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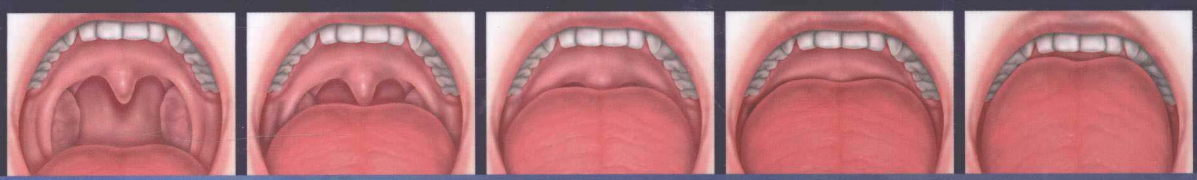
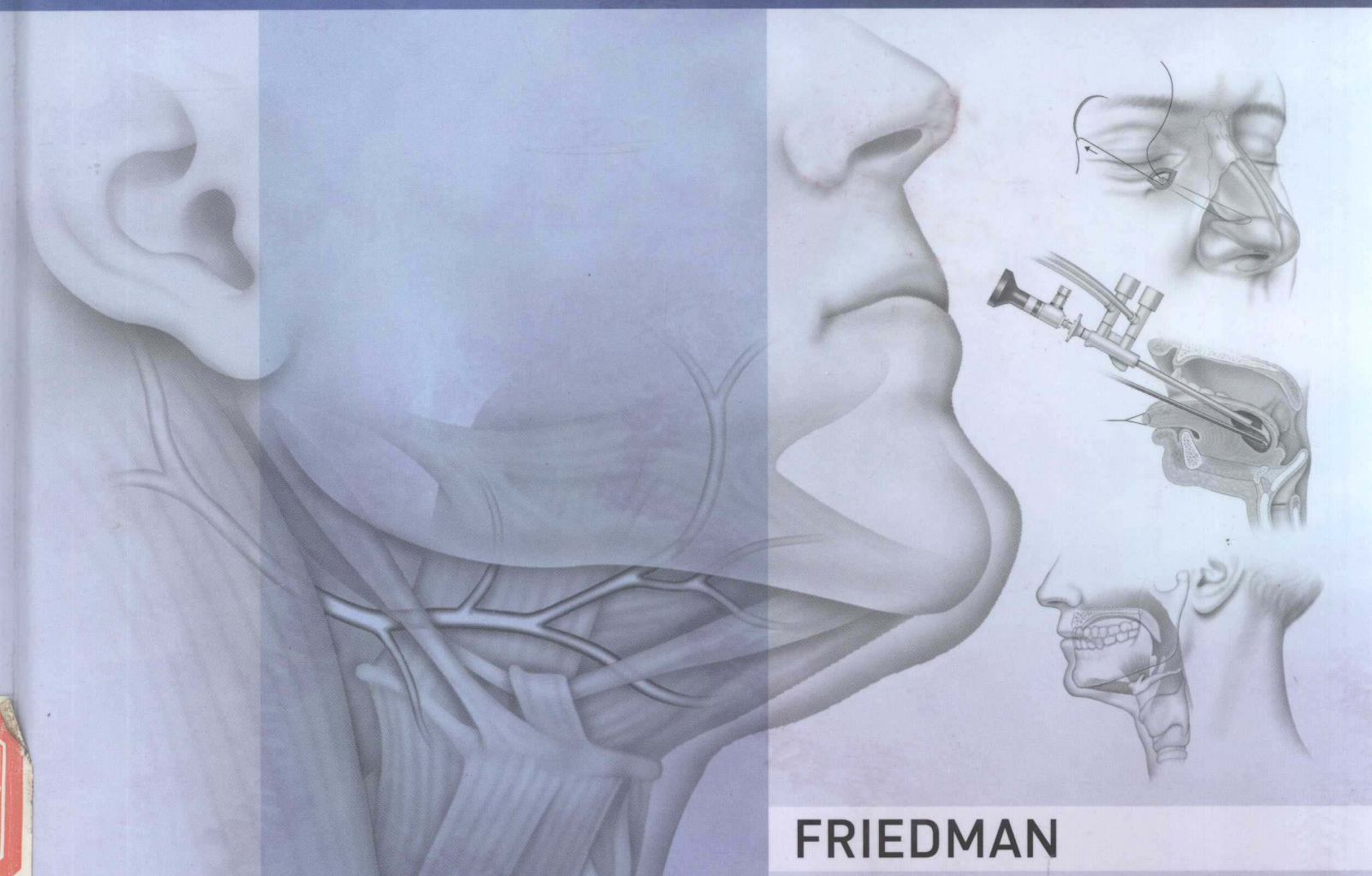


SURGICAL AND NON-SURGICAL THERAPY



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CHAPTER 1

The role of the otolaryngologist in the treatment of snoring and obstructive sleep apnea

Michael Friedman

Sleep medicine is perhaps the youngest medical specialty recognized by the American Board of Medical Specialties (ABMS). The specialty has just recently been recognized and will offer its first qualifying board examinations in 2007. Prior to that time, the American Board of Sleep Medicine recognized itself and granted certification to its own members, but was never recognized as a medical specialty. People from medical and non-medical backgrounds interested in sleep medicine often took additional training and thus qualified to be certified in this field. Although the clinical and research people involved includes a large variety of different areas, the most common specialists have a background in pulmonary medicine or neurology. Otolaryngologists have played a distant role as consultants for the definitive treatment of sleep medicine.

Neurologists have training and expertise in interpretation of polysomnographic data, which overlaps with interpretation of electroencephalograms. Pulmonologists are experienced and trained in the use of continuous positive airway pressure (CPAP). Otolaryngologists are trained and have special expertise in the upper airway. For many years, otolaryngologists were not involved in the primary care of obstructive sleep apnea. We would often deal with obstructive sleep apnea/hypopnea syndrome (OSAHS) patients if they were specifically referred for surgery. Otolaryngology, however, is a specialty that deals with both surgical and non-surgical treatment of the upper airway. We have the expertise to obtain a proper history, to do a proper examination, and to assess dysfunction and disorders of the upper airway that no other specialist has. We, therefore, are the comprehensive treaters of such disorders as chronic rhinosinusitis and neoplasms of the upper airway. We do not wait for patients to be referred in for definitive treatment of these disorders.

We screen our patients for symptoms and we always include a thorough examination of the upper airway that would alert us to abnormalities that require additional history or diagnostic testing when it comes to problems with chronic rhinosinusitis or upper airway neoplasms. When diagnostic testing is ordered, we control those tests and independently evaluate the results even when other specialists – such as radiologists – interpret the examinations. After the evaluation, we help guide the patient to select the proper treatment.

We should take this approach for patients with OSAHS. OSAHS is extremely common and often overlooked by both patients and physicians. We should screen every patient for both symptoms and physical findings suggestive of OSAHS. This should be part of a thorough routine upper airway examination. We should have enough basic understanding of polysomnography to determine if the testing that is done is adequate. We should be able to offer our patients both surgical and non-surgical treatment.

Many patients seen by sleep specialists are never informed of surgical options. They are told that CPAP therapy is their only option. Although CPAP therapy is clearly safer and more effective than any surgical therapy, there is a huge number of patients who cannot or will not use CPAP.

Sleep medicine is a dynamic and rapidly growing field in which opportunities for physicians of many backgrounds abound. The newly recognized status of the field and the establishment of a certification examination by the

Box 1.1 Symptoms associated with obstructive sleep apnea

Adults	Children
Heavy persistent snoring	Snoring
Excessive daytime sleepiness	Restless sleep
Apneas as observed by bed partner	Sleepiness
Choking sensations while waking up	Hyperactivity
Gastroesophageal reflux	Aggression and behavioral disturbance
Reduced ability to concentrate	Frequent colds or coughing
Memory loss	Odd sleeping positions
Personality changes	
Mood swings	
Night sweating	
Nocturia	
Dry mouth in the morning	
Restless sleep	
Morning headache	
Impotence	

ABMS will accelerate the growth of the field and increase the importance of board certification of practitioners. The public will increasingly expect that physicians who provide comprehensive sleep medicine services will have ABMS Sleep Medicine board certification. The new ABMS examination, starting in 2007, will be co-sponsored by the American Board of Internal Medicine, the American Board of Psychiatry and Neurology, the American Board of Pediatrics, and the American Board of Otolaryngology.

There are three pathways that qualify physicians to sit for the new examination: (1) certification by one of the primary sponsoring boards and the current ABMS; (2) certification by one of the primary sponsoring boards and completion of training in a 1-year sleep medicine fellowship program, not overlapping with any other residency or fellowship; and (3) clinical practice experience: this clinical practice experience pathway consists of a 5-year 'grandfathering' period open to physicians who are board certified in one of the sponsoring specialty boards and who can attest that he or she has the equivalent of 1 year of clinical experience in sleep medicine during the prior 5 years. This experience could, for example, be gained by an individual practitioner who has devoted one-third of his or her practice to sleep medicine over 3 years, or by someone who spent 25% of their practice in the field over the past 4 years. Physicians in the clinical practice pathway would also have to attest to a specified minimum number of patients seen and polysomnograms and multiple sleep latency tests read. At the end of this initial 5-year period, the only route to board eligibility will be through an accredited fellowship training program. This creates a one-time, unprecedented opportunity for pulmonologists, neurologists, psychiatrists, and other physicians already working in the field to sit for the board examination.

While no one knows the number of 'unboarded' sleep medicine practitioners, we are confident there are a considerable number of otolaryngologists and other specialists who practice sleep medicine who could, with a little work, become board certified in the next 5 years. The necessary work might include strategic use of continuing medical education activities in sleep medicine, reading review articles and texts, reviewing cases with experts, and board review courses. We believe a larger number of boarded sleep medicine physicians will be good for the field and, additionally, good for patient care, and will help address future workforce issues in sleep medicine.

Whether we choose to be boarded in sleep medicine or not, we are clearly the upper airway experts and, as such, should take the primary responsibility for treating this disorder. We can certainly consult with sleep specialists during the care of our patients. We should, however, be involved in the diagnosis, testing, and treatment in all areas.

CHAPTER 2

Signs and symptoms of obstructive sleep apnea and upper airway resistance syndrome

Christian Guilleminault and Shanon Takaoka

1 INTRODUCTION

Obstructive sleep apnea (OSA) and upper airway resistance syndrome (UARS) represent two distinct but related entities in the spectrum of sleep-disordered breathing (SDB). OSA is characterized by repetitive partial or complete collapse of the upper airway during sleep, resulting in disruptions of normal sleep architecture and usually associated with arterial desaturations.[1] If these respiratory events occur more than five times per hour of sleep and are associated with symptoms, most commonly snoring, excessive daytime fatigue, and witnessed apneas, the term obstructive sleep apnea/hypopnea syndrome (OSAHS) is applied.[2] UARS is a more recent entity and describes patients with symptoms of OSA and polysomnographic evidence of sleep fragmentation but who have minimal obstructive apneas or hypopneas (Respiratory Disturbance Index < 5) and do not exhibit oxyhemoglobin desaturation.

Epidemiologically, OSAHS is estimated to affect 2–5% of the population.[3–5] Although it can occur at any age, OSAHS typically presents between the ages of 40 and 60 and increases with age.[6–8] Men are twice as likely to develop OSAHS with an estimated prevalence of 4% vs. 2% in women.[8] Other at-risk groups include postmenopausal women[9–11] who have a two to three-fold increase in prevalence of OSAHS, Pacific Islanders, Hispanic-Americans, and blacks.[5,12–15] Additionally, obesity and weight gain have been shown to be important risk factors in the development and progression of OSAHS in middle-aged adults.[8,16] UARS epidemiology is less well characterized, and to date, there has been no reliable assessment of its prevalence in the general population. Compared to OSAHS, there appears to be no gender bias,[17] and patients with UARS are commonly non-obese (mean Body Mass Index of $25\,kg/m^2$)[18,19] and are frequently younger (mean age of 37.5 years).[18]

OSAHS has been shown to be a gradually progressive disease, even in the absence of weight gain.[16,20] Some have attributed this slow progression to upper airway damage characterized by palatal denervation with a localized polyneuropathy and inflammatory cell infiltration of the soft palate thought to be caused by snoring-related vibrations and/or large intraluminal pressure oscillations in the setting of obstruction.[21,22] As OSAHS worsens in severity, it has been shown to be associated with the development of significant medical co-morbidities, including hypertension, cardiovascular disease, stroke, obesity, and insulin resistance. Furthermore, the presence of OSAHS has been linked to an increased risk of motor vehicle accidents,[23–25] impaired daytime performance and quality of life,[26,27] and increased mortality independent of co-morbidities.[28,29]

2 CLINICAL ASSESSMENT

A thorough history (with participation by the bedpartner if possible) and physical examination are integral to the initial evaluation of patients with suspected SDB. However, studies show that the predictive value of these clinical tools is poor. In an observational study of 594 patients, the sensitivity and specificity of subjective clinical impression in determining the presence of OSAHS were 60% and 63% respectively.[30] This study also showed that history, physical examination, and clinician impression were only able to predict OSAHS in about 50% of patients. Furthermore, none of the commonly reported symptoms alone has sufficient predictive value to provide an accurate diagnosis of OSAHS.[31] Diagnostic accuracy can be improved by identifying constellations of symptoms, such as snoring and witnessed apneas, which increase the sensitivity and specificity of OSAHS diagnosis to 78% and 67% respectively.[32,33] Ultimately, overnight polysomnography remains the gold standard in the initial diagnosis of SDB and the only means of distinguishing OSAHS from UARS. However, as such testing can be expensive, time-consuming, and may not always be readily available, a thorough history and physical examination remain important tools to identify those patients who need further evaluation by polysomnography.

3 CLINICAL PRESENTATION OF OSA

3.1 SLEEP-RELATED (NOCTURNAL) SYMPTOMS

Snoring is the most frequently reported symptom in OSAHS and is found in 70–95% of such patients.[34] Typically, the snoring may have been present for many years but has increased with intensity over time and is further exacerbated by nighttime alcohol consumption, weight gain, sedative medications, sleep deprivation, or supine position. Snoring may become so loud as to be greatly disruptive to the bedpartner and is often a source of relationship discord; in one report, 46% of patients slept in a different room from their partners.[35] The characteristic snoring pattern associated with OSAHS is one of loud snores or brief gasps alternating with 20- to 30-second periods of silence. Because snoring is so common in the general population (35–45% in men, 15–28% in women[8,36]), it is a poor predictor of OSAHS; however, only 6% of patients with OSAHS do not report snoring and its absence makes OSAHS unlikely.[37] Corroboration with bedpartners is important as approximately 75% of patients who deny snoring are found to snore during objective measurement.[38]

Witnessed apneas are observed by up to 75% of bedpartners and are the second most common nocturnal symptom reported in OSAHS.[30,39] Occasional apneas are normal and do not cause symptoms; however, as the frequency of apneas increases, a certain threshold may be exceeded which results in symptomatic disease. This threshold is variable and unique to each patient such that some patients with a low Respiratory Disturbance Index (RDI) may be profoundly symptomatic while others with frequent respiratory events present with relatively few complaints.[40] Particularly in OSAHS of milder severity, the apneic episodes are usually associated with maintenance of respiratory movements and are terminated by loud snorts, gasps, moans, or other vocalizations and sometimes with brief awakenings and body movements. In more severe disease, cyanosis can occur along with the cessation of respiratory movement during the apnea which will often cause considerable distress to the bedpartner. Body movements at the time of arousals in severe OSAHS can be frequent and sometimes violent. Patients themselves are rarely aware of the apneas, vocalizations, frequent arousals, movements, or brief awakenings, although the elderly are particularly sensitive to the frequent nocturnal awakenings and will report insomnia and unrefreshing sleep.[1]

Nocturnal dyspnea, sometimes described by patients as a sensation of choking or suffocating, has been observed in 18–31% of patients with OSAHS.[35,41,42] These episodes typically occur with arousal, are associated with feelings of panic and anxiety, and generally subside within a few seconds. During apneas or hypopneas, greater negative intrathoracic pressures are generated as patients increase their inspiratory efforts to overcome the upper airway obstruction. This increases venous return to the heart and thus elevates pulmonary capillary wedge pressure which produces the sensation of dyspnea.[43,44] Other important causes of paroxysmal nocturnal dyspnea include left heart failure, nocturnal asthma, acute laryngeal stridor, or Cheyne–Stokes respirations; however, these episodes tend to be longer in duration and may also occur during the daytime.[39] Further investigation may be warranted to differentiate OSAHS from these other entities although they may also coexist.

Other common symptoms of OSAHS include drooling in about one-third and dry mouth in up to three-quarters of patients.[35] In one study of 668 patients with suspected SDB, dry mouth was observed in 31.4% of patients with confirmed OSA as compared to 16.4% in primary snorers and 3.2% in normal subjects.[45] Furthermore, there was a linear increase in prevalence of dry mouth as the severity of OSA increased. Sleep bruxism is also a common finding, occurring in 4.4% of the general population, with OSAHS patients being at higher risk (odds ratio 1.8) of reporting sleep bruxism.[46] In a small study of 21 patients, 54% of those with mild OSA and 40% of those with moderate disease were diagnosed with bruxism which was not observed to be directly associated with respiratory events but rather seemed to be related to sleep disruption and arousal.[47–49]

In addition to the common complaint of restless sleep and frequent awakenings, up to half of patients with OSAHS report nocturnal sweating that typically occurs in the neck and upper chest area.[41,42] This symptom is likely due to the increased work of breathing and respiratory effort in the setting of repetitive airway obstruction,[50] but may also be a manifestation of the autonomic instability observed in OSAHS. Similar to dyspnea, nocturnal diaphoresis is a highly non-specific symptom with a broad differential diagnosis, including perimenopausal state, thyroid disease, tuberculosis, lymphoma, and myriad other co-morbid conditions that warrant further investigation.[51]

Gastroesophageal reflux (GER) also occurs with greater frequency in patients with OSAHS with prevalence rates of 64–73%, but appears to be unrelated to the severity of SDB.[52,53] As GER and OSA share several risk factors, the exact relationship between the two entities has been difficult to characterize. However, it has been postulated that OSAHS may contribute to GER via the following mechanism: upper airway obstruction results in increased intraabdominal pressure combined with more negative intrathoracic pressure that produces an increased transdiaphragmatic pressure gradient, thereby promoting reflux of gastric contents into esophagus.[50] In a long-term study of 331 patients, treatment of OSAHS with nasal positive pressure resulted in a 48% decrease in the frequency of nocturnal GER symptoms with higher pressures being associated with greater improvement, suggesting that OSAHS may indeed be a causal factor in nocturnal gastroesophageal reflux.[54]

Nocturia has also been observed with increased frequency in OSAHS with a reported 28% of patients experiencing four to seven episodes nightly.[55] The presence and frequency of nocturia have been shown to be related to the severity of OSA,[55,56] and the proposed physiologic mechanisms include increased atrial natriuretic peptide secretion with a corresponding increase in total urine output,[57] or an increase in intraabdominal pressure. One study of 80 patients showed that although a majority of nocturnal awakenings were actually due to sleep-related phenomena (78.3% of 121 total awakenings), patients voluntarily urinated with each arousal and were only able to identify the correct source of the awakenings in five instances (4.9%).[58] These data show that patients mistakenly attributed their arousals to need to urinate when they were in fact due to SDB, suggesting that patient misperception may be a contributing factor to the increased frequency of reported nocturia in OSAHS.

3.2 DAYTIME SYMPTOMS

While the nocturnal symptoms of OSAHS are characteristic and tend to be more specific for the disease, the common daytime symptoms are less specific as they can result from abnormal sleep of any cause. Excessive daytime sleepiness (EDS) is the most common daytime complaint in patients with OSAHS;[30,35,41] however, as 30–50% of the general population also report moderate to severe sleepiness,[8,59] this symptom alone is a poor predictor of OSAHS. EDS is caused by sleep fragmentation leading to frequent arousals and insufficient sleep;[40] it is manifested by the inappropriate urge to sleep, particularly during relaxing, sedentary activities (i.e. watching television, reading). As it worsens, the inability to control sleepiness can result in dozing during meetings, active conversations, and at mealtimes. When severe, EDS can be a cause of motor vehicle and machinery accidents, poor school or job performance, and relationship discord.

Establishing the presence and severity of inappropriate daytime sleepiness can be challenging. EDS is often insidious, may be subtle, and is often confused with fatigue or lethargy. Patients themselves often have a poor perception of EDS severity and underestimate their level of impairment.[60] Co-morbidities such as chronic insomnia, depression, fibromyalgia, and other organic diseases, as well as medication use or substance abuse, may contribute to the symptom of inappropriate sleepiness. The clinician should focus on the urge to sleep in passive situations, since both physical and mental activity can mask underlying EDS.

The Epworth Sleepiness Scale (ESS) is a simple, self-administered questionnaire that is a quick and inexpensive tool with a high test–retest reliability,[2] and is thus a practical means of measuring the general level of daytime sleepiness. Patients are asked to assess their probability of falling asleep in eight situations commonly encountered in daily life, and a numeric score is tabulated. Higher Epworth scores, reflecting increased average sleep propensity, are able to distinguish patients with OSAHS from primary snorers and appear to correlate with severity of OSA as well as sleep latency measured objectively during multiple sleep latency testing.[35,61,62] The major disadvantage of the ESS is its reliance on subjective reporting by patients who may underestimate or intentionally underreport severity of daytime sleepiness.[39]

Morning or nocturnal headaches are reported in about half of patients with OSAHS, are typically dull and generalized, last 1 to 2 hours, and may require analgesics.[1,35] However, this symptom is non-specific as morning headaches occur in 5–8% of the general population and have been associated with many other entities, including other sleep disorders as well as depression, anxiety, and various medical conditions.[63,64] These headaches have been attributed to nocturnal episodes of oxygen desaturation, hypercapnia, cerebral vasodilatation with resultant increases in intracranial pressure, and impaired sleep quality with corresponding polysomnographic evidence of decreased total sleep time, sleep efficiency, and amount of REM sleep.[65,66] It has been observed that treatment of the underlying OSAHS results in disappearance of the morning headaches.

Neurocognitive impairment has also been observed in patients with OSAHS, although there is no practical method of quantifying such deficits in this setting. The processes most affected in OSAHS appear to be vigilance, executive functioning, and motor coordination.[67] Decreased vigilance is a result of sleep fragmentation, which can also lead to diminished concentration and memory (short term and long term). However, psychomotor impairment appears to be largely related to hypoxemia with more severe OSAHS potentially resulting in irreversible anoxic brain damage.[68–70] This hypothesis is supported by the observation that psychomotor deficits in patients with severe OSAHS are only partially reversible with treatment of the underlying sleep disorder with continuous positive airway pressure.[68,69]

Patients with OSAHS have a tendency to report decrements in their quality of life and often experience concomitant mood and personality changes. Depression is the most common mood symptom, and daytime sleepiness has been identified as a reliable predictor.[63,71] Other behavioral manifestations of OSAHS include anxiety, irritability, aggression, and emotional lability.[72] Treatment with continuous positive airway pressure has been shown to ameliorate symptoms of depression and thereby improve quality of life in some patients.[73,74] Sexual dysfunction, manifested primarily as erectile dysfunction and decreased libido, is also associated with OSAHS, and appears to be fully reversible with treatment of the underlying sleep disorder[75,76] (Table 2.1).

3.3 CLINICAL SIGNS

Physical examination of the patient with suspected OSAHS and UARS can reveal characteristic findings suggestive of

Table 2.1 Sleep and wake-related symptoms of OSAHS

Nocturnal symptoms	Daytime symptoms
Snoring	Excessive daytime sleepiness
Witnessed apneas	Morning headaches
Dyspnea (choking/gasping)	Neurocognitive impairment:
Drooling	vigilance (secondary impact on concentration and memory)
Dry mouth	executive functioning
Bruxism	motor coordination
Restless sleep/frequent arousals	Diminished quality of life
Gastroesophageal reflux	Mood and personality changes:
Nocturia	Depression
	Anxiety
	Irritability
	Sexual dysfunction:
	decreased libido
	impotence
	abnormal menses

upper airway obstruction and associated SDB. Blood pressure should always be recorded as both hypertension and hypotension have been found in patients with SDB. Obesity has also been frequently associated with OSAHS,[8,37] particularly in women, and measurement of height and weight followed by calculation of Body Mass Index (kg/m^2) to define and quantify obesity are important components of the physical examination. Grunstein and colleagues demonstrated that a BMI of at least $25 kg/m^2$ was associated with a 93% sensitivity and 74% specificity for OSAHS.[77] However, 18 to 40% of affected patients are less than 20% above ideal body weight,[35] and patients with UARS are typically non-obese.

Increased neck circumference has consistently been shown to be a more reliable clinical predictor of OSAHS and has been shown in one study to correlate with severity of disease.[30,78,79] Katz and colleagues showed that patients with OSAHS had an increased mean neck circumference (measured at the superior aspect of the cricothyroid membrane with the patient upright) of 43.7 cm ± 4.5 cm versus 39.6 cm ± 4.5 cm in control subjects ($P = 0.0001$).[80] Likewise, Kushida et al. found that neck circumference of 40 cm was associated with a sensitivity of 61% and specificity of 93% for OSAHS.[81] Thus, neck circumference should be routinely measured during physical examination, and if greater than 40 cm, underlying OSAHS must be considered and further investigated.

Examination of the upper airway is an essential component of the clinical evaluation of all patients with suspected SDB in order to identify potential areas of airway narrowing as well as to guide future therapies. The evaluation for both OSAHS and UARS is the same, and patients should be examined in both the upright and supine positions to optimize the detection of those anatomic features that predispose to SDB. The hallmark of both OSAHS and UARS is a crowded upper airway which is manifested by various characteristic morphologic abnormalities of the craniofacial, pharyngeal, dental, and nasal anatomy. Thorough inspection of these particular components of the upper airway, preferably with the aid of direct laryngoscopy, is essential in the evaluation of patients with suspected SDB.

The craniofacial abnormalities most commonly associated with airway narrowing and SDB are retrognathia[82] and high arched palate. Retrognathia, also known as mandibular retroposition, is a result of delayed growth of the mandible, maxilla, or both and is associated with posterior displacement of the tongue base.[81] Thus, the finding of retrognathia signifies a narrowed upper airway, particularly in the region of the retroglossal space. Patients with SDB are also commonly found to have high arched hard palates due to the early, forced expansion of the lateral palatine processes over the posteriorly displaced tongue prior to midline fusion. The significance of retrognathia and high arched palate in SDB was supported by Kushida et al., who described four craniofacial parameters indicative of airway narrowing: maxillary intermolar distance, mandibular intermolar distance, palatal height, and dental overjet (a sign of mandibular insufficiency).[81] Palpation of the temporomandibular joint with mouth opening may reveal varying degrees of dislocation (TMJ 'click') which confers greater risk of airway collapse in the supine patient due to posterior displacement during sleep.[50]

Examination of the pharyngeal structures may reveal further evidence of airway restriction and underlying SDB. These findings include macroglossia (often associated with lateral lingual scalloping by adjacent teeth), erythema and edema of the uvula due to snoring, and redundant lateral wall soft tissue with tonsillar pillar hypertrophy, and tonsillar enlargement (graded with 0–4 scale) which is a particularly significant cause of obstruction in children.[50] The degree of oropharyngeal crowding can be further characterized by the Friedman tongue position, formerly called the modified Mallampati score, which incorporates visual assessment of size, length, and height of the soft palate and uvula and is similar to the Friedman Tongue Index (further described in other sections of this text). These structures represent the anterior limit of the upper airway, and a low-lying, elongated, or enlarged soft palate/uvula decreases airway caliber and increases susceptibility to obstruction. This scoring system was initially developed to assess difficulty of airway intubation; a numeric grade from 1 to 4 is assigned depending on the relative size and positions of the soft palate, tip of the uvula, tongue, and tonsillar pillars. The Friedman tongue position has been shown to be an independent predictor of both the presence and severity of OSA with an average two-fold increase in odds of having OSA for every one point increase in the Friedman tongue position.[83] However, the gold standard in pharyngeal evaluation remains direct laryngoscopy as it is the most reliable means of identifying actual static and dynamic restriction of the retroglossal or retropalatal space, which are common sites of obstruction during sleep.

Abnormalities of dentition are often observed in patients with SDB and are typically reflective of maxillomandibular

Table 2.2 Common physical findings in patients with OSAHS
Craniofacial
Retrognathia High arched palate Temporomandibular dislocation
Pharyngeal
Macroglossia Erythema/edema of uvula Elongated, low-lying soft palate Tonsillar pillar hypertrophy Tonsillar enlargement Retropalatal, retroglossal space restriction
Dental
Overjet Malocclusion Bruxism Orthodontia
Nasal
Asymmetric, small nares Inspiratory collapse of alae and internal valves Septal deviation Inferior turbinate hypertrophy

deficiencies and airway crowding. Patients with these physical features will frequently report previous wisdom teeth extraction and/or childhood orthodontia. Dental overjet is a common sign of underlying mandibular retroposition and refers to the forward extrusion of the upper incisors beyond the lower incisors by more than 2.2 mm. Findings of dental malocclusion and overlapping teeth indicate a restricted oral cavity that is prone to collapse. Because OSAHS has been associated with bruxism, evidence of teeth grinding should also be noted.

Examination of the nose is the last important component of the upper airway evaluation. Although nasal obstruction is rarely the sole cause of SDB, it appears to occur with higher frequency in OSAHS[84] and may contribute significantly to increased upper airway resistance and the development of UARS.[85] Furthermore nasal obstruction in children may cause chronic mouth breathing and secondarily result in abnormal craniofacial development. Inspection of the nose should note the size and symmetry of nares, collapsibility of internal/external valves and nasal alae with inspiration, evidence of septal deviation or prior nasal trauma, and hypertrophy of the inferior nasal turbinates.[50] Identifying nasal obstruction can be particularly important in patients with SBD who have difficulty tolerating nasal continuous positive airway pressure (CPAP) therapy as treatments such as septoplasty and turbinate reduction can decrease nasal resistance with resultant improvements in CPAP compliance and comfort (Table 2.2).

4 CLINICAL PRESENTATION OF UARS

UARS was first described in children in 1982,[86] although it was later observed that a population of adults with chronic daytime sleepiness but without typical polysomnographic evidence of OSA (apneas, hypopneas, or oxygen desaturations) exhibited increased inspiratory effort during sleep detected by esophageal manometry that resulted in transient, repetitive alpha EEG arousals.[18] Although these increases in upper airway resistance were not sufficient to cause detectable airflow abnormalities or oxyhemoglobin desaturation on routine sleep testing, the required increase in inspiratory work to overcome the elevated resistive load appeared to lead to recurrent arousals and sleep fragmentation and daytime hypersomnolence.

As such, there is considerable overlap between the symptoms of OSAHS and UARS, with excessive daytime sleepiness, snoring, and restless sleep being frequent complaints in UARS. However, key differences do exist, and recent data suggest that chronic insomnia is much more common in UARS than in OSAHS.[50] Many adult UARS patients report frequent nocturnal awakenings with an inability to fall back asleep (sleep maintenance insomnia) as well as difficulties with sleep initiation (sleep-onset insomnia). Chronic insomnia in UARS has been attributed to cognitive-behavioral conditioning resulting from frequent sleep disruptions.[87] Parasomnias are more prevalent in young patients with UARS, with sleepwalking with or without night terrors and associated confusional arousal being most common.[88] Treatment of UARS typically results in resolution of the parasomnias.

While daytime symptoms are also similar to those seen in OSAHS, adult patients with UARS are more likely to complain of daytime fatigue rather than sleepiness.[50] Cold hands and feet are described in half of UARS patients, and about a quarter (typically teenagers and young adults) will report symptoms of dizziness or orthostatic hypotension, with lightheadedness upon rapid change of positions.[89] This orthostatic intolerance may be related to the finding that roughly a fifth of UARS patients exhibit resting hypotension (systolic blood pressure < 105 mm Hg, diastolic < 65 mm Hg) as compared to OSAHS,[90] which is typically associated with hypertension.[91–93]

Lastly, Gold and colleagues observed that symptoms closely resembling those found in the functional somatic syndromes were more often reported by patients with UARS than by those with OSAHS.[17] Complaints such as headache, sleep-onset insomnia, and irritable bowel syndrome were more prevalent in UARS and decreased progressively as severity of SDB increased. Because of the frequency of these and other related non-specific somatic complaints such as fainting and myalgias, it is not uncommon for UARS to be misinterpreted as one of the many functional somatic syndromes, including chronic fatigue syndrome, fibromyalgia, irritable bowel syndrome, temporomandibular joint syndrome, or migraine/tension headache syndrome (Table 2.3).

Table 2.3 Major clinical features differentiating OSAHS and UARS

OSAHS	UARS
Men > women	Men = women
Obese	Non-obese
Hypertension	Hypotension
Hypersomnia, excessive daytime sleepiness	Insomnia, fatigue
Rare somatic symptoms	Somatic symptoms

5 SLEEP-DISORDERED BREATHING IN WOMEN

In the early descriptions of OSAHS, the vast majority of data came from studies performed on male subjects, and women were characterized as the 'forgotten gender.'[94] Preliminary observations of SDB in women appeared to suggest a strong correlation with obesity, resulting in a clear bias against the recognition of this entity in non-obese women (BMI $< 30\,kg/m^2$), even in the presence of clinical and polysomnographic findings of OSAHS.[94,95] As a result, women experienced longer durations of symptoms and significant delays in appropriate referral and diagnosis compared to age-matched men.[94,96]

It is now recognized that women often present with different symptoms of SDB, report 'typical' symptoms less frequently, and may underestimate symptom severity as compared to men, which may be additional factors contributing to disease underrecognition in this population.[97] Symptoms more commonly observed in women include non-specific somatic complaints, such as insomnia, fatigue, myalgias, and morning headache.[17,98] Amenorrhea and dysmenorrhea have been reported in 43% of women with SDB.[94] Depression, anxiety, and social isolation occur with higher frequency in women as compared to men with SDB.[94,99] While women with OSAHS do tend to be more obese than men with similar disease severity, those with UARS are typically non-obese, younger, and have fewer witnessed apneas.[18,100]

6 SUMMARY

Although the understanding of sleep-disordered breathing continues to evolve, it is clear that both OSAHS and UARS are important causes of morbidity and mortality in a significant portion of the population. Snoring, excessive daytime sleepiness, and witnessed apneas are the hallmark symptoms of OSAHS, and although there is considerable overlap with UARS, there are several notable differences as well. Increased neck circumference and craniofacial characteristics suggesting airway crowding are also highly associated with SDB. Women tend to present with less typical complaints and may require a higher index of suspicion for diagnosis. Although it remains debatable whether OSAHS and UARS are distinct entities or represent ends of a continuum, the more clinically relevant task is the recognition that symptomatic patients without characteristic evidence of OSA may still have UARS, and thus warrant further evaluation with polysomnography.

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CHAPTER 3

Airway evaluation in obstructive sleep apnea

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1 METHODS OF AIRWAY EVALUATION

As the interest in sleep-disordered breathing (SDB) has increased, various attempts have been made to assess upper airway anatomy in patients with this relatively frequent disorder. From the very beginning, researchers and clinicians used a multitude of different techniques not only to reveal potential differences in upper airway anatomy to better understand the origin and the pathophysiology of the disease but also to improve patient management and treatment success. While the value of thorough clinical assessment remains indubitable, the value of the Mueller maneuver has been questioned from the beginning. Static radiologic imaging techniques such as x-ray cephalometry, computed tomography (CT) scanning and magnetic resonance imaging (MRI) have been used mostly to detect differences in airway anatomy. Dynamic scanning protocols (e.g. ultrafast CT or cine MRI) and multiple pressure recordings have been used to gain insights into the mechanism and level of airway obstruction. Upper airway endoscopy has been inaugurated during sleep and sedated sleep to directly visualize airway obstruction, and the assessment of critical closing pressures has been used to quantify upper airway collapsibility.

2 CLINICAL EXAMINATION AND CLINICAL SCORES

A clinical examination including an endoscopy of the upper airway during wakefulness still constitutes the basis of every airway evaluation in snorers and obstructive sleep apnea (OSA) patients. Anatomic and static clinical findings were the first parameters to be evaluated in order to improve treatment success. The impact of enlarged palatine tonsils became evident in the surgical experiences with children. If performed simultaneously, tonsillectomy was described by most authors as a positive predictive factor for a successful uvulopalatopharyngoplasty (UPPP). All the other anatomic parameters such as the size of the uvula, the existence of longitudinal pharyngeal folds and so forth did not show any relationship to the success rate of UPPP if evaluated separately. In contrast to the significant influence of enlarged tonsils in palatal obstruction, equivalent clinical finding for tongue base obstructions could not be detected. Woodson and Wooten only found hints that the oropharynx was normal in cases with retrolingual obstruction.[1]

Aware of this dilemma, Friedman et al. developed a clinical four degree staging system incorporating the tonsil size, the position of the soft palate, the tongue size, and the Body Mass Index (BMI).[2] This anatomic staging system predicted the success rate better than OSA severity did.[3] One may argue that the staging system merely reflects the clinical examination of an experienced sleep physician; nevertheless, such a system may be particularly helpful for less experienced observers.

Whether there are further predictive anatomic parameters for other surgical strategies has not been evaluated to date. The subjectivity of the assessment and the variability of the nomenclature of the clinical findings are significant limitations in this context.

3 THE MUELLER MANEUVER

Snoring as well as apneas can be simulated by most people and a direct effect of the Mueller Maneuver may be seen during wakefulness. Thus, snoring simulation and the effects of the Mueller Maneuver have been used in upper airway evaluation before surgical intervention in patients to predict surgical outcome and to improve patient selection. Nevertheless, the value of this relatively simple examination has been questioned repeatedly in the past.

3.1 TECHNIQUES OF THE MANEUVER

In order to be able to compare results between different investigators and patients as well as before and after an

intervention, the Maneuver should be performed and documented in a standardized fashion. Due to its simplicity the classification according to Sher has been widely used to describe the finding obtained during the Maneuver.[3] In this classification, four degrees of airway obstruction at the different levels are defined, ranging from minimal to complete occlusion. Furthermore, any visible obstruction linked to the epiglottis is described. The reproducibility and inter-rater reliability of the results remain problematic. Taking all the available data into account, the reliability of the Mueller Maneuver remains highly questionable and the evaluation of the Maneuver seems highly subjective and hard to reproduce.

3.2 PREDICTING AIRWAY OBSTRUCTION DURING SLEEP AND SURGICAL SUCCESS

There is some evidence that the sites of obstruction detected with the Mueller Maneuver do not reliably reflect the sites of obstruction during sleep. This could be demonstrated through a comparison with videoendoscopy, multi-channel pressure recordings, and dynamic MRI during sleep. Table 3.1 shows the different sites of airway obstruction detected with the different methods of airway evaluation according to selected examples from the literature.

The impact of body position on the significance of the Mueller Maneuver remains unclear. During the Mueller Maneuver, healthy subjects may produce extreme negative pressures of −80 mbar without any signs of pharyngeal collapse.[4] This clearly demonstrates the significant differences in upper airway collapsibility during wakefulness and sleep. All the data given do not support the idea that the results obtained by the Mueller Maneuver may be transferred to natural sleep.

Various research groups were not able to better predict the success rates obtained with UPPP when using the Mueller Maneuver. Some authors considered an additional retrolingual collapse during the Mueller Maneuver as an exclusion criterion for a UPPP or performed a

Table 3.1 Distribution of the sites of obstruction detected by different methods of airway evaluation (selected literature)

Method	Author	Diagnosis	*n*	Palatal	Retrolingual	Combined	Epiglottis	No result
Mueller Maneuver	Petri et al.	OSAS	30	8	0	22	n. d.	0/30
	Sher et al.	OSAS	171	101	56	14	2/101	0/171
	Skatvedt	SBAS	20	4	0	4	n. d.	0/20
	Sum (mean value %)		*221*	*113 (51%)*	*56 (25%)*	*40 (18%)*	*2 (1%)*	*0/221 (0%)*
Endoscopy during sleep	Launois et al.	OSAS	18	11	2	5	n. d.	8/26
	Woodson and Wooten	OSAS	11	5	6	n. d.	n. d.	n. d.
	Sum (mean value %)		*29*	*16 (55%)*	*8 (28%)*	*5 (17%)*		*8/26 (31%)*
Endoscopy under sedation	Croft and Pringle	SBAS	56	25	n.d.	31	0	15/71
	Pringle and Croft	SBAS	70	33	9	28	0	20/90
	Camilleri et al.	SBAS	25	17	0	8	0	2/27
	Hessel et al.	SBAS	340	111	8	221	n.d.	n.d.
	Steinhart et al.	SBAS	306	139	23	134	10	16/322
	Den Herder et al.	SBAS	127	65	15	47	n.d.	n.d.
	Quinn et al.	Snoring	50	35	4	5	6	4/54
	Marais	Snoring	168	101	52	13	2	37/205
	El Badawey et al.	Snoring	46	8	2	36	n.d.	5/55
	Abdullah et al.	Snoring	30	12	0	18	0	n.d.
	Abdullah et al.	OSAS	89	12	4	71	2	4/93
	Sum (mean value %)		*1307*	*558 (43%)*	*117 (9%)*	*612 (47%)*	*20 (1.5%)*	*103/917 (11%)*
Pressure recordings during sleep	Hudgel	OSAS	9	4	5	0	n.d.	0/9
	Chaban et al.	OSAS	10	5	5	0	n.d.	n. d.
	Metes et al.	SBAS	51	30	7	n.d.	n.d.	13/51
	Tvinnereim and Miljeteig	OSAS	12	6	2	n.d.	4 (?)	0/12
			20	2	5	10	n. d.	0/20
	Skatvedt	SBAS	20	5	4	9	2 (?)	0/20
	Katsantonis et al.	OSAS	11	8	3	n.d.	n.d.	n.d.
	Woodson and Wooten	OSAS						
	Sum (mean value %)		*133*	*60 (47%)*	*31 (23%)*	*19 (14%)*	*6 (4.5%?)*	*13/112 (12%)*

SBAS, patients with primary snoring or OSAS; OSAS, only patients with OSAS; Palatal, nasopharynx, tonsils, soft palate and/or lateral pharyngeal wall; Retrolingual, tongue base and/or hypopharynx; Epiglottis, exclusively epiglottis; No result, either the method was not tolerated or the result was not utilizable, n.d.: not detected.

partial resection of the epiglottis in UPPP failure patients with laryngeal obstruction during the Mueller Maneuver by partial resection of the epiglottis.

3.3 SIGNIFICANCE OF THE MUELLER MANEUVER

The Mueller Maneuver is a safe and simple examination that does not exert relevant strain on the patient. The reliability of the Mueller Maneuver is insufficient and the results of the Mueller Maneuver cannot be transferred to natural sleep. A hypopharyngeal collapse may indicate the exclusion of patients from UPPP. Altogether, the Mueller Maneuver does not facilitate patient selection for the varying surgical interventions used in OSA patients.

4 X-RAY CEPHALOMETRY

Over the years, lateral x-ray cephalometry has become one of the standard diagnostic tools in patients with SDB, especially with regard to the evaluation of the skeletal craniofacial morphology. Not specifically developed for the fields of SDB, imaging techniques and standards for data analysis have been incorporated from the field of maxillofacial surgery, where it has already been used for decades.

4.1 PROVIDING INSIGHTS INTO THE PATHOPHYSIOLOGY OF SDB

Extensive literature is available comparing upper airway anatomy and dentofacial structures using x-ray cephalometry between OSA patients and healthy controls. In siblings a significantly longer distance from the hyoid bone to the mandibular plane has been documented in those affected by SDB.[5] Further differences were described by different working gropus. The concrete results are often difficult to compare, as the authors not only use different landmarks and parameters but also sometimes rather complex calculated indices and ratios to describe the differences they found. Therefore, the following findings in OSA patients can only be a selection: longer soft palates, reduced minimum palatal airway widths, increased thickness of the soft palate, differences in calculated craniofacial scores, increased pharyngeal lengths, retroposition of the mandible or the maxilla, micrognathia, increased mid-facial heights, and differences in hyoid bone position. In general, the differences are more pronounced in non-obese patients, suggesting that craniofacial changes play a dominant role in this subgroup. Furthermore, substantial differences in maxillofacial appearance of different ethnic groups need to be taken into account.

Various authors could demonstrate that the aberrations in craniofacial morphology they found in OSA patients were more pronounced in patients with severe OSA. Dempsey et al. demonstrated that in non-obese patients and in patients with narrow upper airway dimensions, four cephalometric dimensions were the dominant predictors of Apnea/Hypopnea Index (AHI) level, accounting for 50% of the variance.[6] Rose et al. questioned the diagnostic relevance of x-ray cephalometry for OSA, as they found no direct correlation between skeletal cephalometric findings and OSA severity; nevertheless, they also reported a correlation with hyoid bone position.[7]

4.2 X-RAY CEPHALOMETRY AND THERAPEUTIC INTERVENTIONS

One of the dominant indications for performing x-ray cephalometry has been treatment with oral appliances. Especially with regard to the evaluation of potential predictive parameters for treatment success and dental side effects, x-ray cephalometry has been the standard diagnostic tool. As early as 1995, Mayer and Meier-Ewert, two of the fathers of treatment with oral appliances in Europe, looked for cephalometric predictors of treatment success[8] and reported that specific cephalometric variables were indeed predictive for the therapeutic effect. Other authors have confirmed the existence of predictive cephalometric parameters, especially in relation to hyoid bone position and oropharyngeal airway dimension. Nevertheless, the problems related to different nomenclature and selection of airway parameters described above remain.

X-ray cephalometry has also been evaluated with regard to potential predictive parameters for postoperative results of UPPP alone or in combination with other approaches. To date there is no convincing evidence that skeletal measurements obtained with x-ray cephalometry could predict the outcome of UPPP. Nevertheless, lateral x-ray cephalometry is the standard tool in the preoperative evaluation of the craniofacial skeletal anatomy before maxillomandibular advancement surgery. It can be regarded as a mandatory procedure and its value is not questioned.[9]

4.3 X-RAY CEPHALOMETRY IN PATIENT MANAGEMENT

X-ray cephalometry has provided substantial insights into the pathophysiology of OSA, demonstrating significant craniofacial characteristics associated with this disease. Although the results are not easy to compare, specific cephalometric characteristics have been repeatedly mentioned as a risk factor for OSA and correlate with the