effect of corners is cumulative and grows exponentially with increased downstream distance since the corners do not fully form until halfway down the model (see Fig. 36.6c). However, when reducing the area by 10% the circle geometry has the

largest increases in pressure drop (90% at 350 ml/s and 96% at 450 ml/s), while the rectangle to rectangle has the lowest increases over its base constant area model (32% at 350 ml/s and 35% at 450 ml/s) and the circle to rectangle comes in at a close second (38% at 350 ml/s and 40% at 450 ml/s). Further reductions in exit area to 25% show the circle to circle and circle to rectangle have the same pressure drop increases at 124% and 133% for 350 ml/s and 450 ml/s, respectively, while the rectangle to rectangle pressure drops only increase by 104% and 112%. A final reduction in area to 50% at the model exits again show the rectangle to rectangle performs the best at only 389% and 421% pressure drop increases as compared to 461% and 498% for the circle to circle and 464% and 500% for the circle to rectangle. This may show one influence why the pharyngeal region has evolved to be more rectangular instead of circular, since the relative pressure drop increases with constricting area are less for rectangular shapes.

All results show as the exit area decreases, the flow resistance increases, and the pressure drop increases in a non-linear fashion. This is shown more explicitly in Fig. 36.8a, which plots the pressure drop values as functions of geometry and area reduction for the models presented in Table 36.1 as well as with the inviscid pressured drops (upward facing triangles) calculated with the simplified conservation of mass and Bernoulli equations as discussed in ▶ Sect. 36.1.1. This figure shows the inviscid theory provides trend-wise insight into basic fluid dynamics, but it cannot account for real behavior with viscous flows; the difference between viscous results and inviscid theory increases with increased fluid dynamic change caused by decreased cross-sectional area. Figure 36.8b plots the respective flow resistances in Pascal-seconds per kilogram using Eq. 36.6 from ▶ Sect. 36.1.1, which some researchers argue is a more representative measure to use when comparing various pressure drops and mass flow rates, although the current figure only shows minimal data collapse due to comparing only two mass flow rates. Perhaps over a wider range of mass flow rates and more realistic pressure drops for patient-specific anatomies plotting the flow resistances would be more insightful than pressure drops alone.

36.2 The CFD Process

This section provides an overview of the four basic types of computational fluid dynamics solution strategies with a highlight of the most popular and cost-effective option. Tips and strategies for completing high-quality CFD simulations using modern CFD software packages are also included.

36.2.1 Types of CFD Solution Methods

There are four basic strategies for solving the mass, momentum, and energy conservation equations, and include the Reynolds-averaged Navier-Stokes (RANS) technique, the Large Eddy Simulation (LES) technique, the Detached Eddy Simulation (DES) technique, and the Direct Numerical Simulation (DNS) technique. The RANS technique is by far the most popular use of computational fluid dynamics due to its lower need for computational resources (memory and processor speed) as compared to the other three methods, and the common mention of CFD refers to this approach. The Navier-

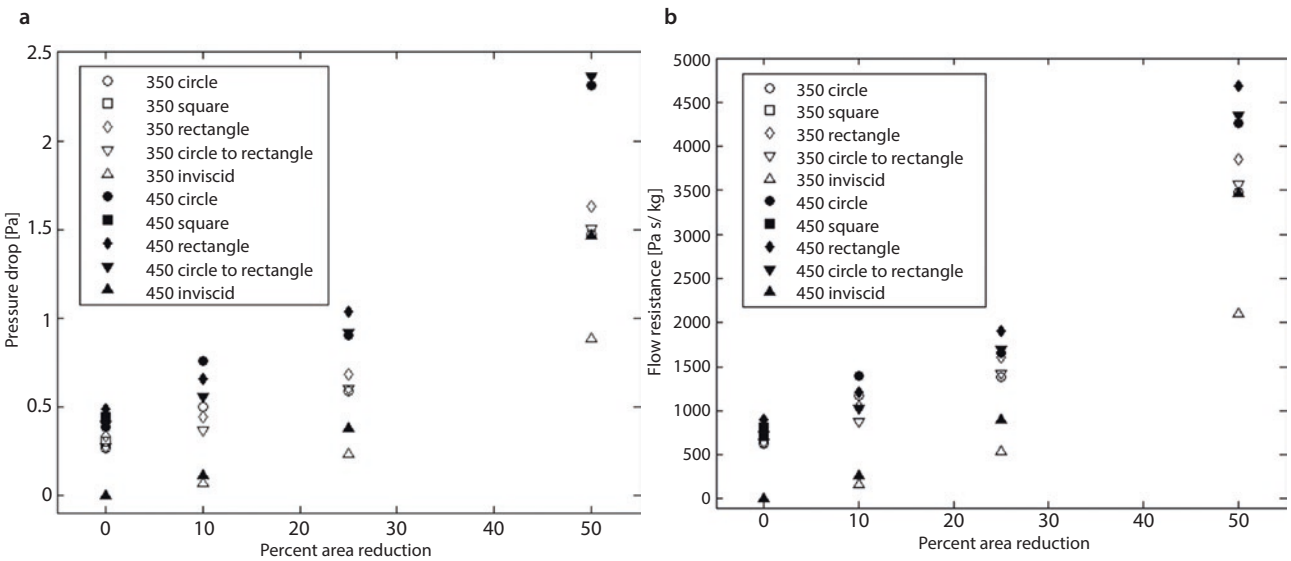

Fig. 36.8 Effects of area reduction on **a** pressure drop and **b** flow resistance

Stokes equations are modified by decomposing an instantaneous property into its time mean and fluctuation about its mean in an attempt to impose the effects of turbulence without actually simulating the direct motions of turbulence. For example, the x-direction velocity would be decomposed into $u = \bar{u} + u'$, where the overbar indicates the time mean and the prime indicates the fluctuating component. The time mean and fluctuating quantities are then substituted into the mass, momentum, and energy equations before all equations in the set are time-averaged, resulting in a set that describe the evolution of the mean velocity field. The two-dimensional x-momentum equation of the steady and incompressible RANS equations is presented below in Eq. 36.12, where the constant density has already been divided:

$$\bar{u}\frac{\partial \bar{u}}{\partial x} + \bar{v}\frac{\partial \bar{v}}{\partial y} + \frac{\partial\left(\overline{u'v'}\right)}{\partial x} = -\frac{1}{\rho}\frac{\partial \bar{p}}{\partial x} + \frac{\mu}{\rho}\left(\frac{\partial^2 \bar{u}}{\partial x^2} + \frac{\partial^2 \bar{u}}{\partial y^2}\right) \tag{36.12}$$

The third term on the left-hand side is called the Reynolds stress term and represents the effects of turbulent fluctuations on momentum transport. This term requires a turbulence model to calculate its magnitude based on local flow properties; that is what the RNG low-*Re* turbulence model did for the simulation results presented in ▶ Sect. 36.1.4. Similar terms exist for all momentum and energy equations, while the conservation of mass remains relatively unchanged (and calculates mean mass flows only).

The LES technique was developed to include more time-accurate behavior as it calculates the fluctuating quantities directly. This method operates on the conservation equations in space instead of time by filtering them into a set that calculates the largest scales of motions containing the most energy in the flow. This method requires a great computational expense as it attempts to calculate all fluctuating motions of turbulence down to the smallest grid size employed to discretize the domain, while a subgrid scale model is used to impose the effects of turbulent motions below the grid size; the subgrid scale model is essentially another type of turbulence model for LES simulations. LES is usually more accurate than RANS or unsteady RANS (URANS), but the grid requirements are so large that this method is usually cost-prohibitive due to inadequate computational resource. A hybrid technique known as DES attempts to merge the benefits of RANS and LES, where the solver switches between RANS and LES methods depending on the calculation of the flow at each grid location. Here, a turbulent length scale is calculated from local flow properties, and if it is less than the grid spacing, the RANS technique is used to model the turbulence while LES is applied when the turbulent length scale is larger than the grid spacing where it calculates the motions of fluctuations directly. This method is more accurate than RANS if applied carefully and correctly and requires less grid points and computational power than LES (but more than RANS). The DNS strategy simulates the unmodified, unsteady Navier-Stokes equations throughout a fluid domain, and requires much more computational power than LES since the grid sizes and computational time steps must be small enough to resolve the smallest turbulent fluctuations in the flow field (the Kolmogorov scales) before viscosity converts the smallest viscous motions into heat. White explains the DNS simulation of a 3.3 m/s freestream flow above a flat plate having a domain 10 cm high × 1.2 m wide × 2.4 m long would require five trillion grid points [1]; for comparison, the typical pharyngeal RANS simulation requires less than one million grid points.

36.2.2 RANS and Turbulence Modeling

As noted above, the RANS technique is the strategy most often employed when doing CFD simulations. Since it is the standard technique used for fluid flow analyses in a wide range of industries including low-speed subsonic to high-speed hypersonic flows, there are many different RANS turbulence models developed that impose the effects of turbulence that happens at different flow speeds and conditions. Accuracy with the RANS technique depends on (1) creating the appropriate CFD-ready model, (2) creating the right grid for the problem, (3) selecting the correct boundary conditions, and (4) selecting the correct turbulence model. The first standard turbulence model was the $k - \varepsilon$ model which included an equation to calculate the turbulent kinetic energy (k) and an equation to determine the turbulent dissipation (ε). These two values are used to determine an eddy viscosity, μ_t, which is then substituted into the RANS equations using the following equations for two-dimensional flow:

$$\mu_t = \rho\frac{k^2}{\varepsilon} \tag{36.13}$$

$$-\rho\left(\overline{u'v'}\right) = \mu_t\left(\frac{\partial \bar{u}}{\partial y} + \frac{\partial \bar{v}}{\partial x}\right) \tag{36.14}$$

Modern CFD packages contain a variety of turbulence models used for a variety of situations, but for most pharyngeal and nasal airways a low Reynolds number version should be used (such as the RNG low-*Re* turbulence model or similar).

36.2.3 Importance of Grid Refinement

CFD solvers compute fluid properties throughout a flow field by dividing the domain into small portions called elements. The governing equations are then computed to relate property changes between element centers or they are computed to relate property changes between each grid point defined by the junctions of element vertices. The distribution of elements is critical for accuracy of the solution as there must be enough elements to properly resolve the gradients of flow properties throughout the domain; areas with steeper gradients require higher resolution than areas with minimal gradients. Therefore, a grid refinement study must be performed for a given set of boundary conditions and grid topologies, where several simulations with increasing element counts are required to prove the grid is sufficient for a given problem. Figure 36.9a shows a two-dimensional circle with an octree distribution generated using the pre-processor of the SC/Tetra CFD software package. The octree shows the distribution of grid points where the user determines the locations of different size squares (or cubes in three dimensions) using the software, and the octree guides the development of the final mesh used for the simulation. Many commercially available software packages include a grid generator as part of the pre-processor package, while other software packages exist solely for generating high quality grids for other CFD solvers. The cubic and perpendicular arrangement of elements is called a structured grid, and the governing equations are solved at each element center or vertex to relate properties across the domain; therefore increasing the number of elements increases total simulation calculation time accordingly. Structured grid distributions are mathematically easy to build for rectangular geometries, but many practical geometries include varying levels of surface curvature which can complicate the structured grid building process. For this reason, most commercially available CFD packages include unstructured grid building algorithms that populate the domain with tetrahedral or polyhedral elements according to the size specifications guided by the octree, as shown in Fig. 36.9b for the circular cross-section.

Modern packages can also include prism layers near surfaces to enforce a structured grid where the high property gradients in boundary layer regions exist, also shown in Fig. 36.9b where three prism layers are inserted near the wall. The number of prism layers should be determined during the grid refinement study, where experience has shown the outer prism layer should be no smaller than ½ of the unstructured element size in that region (as specified by the octant distribution). Prism layers are also typically stretched from a smallest size near the surface to a larger size away from the surface; SC/Tetra requires the first prism layer size, growth rate, and number of elements to define the prism implementation. The following equation can be used to determine the outer element size using these three specifications:

$$\text{Outer element size} = (\text{1st prism size}) * (\text{growth rate})^{(\#\text{layers}-1)} \quad (36.15)$$

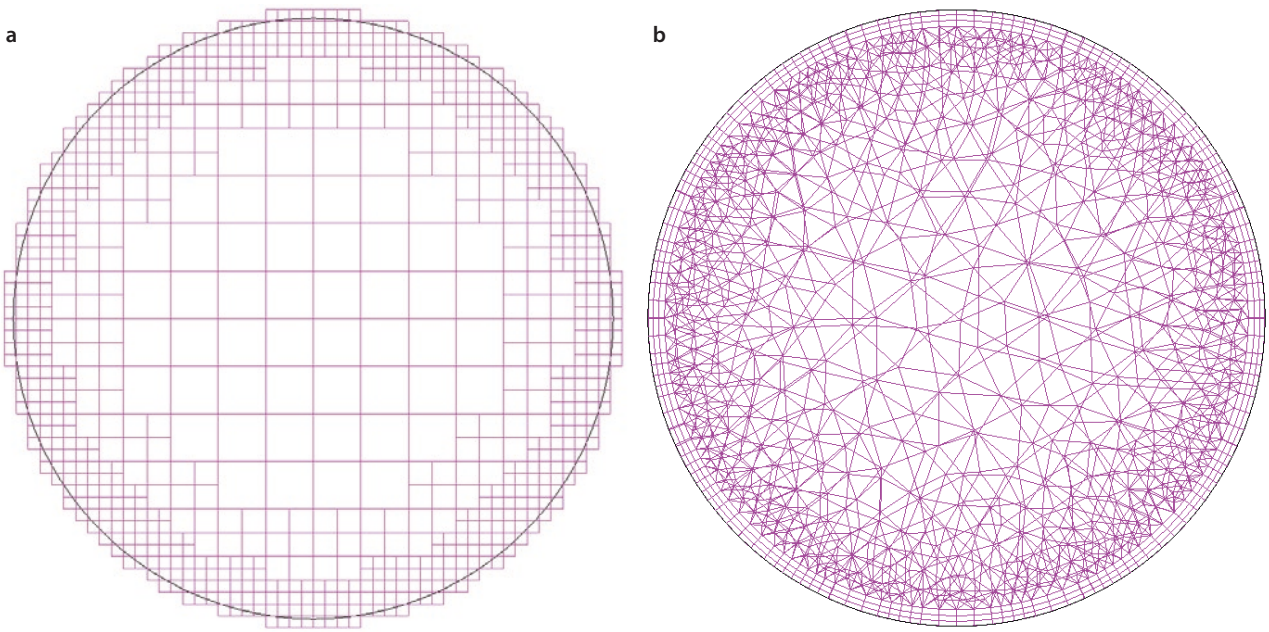

Fig. 36.9 Circular cross-section: **a** octree and **b** unstructured mesh with prism layers

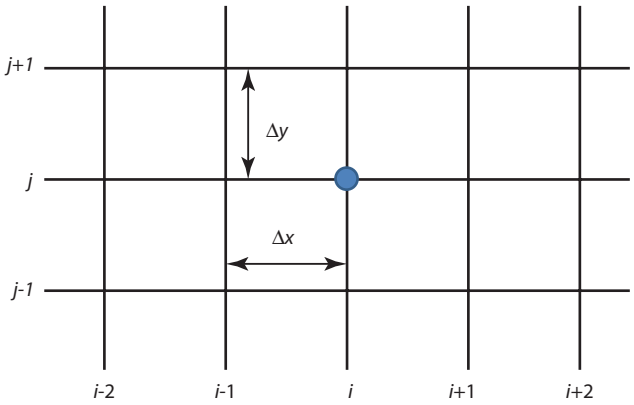

Fig. 36.10 Computational stencil for derivative calculations

The element locations can be determined relative to the octant distribution by adding all successive layer heights together. Accounting for these sizes can help prepare the appropriate octant and prism specifications to ensure numerical accuracy.

Figure 36.10 shows a stencil to help illustrate how the equations are solved on the grid, where each grid point would be assigned an i and j location index. As described in ▶ Sect. 36.1.2, the partial derivative $\partial u/\partial x$ describes how u changes with x. We can calculate this numerically on a grid for the i, j location by taking the difference in the value of u at two different i locations at the same j level:

$$\left(\frac{\partial u}{\partial x}\right)_{i,j} \approx \frac{u_{i+1,j} - u_{i-1,j}}{2\Delta x} \tag{36.16}$$

Likewise, we can calculate the partial derivative $\partial v/\partial y$ using:

$$\left(\frac{\partial v}{\partial y}\right)_{i,j} \approx \frac{v_{i,j+1} - v_{i,j-1}}{2\Delta y} \tag{36.17}$$

The two-dimensional differential conservation of mass for incompressible steady flow, which is the derivative version of Eq. 36.3, still says the amount of fluid leaving in one direction equals the amount of fluid entering in another direction, and is the addition of Eqs. 36.16 and 36.17 as shown below:

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0 \quad \rightarrow \quad \frac{u_{i+1,j} - u_{i-1,j}}{2\Delta x} + \frac{v_{i,j+1} - v_{i,j-1}}{2\Delta y} = 0 \tag{36.18}$$

Applying the above equation on the i, j location allows calculation as long as information is known at the four grid points around it, which also requires the domain to be populated with initial values everywhere at each grid point; the freestream values are typically assumed as the initial values unless otherwise specified. There are also alternative formulations of the derivative terms depending on the surrounding information used; for example, we could also calculate the $\partial u/\partial x$ term like this:

$$\left(\frac{\partial u}{\partial x}\right)_{i,j} \approx \frac{u_{i+1,j} - u_{i,j}}{\Delta x} \tag{36.19}$$

The above is called a forward difference since it uses information ahead of the current grid point, while Eq. 36.16 is a central difference since it uses information on both sides of the current grid point to relate properties across physical space. Which formulation to employ depends on what information is available. Table 36.2 presents common difference schemes for first and second derivatives where u is the variable across the x dimension. The same formulas work for other variables and directions. The differential conservation of mass is solved with the RANS equations for typical CFD studies, which also includes an energy equation if heat transfer or fluid compressibility is important to the problem (neither of which are needed for typical pharyngeal and nasal airway simulations). The influence of the input information on the boundaries of the domain, called the boundary conditions, eventually propagates throughout the domain by relating information across adjacent grid points in the partial derivative terms of the governing equations as seen above, and each iteration or cycle of the solver computes these equations at every grid point and updates property values each time. Once the changes in property values calculated with the equations are under a small tolerance everywhere in the domain, the solver is converged to the solution for the particular set of boundary conditions, modeling choices, and grid implementation. If either of those three things changes, the solver may converge to a different solution.

As noted earlier, a grid refinement study must be performed to ensure a proper grid is used for the simulation before any results can be believed. The idea of the grid refinement study is to perform successive simulations on different grids while using a metric to judge appropriateness of a grid. For pharyngeal and nasal simulations, the total pressure drop across the domain is a good metric since it is the most important result from the simulation. A course grid simulation should be attempted first, and the grid in successive simulations should be refined in areas of property gradients from the previous simulation's result. Once the metric converges, i.e. the pressure drop asymptotes to a constant value, the grid is usually considered as converged. The mathematical background of the grid convergence idea is founded on the formal calculus definition of a derivative, as shown below:

$$\frac{du}{dx} = \lim_{(x_2 - x_1)\rightarrow 0} \frac{u_2 - u_1}{x_2 - x_1} \tag{36.20}$$

Table 36.2 Common difference schemes for first and second partial derivatives

Derivative	Backward	Central	Forward
$\left(\frac{\partial u}{\partial x}\right)_{i,j}$	$\frac{-u_{i-1,j}+u_{i,j}}{\Delta x}$	$\frac{-u_{i-1,j}+u_{i+1,j}}{2\Delta x}$	$\frac{-u_{i,j}+u_{i+1,j}}{\Delta x}$
$\left(\frac{\partial^2 u}{\partial x^2}\right)_{i,j}$	$\frac{u_{i-2,j}-2u_{i-1,j}+u_{i,j}}{\Delta x^2}$	$\frac{u_{i-1,j}-2u_{i,j}+u_{i+1,j}}{\Delta x^2}$	$\frac{u_{i,j}-2u_{i+1,j}+u_{i+2,j}}{\Delta x^2}$

This says the actual derivative calculation performed using information at grid points 1 and 2 gets closer to the true derivative as the spacing in between the grid points limits towards zero (i.e. gets smaller and smaller). This is the essence of the grid refinement study – getting the grid sufficiently refined in regions where flow properties have gradients, meaning the calculated gradients on the employed grid do not change with further refinement and the grid spacing in between elements is then sufficiently small where larger gradients require smaller grid spacings. The goal of grid generation is to find the minimum number of elements that can produce the same resolution of gradients during the simulation, since increasing grid count increases simulation time. However, if too many elements are employed simulation accuracy can diverge due to computer truncation error, as more equations are solved with increasing element count and each calculation can only include a specific number of decimals related to the precision of the computer processor. So, the additive calculation truncation error over the number of grid points and simulation cycles can overcome the accuracy gained by smaller grid spacings if too many elements are used; this could be indicated by solution divergence as judged by checking the metric as described earlier.

One last grid metric is important to understand when simulating transitional and turbulent flows. Since transitional and turbulent flows exhibit turbulent stresses (fluctuations) in the near-wall region that do not occur for strictly laminar flows, transition and turbulence models require more grid elements in the boundary layer which are closer to the surface to properly resolve the near-wall behavior. The metric used to quantify surface-normal grid element distributions is the non-dimensional wall coordinate, y^+, defined below:

$$y^+ = \frac{y\sqrt{\frac{\tau_w}{\rho}}}{\nu} \tag{36.21}$$

Here τ_w is the shear stress at the wall and ν is the kinematic viscosity coefficient equal to the molecular viscosity coefficient (μ) divided by the fluid density. The first grid placement adjacent to the wall, y_1^+, is of particular importance and should be located in a certain range depending on the turbulence model used. For example, many RANS turbulence models require $y_1^+ < 1$ for greatest accuracy. This grid metric should be assessed as part of the grid refinement study. Commercial CFD packages typically contain guidance in software documentation for the appropriate y_1^+ value or range for the transition and turbulence models included with the software.

36.2.4 Model Creation

Creating a CFD-ready model can at times be as challenging as conducting a proper CFD simulation. In general, the model should contain as much detail as necessary to retain the important geometric or anatomical features but not too detailed to require excessive grid elements to resolve geometry and flow gradients. Simple models can be created in Computer Aided Design (CAD) packages and exported in file formats that can be directly read by modern CFD pre-processors including stereolithography (*.stl), step (*.stp), and parasolid (*.x_t) file types. For patient-specific models, additional software is typically necessary to translate the DICOM (Digital Imaging and Communications in Medicine) medical imaging file format usually obtained with computed tomography (CT) or magnetic resonance imaging (MRI) scanning into one of the types mentioned above that is accepted by CFD pre-processors. Some of the additional software also may contain a companion CAD program that allows modification of the DICOM information before exporting for CFD analysis.

36.2.5 Post-Processing

Modern CFD packages usually also contain their own post-processing software which allows examination of the converged solution result from the CFD solver; but

like grid generation during the pre-processing stage, there are also stand-alone software packages designed for interpreting and displaying result files from a variety of computational programs. As noted above, the average pressure drop across the domain is very important for pharyngeal and nasal studies as it directly relates to the inhalation or expiration force necessary to move the mass flow of air through the airway. Other quantities of interest may include velocity magnitudes, surface pressure and shear stresses, as well as various turbulence quantities such as turbulent kinetic energy or eddy viscosity. These quantities can typically be displayed on surfaces, across cross-sectional planes identified by the user, or extracted as line plots for further plotting with programs like Microsoft's Excel or Mathworks' Matlab.

36.3 Example Applications

This section includes several sample CFD results for pharyngeal and nasal airways from simulations conducted by the authors and their research teams. They are intended to show the type of fluid dynamic and anatomic understanding that can be learned during the CFD simulation process.

36.3.1 Pharyngeal Airway

Figure 36.11 presents contour plots across the mid-sagittal plane of a typical pharyngeal airway including static pressure, velocity, turbulent kinetic energy, and eddy viscosity where the airflow was directed from the top down to the bottom; contour levels are different for each variable and were selected to highlight the changes in properties across the domain where the contour indicates high values as red and low values as blue. The pressure contour shows the typical fluid flow behavior with higher pressure at the inlet and lower pressure towards the exit. The difference in average pressures between the inlet and exit equal the pressure drop across the domain. Comparing the pressure and velocity contours reveals how pressure decreases where velocity increases, and vice versa, due to the same logic as discussed in sections 1.2 and 1.3. The velocity contour also shows the effect of the viscous boundary layer near surfaces as the velocity has lower values near walls. The phenomenon of airway collapse during breathing can also be understood from these contours, as the higher velocity caused by the airway constriction is accompanied by lower static pressures which tend to pull in the airway even further, thereby exacerbating the relation between anatomic constrictions, velocity increases, and pressure decreases. Even though the Reynolds number for this simulation was too low to consider the flow as fully turbulent, the contour plots show velocity fluctuations are still generated throughout the domain due to anatomy changes and velocity increases. Areas where the eddy viscosity increases can also be noticed, and both of these phenomena show why turbulence modeling may be necessary for internal flows even though the bulk flow Reynolds number indicates a laminar flow regime. In general, any mechanism that can increase velocity fluctuations, such as anatomy change and velocity increases, can induce turbulent fluctuations that require turbulence modeling to account for their effects. The dimensions of the anatomy itself can also be examined from the CFD process, as shown in the figure.

Huynh et al. [2] performed CFD analyses on the pre-treatment and post-treatment pharyngeal anatomies of four patients undergoing maxillomandibular advancement (MMA) surgery, where patient-specific models were constructed from CT scan data; five patients were scanned, but only four sets of CFD-ready models could be generated and studied using CFD. Results showed three of the four patients exhibited over 90% reductions

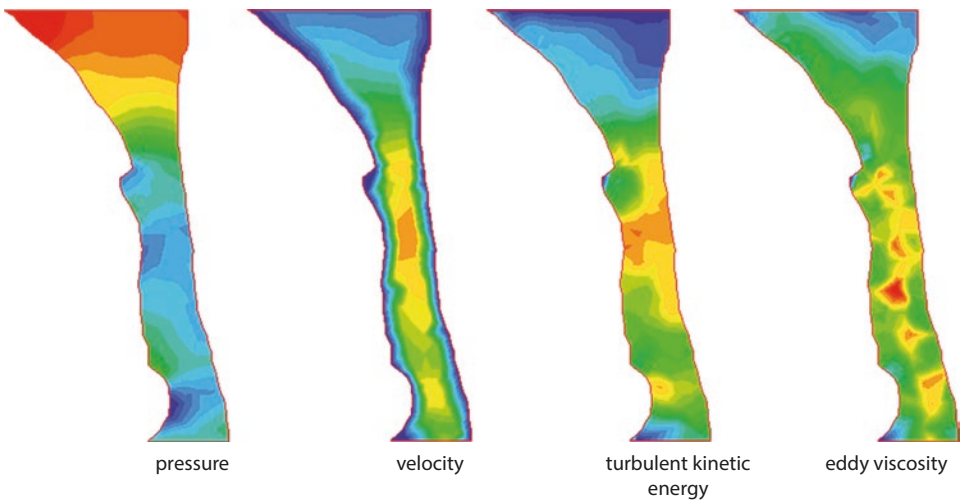

Fig. 36.11 Typical CFD results of a pharyngeal region; red indicates high values while blue indicates low values

in the pressure drop required for inhalation after the surgery, even though the Reynolds number increased into the transitional regime after the advancement surgery. More importantly, results showed the one patient with a post-surgery anatomy that exhibited an increase in required pressure drop also exhibited a decrease in local hydraulic diameter even with the advancement of the maxillomandibular region due to anatomy change that increased the local perimeter faster than the cross-sectional area to lower the hydraulic diameter of Eq. 36.11. Perhaps this should serve as a guide for surgeons to ensure increases in hydraulic diameter for constricted anatomical locations when planning surgical changes since only using linear indicators referenced to skeletal landmarks does not guarantee an improvement in breathing ease. Kim et al. have also showed the learning achieved using CFD analyses retains statistical relevance as well [3]. There are many more good quality examples of using CFD to analyze pharyngeal airways available in the open literature which can easily be found from a google scholar search or through typical library literature review searches, and the above is selected to only give an idea of the information that can be learned from careful application of computational tools.

Figure 36.12 presents results from a fluid-structure interaction (FSI) simulation which couples a fluid solver to a structural finite element analysis (FEA) software in order to include the effect of wall compliance in the simulation for a patient who had the MMA surgery, where surface stresses computed in the CFD are passed to the structural solver to calculate surface displacements which are then passed to the CFD solver to redefine boundaries which adjust the fluid behavior; this process repeats itself until the specified simulation time expires. Although these types of simulations include a large degree of uncertainty due to unknown material properties and wall thicknesses, valuable insight can be gained into the dynamics of airway morphing and collapse due to changing fluid dynamics inside the airway which occur during a normal breathing cycle. The left two images of the pre-treatment model (constructed from CT scan data) include wall displacement and velocity vectors at the time of maximum wall displacement for a sinusoidal inhalation boundary condition, showing how the velocity magnitude increase is linked to inwards wall displacement which would further exacerbate a breathing problem like sleep apnea. The two images on the right side are the wall displacement and velocity vectors for the post-surgery model at its maximum displacement condition, showing how opening the constricted airway to increase the hydraulic diameter through surgical means reduced the velocity magnitude (according to theory outlined in sections 1.1 and 1.2) and associated wall displacement. Although FSI simulations can include effects of wall compliance, the structural properties (modulus of elasticity, Poisson's ratio, etc.) and variety in those behaviors due to patient-to-patient variation complicates the universal acceptance and further application of FSI methods. In addition, high-resolution imaging is required if accurate wall thicknesses are to be determined for the pharyngeal airways. Understanding how the pharynx is structurally supported should also be improved for more accurate FSI simulations. Again, results are presented here to give the reader an idea of the type of information that can be learned through the use of computational tools.

36.3.2 Nasal Airway

Figure 36.13 presents results of CFD simulations for the nasal anatomy of a patient undergoing rapid maxillary expansion (RME), where the left image contains the computational model developed using CT scan data and the middle and right images contain static pressure distributions at position 3. The reader should notice the

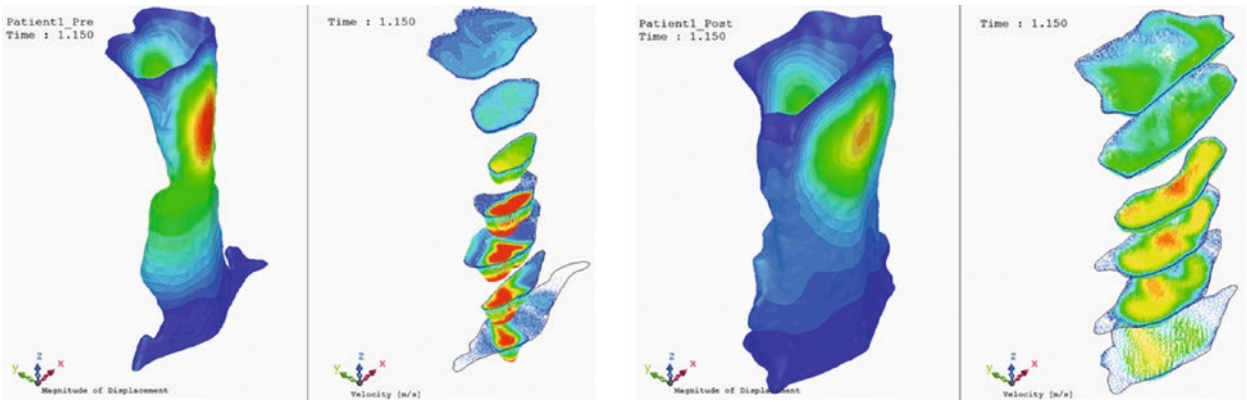

Fig. 36.12 Fluid-structure interaction simulations of pharyngeal region, before and after maxillomandibular advancement treatment; scale of images is not consistent

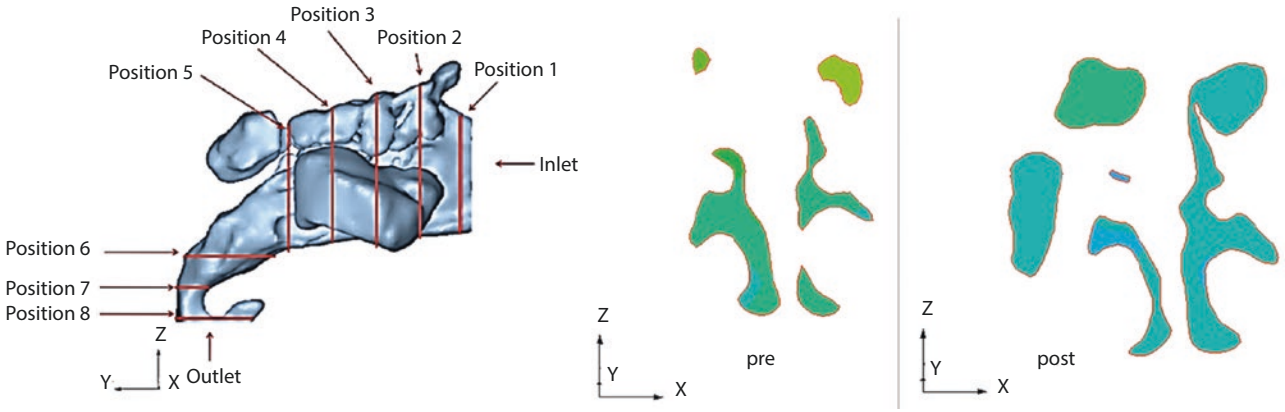

Fig. 36.13 Nasal airway CFD simulations

change in anatomy between the middle and left images, which indicate the flow path is significantly different at position 3 between the pre-treatment and post-treatment anatomies. It is difficult to understand what amount of this change is due to the RME treatment and what amount of this change is due to other features affecting the imaging process, such as the alternating congestion and decongestion associated with the nasal cycling process which can influence the CT scan results. For two patients examined pre-treatment and post-treatment undergoing 6.4 and 7.1 mm width expansions due to the RME appliance, respectively, CFD results showed patient 1 had greater than 53% improvement in pressure drop for 250 and 500 ml/s inhalation flow rates, while patient 2 had greater than 85% improvement at the same flow rates.

36.4 Summary and Outlook

This chapter described the fundamental concepts of fluid flow and computational prediction for understanding pharyngeal and nasal airway fluid dynamics as functions of cross-sectional area, mass flow rate, and breathing force. CFD simulations on simple shapes showed rectangular shapes exhibit smaller pressure drop increases with constricting area. The importance of the grid refinement study was also described to ensure simulation results are computed on a grid that is appropriate for a given model and set of boundary conditions. For anatomies exhibiting constricted airways, CFD results have shown that an increase in hydraulic diameter may be necessary to decrease required breathing force, and an increase in hydraulic diameter throughout an airway may be a better predictor of success for the maxillomandibular advancement surgery intended to alleviate breathing problems instead of traditional linear advancements relative to skeletal landmarks.

Modern computational tools have allowed more detailed study of human anatomies and fluid flow analyses on patient-specific anatomical models, and as more groups continue to use these tools the fluid dynamics rationale for and implications of surgical treatments can be more generally understood by the dentofacial medical community. Increased future use of computational tools may also lead to more predictive pre-treatment information than current strategies typically involve, and it is not difficult to imagine a time when a CT scan and associated CFD simulation can identify problem areas in a patient's anatomy and that same model data is then morphed or modified in key areas using CAD tools before further analysis with CFD in order to understand what types of surgical modifications cause what fluid dynamic behaviors. This process could be accomplished today with existing software tools and could inform surgical procedure, if the need and funding were available. Improved knowledge of the mechanical properties and responses of the pharyngeal and nasal airway walls is also necessary to make fluid-structure interaction simulations more meaningful, as most currently should only be interpreted as useful for predicting trend-wise information instead of real tissue responses since the mechanical responses of patient-specific tissues are not sufficiently understood. Several researchers are already developing methods to use modified ultrasound and magnetic resonance imaging techniques to determine mechanical properties of living tissue material [4, 5], and these methods could potentially inform more accurate patient-specific simulations that account for fluid and structural interactions with mechanical properties specific to each patient. Although these two suggestions represent advancements to come, existing computational tools are accurate and easy enough to use by a wide variety of skill levels as long as the few concerns highlighted in this chapter are sufficiently addressed.

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Supplementary Information

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