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Obstructive sleep apnea in older adults

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Abstract:

This review presents basic information on the epidemiology, classification, diagnostic criteria, and risk factors of obstructive sleep apnea (OSA), and then discusses its most important aspects in older adults, taking into account differences in clinical presentation and therapeutic approach compared with younger patients. Potential difficulties in establishing the correct diagnosis are highlighted, emphasizing the need for diagnostic vigilance. A careful medical history that includes characteristic symptoms plays a key role. The diagnosis is based on polysomnography, although the greater availability of polygraphy justifies its use in many cases. The goals of OSA treatment in older adults are to reduce night- and daytime symptoms and to decrease the risk of serious cardiovascular and cerebrovascular events. Treatment methods should be individualized according to symptoms, comorbidities, the older patient's daily functioning, and the ability to cooperate. Continuous positive airway pressure (CPAP) remains the treatment of choice for moderate and severe OSA, although other therapeutic approaches may also be used in older patients.

Introduction

Population aging has become a reality in both Poland and Europe and represents one of the major demographic challenges of the 21st century. The number of people aged over 65 years worldwide is expected to more than double over the next 30 years. In Poland, this age group already accounts for more than 20% of the population, with important economic and healthcare consequences [1, 2, 3]. Sleep-disordered breathing is therefore an increasingly relevant clinical issue. Its prevalence rises with age, but its clinical presentation in older adults differs from that observed in younger people. The pathophysiology of sleep-disordered breathing after the age of 65 years, its clinical symptoms, health consequences, and therapeutic approach all differ to some extent from those described in middle-aged patients, and these differences deserve to be emphasized.

Classification and epidemiology

The classification of sleep-related breathing disorders proposed by the American Academy of Sleep Medicine is shown in Table 1 [4]. The two most common forms are primary (habitual) snoring and obstructive sleep apnea (OSA). Depending on the study, primary snoring without OSA affects up to 20-45% of the general population, and its prevalence increases with age. After the age of 60 years, it has been reported in as many as 60% of men and 40% of women. Although primary snoring does not initially cause major health consequences, snoring itself may predispose to the development of OSA; therefore, its presence should be considered an indication for further sleep assessment and causal treatment. Snoring without OSA is not associated with clinically relevant oxygen desaturation or major disruption of sleep architecture, but long-standing habitual vibration of the soft-palate tissues may promote local inflammation and peripheral neuropathy, thereby facilitating the development of OSA [5]. A long history of primary snoring, which is common in older adults, may therefore contribute to the increasing occurrence of sleep apneas that often remain unrecognized and untreated.

Table 1. Classification of sleep-related breathing disorders according to the American Academy of Sleep Medicine [4].

1. Obstructive sleep apnea disorders a) adults b) children
2. Central sleep apnea disorders a. with Cheyne-Stokes breathing b. due to a medical disorder without Cheyne-Stokes breathing (vascular, degenerative, post-traumatic, or neoplastic disorders of the central nervous system) c. due to high-altitude periodic breathing d. due to a medication or substance e. primary central sleep apnea f. primary central sleep apnea of infancy or prematurity g. treatment-emergent central sleep apnea
3. Sleep-related hypoventilation disorders a. obesity hypoventilation syndrome b. congenital central alveolar hypoventilation syndrome c. late-onset central hypoventilation with hypothalamic dysfunction d. idiopathic central alveolar hypoventilation e. due to a medication or substance f. due to a medical disorder (respiratory, pulmonary, or neuromuscular disease)
4. Sleep-related hypoxemia

5. Isolated symptoms and normal variants
   a. snoring
   b. catathrenia

After primary snoring, OSA is the most common sleep-related breathing disorder. The prevalence reported in the literature varies widely: European studies generally estimate it at 2-12%, whereas American studies report values as high as 7-24%. All authors emphasize that OSA becomes substantially more common with advancing age and may affect approximately 49-90% of older adults. This wide range reflects the diagnostic difficulties encountered in older populations: both the clinical picture and questionnaire results may be distorted by nonspecific symptoms related to multimorbidity, memory impairment, and reduced patient cooperation. In middle age, OSA is diagnosed two to three times more often in men than in women, whereas this sex difference becomes less pronounced in older age groups [3, 5, 6, 7, 8].

Definitions

Obstructive apnea is defined as a reduction in airflow of at least 90% lasting for at least 10 seconds, with preserved or increased inspiratory muscle effort.

Hypopnea is defined as a reduction in airflow of at least 30% lasting for at least 10 seconds, accompanied by a decrease in arterial oxygen saturation (SaO2) of at least 3% (or 4%) from the pre-event baseline or by a respiratory effort-related arousal. The number of apneas and hypopneas per hour of sleep is expressed as the apnea-hypopnea index (AHI).

A respiratory effort-related arousal (RERA) is identified when increasing respiratory effort or flow limitation that does not meet criteria for apnea or hypopnea persists for at least 10 seconds and results in an arousal, i.e., an abrupt shift in sleep toward a lighter stage or wakefulness. The number of apneas, hypopneas, and RERAs per hour of sleep is expressed as the respiratory disturbance index (RDI). Because RERAs are difficult to assess reliably and esophageal pressure monitoring is rarely used in routine practice, AHI remains the most commonly applied diagnostic index. When RERAs cannot be measured, AHI is often treated as equivalent to RDI for practical purposes [4, 6, 9].

Diagnostic criteria for OSA

According to the American Academy of Sleep Medicine (2014), OSA is diagnosed when the following criteria are met [4,6]:

A. One or more of the following is present:

- excessive daytime sleepiness, non-restorative sleep, daytime fatigue or insomnia;
- awakening with breath-holding, gasping or choking;
- habitual snoring and/or witnessed apneas reported by a bed partner;
- or hypertension, ischemic heart disease, congestive heart failure, atrial fibrillation, type 2 diabetes, mood or cognitive disorders, or a history of stroke.

B. Polysomnography or cardiorespiratory polygraphy demonstrates AHI >=5 events/h, predominantly obstructive apneas and hypopneas with preserved respiratory effort.

C. AHI >=15 events/h with obstructive apneas and hypopneas and preserved respiratory effort.

OSA is diagnosed when criteria A and B are both fulfilled, or when criterion C is fulfilled on its own. Severity of OSA is classified as mild (AHI >=5 and <=15), moderate (AHI >15 and <=30), and severe (AHI >30) [4,6].

Risk factors and pathophysiology of OSA

The most important risk factors for OSA include the following:

- Obesity and body-fat distribution (particularly central adiposity). These are major risk factors in middle-aged patients. In older adults, neck circumference is a less reliable risk marker because other anatomical factors become increasingly important. Notably, parapharyngeal fat deposition increases with age regardless of BMI in both women and men [10].
- Age. OSA becomes more common with aging, mainly because of anatomical and functional changes in the upper airway. Aging is associated with elongation of the pharyngeal structures and soft palate, a relatively larger anterior tubercle of the atlas in relation to airway width, increased parapharyngeal fat deposition, and reduced tone and responsiveness of the muscles that maintain pharyngeal patency. These changes increase the susceptibility of the upper airway to collapse during sleep [2,7]. Sleep is also accompanied by a reduction in respiratory drive and ventilatory responsiveness to carbon dioxide, which is particularly important in older adults with reduced muscle tone [8]. Reactivity of the genioglossus muscle to oxygen levels also deteriorates with age [7]. In addition, tooth loss and the use of hypnotic medications (sleeping pills), which further reduce muscle tone, may facilitate obstruction. Insomnia and altered sleep architecture can lead to overuse of hypnotics in older patients [2], while complete edentulism with removal of dentures at night may also predispose to airway obstruction [7]. The principal age-related mechanisms that increase upper-airway collapsibility and their main clinical consequences are summarized in Figure 1.

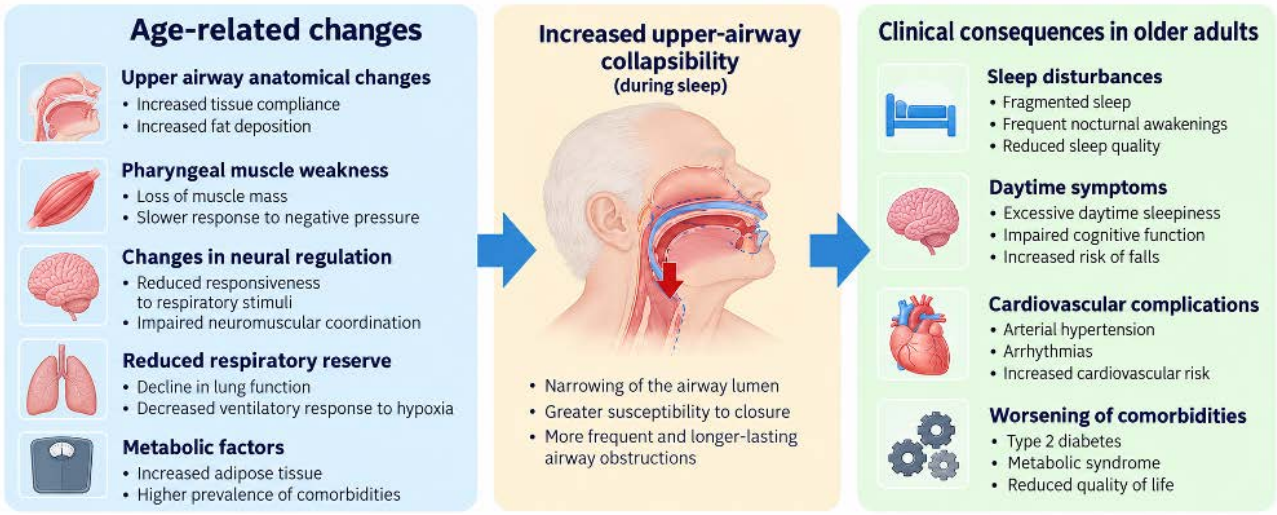


Figure 1. Age-related mechanisms increasing upper-airway collapsibility and their main clinical consequences in older adults [2, 3, 7, 8, 13, 14].

- Sex. Male predominance becomes less marked with age. During and after menopause, the protective effects of estrogen and progesterone diminish, altering fat distribution and respiratory-center responsiveness to hypoxemia and hypercapnia. OSA becomes more common in postmenopausal women independently of BMI, although disease severity tends to remain lower than in men of the same age [11]. A protective effect of hormone replacement therapy has been described [7].
- Smoking and excessive alcohol consumption. Both promote local inflammation, tissue remodeling, and reduced muscle tone, predisposing to snoring and pharyngeal collapse. Their cumulative effect may be particularly relevant in older adults [5]. Gastro-esophageal reflux has been described both as a consequence of OSA and as a potential contributor to pharyngeal tissue remodeling and upper-airway obstruction [6,12].
- Endocrine disorders, including hypothyroidism, acromegaly, and Cushing syndrome or Cushing disease, may contribute to OSA together with the factors listed above [5,6].

- Genetic and racial factors. Familial predisposition and ethnicity-related anatomical characteristics and susceptibility to obesity may influence OSA risk [5,6].

These factors reduce upper-airway width and increase the risk of pharyngeal collapse during sleep. Airway patency depends on the balance between two opposing forces: negative inspiratory pressure, which draws the soft tissues inward, and the activity of pharyngeal dilator muscles, which promotes airflow. Additional anatomical abnormalities - including mandibular hypoplasia or retrognathia, enlarged uvula, hypertrophy of adenoid or palatine or lingual tonsils, an elongated soft palate, deviated nasal septum, inferior turbinate hypertrophy, and nasal polyps - may further contribute to sleep-related obstruction. When the pharyngeal structures collapse during sleep, airflow ceases despite preserved activity of diaphragm, generating increasingly negative intrathoracic pressure and making reopening of the airway more difficult. Respiratory effort increases, while hypoxemia and hypercapnia develop, usually ending with an unconscious arousal, a loud snore, and an effective breath. Critical closing pressure of the upper airway has been reported to be approximately twice as high (i.e., less negative) in older than in younger adults, making obstruction easier regardless of body weight [2]. Compensatory hyperventilation after an apnea causes hypocapnia and transient suppression of respiratory drive, promoting another apnea and perpetuating the cycle throughout the night. Repeated arousals are responsible for sleep fragmentation and lead to sleep deprivation and both night - and daytime symptoms of OSA [2,8].

Clinical characteristics of OSA in older adults

In older people, the mechanisms described above have particular importance because aging itself is associated with multiple sleep disturbances. As a result, OSA may remain undiagnosed for a long time, or its manifestations may be incorrectly interpreted as a natural consequence of age, weakness, insomnia, or comorbid disease [3]. Normal sleep alternates between NREM and REM phases in four to six cycles per night, and preservation of this architecture is essential for normal function and well-being. With age, deep sleep decreases, arousals become more frequent, and lighter sleep occupies a greater proportion of the night. Sleep therefore becomes more fragile and susceptible to disruption. This altered sleep architecture and dysregulation of respiratory control increase the likelihood of recurrent upper-airway obstruction and its consequences. At the same time, fatigue, impaired concentration, nocturia, cognitive decline and behavioral changes are easily attributed to other age-related disorders, further complicating the diagnosis of OSA [3].

Compared with younger patients, older adults with OSA often report less pronounced daytime sleepiness and less frequent loud snoring [7,11]. Short daytime naps may not be perceived as sleepiness but rather as a consequence of weakness or a need for recovery [13]. Nocturia is also less specific for OSA in older adults because cardiovascular disease and prostatic disorders may independently cause this symptom [3]. Comorbid insomnia and sleep apnea (COMISA) is more common in older patients and should prompt sleep study [7].

Other characteristic manifestations include persistent fatigue, reduced muscle strength (sarcopenia), balance impairment, and falls, particularly at night. These features overlap with frailty and are often attributed to aging and multimorbidity, resulting in missed opportunities to investigate coexisting OSA. Despite the well-established association between age and OSA prevalence, diagnostic vigilance does not increase proportionally, and the percentage of undiagnosed cases rises with age. OSA may therefore remain hidden behind other geriatric complaints [7,13]. A relationship between OSA and frailty has been reported. Repeated hypoxic episodes promote chronic low-grade inflammation and oxidative stress, damaging the nervous and cardiovascular systems. This may accelerate dementia, coronary disease, arrhythmias, loss of muscle mass, and reduced physical performance and activity. A vicious cycle may develop in which hypoxia and organ injury promote frailty, while frailty further intensifies the anatomical and physiological changes that predispose to OSA [3,13,14].

As in other age groups, recurrent hypoxia and chronic sleep deprivation adversely affect cognition. Older adults with OSA may develop impaired recent memory and attention, reduced intellectual performance, and poorer

logical reasoning, with an increased risk of traffic accidents related to chronic fatigue. Long-standing recurrent cerebral hypoxia may contribute to progressive dementia, although age-related cardiovascular and neurological changes also play a role. These mechanisms may combine and accelerate loss of independence [7]. In cohorts of patients with dementia, mild OSA has been reported in approximately 70%; another study found AHI >=20 in 48% of patients, while moderate OSA was diagnosed in 49% of older adults with dementia and nocturnal agitation. Studies have also suggested an association between the apolipoprotein E (APOE) gene, a risk factor for Alzheimer disease, and OSA. Correlations between OSA severity and Alzheimer-related brain changes have been reported, but these studies included small groups of patients and require confirmation [2,3,15].

Emotional and behavioral disturbances may accompany cognitive decline in older adults with OSA. Some patients predominantly experience anxiety and depression, whereas others develop mood instability and greater propensity for aggressive behavior [6,8]. Social withdrawal and prolonged inactivity may follow, further reducing physical activity, muscle strength, and functional capacity, and thereby contributing to frailty and worsening OSA [3].

Cardiovascular disease is another major consequence of untreated OSA. Although these conditions are multifactorial, OSA increases the risk of a more severe course and potentially life-threatening exacerbations. Across age groups, OSA is associated with ischemic heart disease, arrhythmias, hypertension, heart failure, and stroke [6,9]. In older adults with AHI >=20, cerebral vascular events - including ischemic stroke and transient ischemic attack (TIA) - and mortality related to these events have been reported more frequently than in age-matched patients with mild OSA [2]. Interestingly, although OSA is a risk factor for coronary disease at all ages, some studies in patients older than 70 years have not demonstrated a higher rate of acute ischemic coronary events, myocardial infarction, or related death. One proposed explanation is a protective adaptation to chronic intermittent hypoxia, in which repeated hypoxic episodes stimulate coronary collateral formation. This phenomenon, however, does not appear to protect the cerebral circulation [3,13].

OSA may also contribute to endocrine and metabolic disturbances. In older adults, impaired glucose metabolism is particularly important, leading to insulin resistance, glucose intolerance, and diabetes. An AHI >=15 has been reported as an independent risk factor for fasting hyperglycemia and glucose intolerance, and type 2 diabetes develops more frequently and earlier in patients with OSA than in those with a normal AHI [6]. More recent evidence supports OSA as an independent contributor to insulin resistance rather than attributing this relationship solely to obesity [16]. In people over 60 years of age, shorter average sleep duration has also been associated with obesity, further reinforcing a bidirectional cycle between obesity and OSA [17]. Age-related endocrine changes and recurrent hypoxia may additionally contribute to frailty through slower metabolism, increased visceral fat deposition, and loss of muscle mass [3].

Because the clinical picture in older adults differs in several respects from that in younger patients, a careful medical history is particularly important. Interpreting reported or observed symptoms in the context of age may sometimes be more useful than screening questionnaires alone. Completion of the Epworth Sleepiness Scale may be difficult in patients with memory or cognitive impairment, and daytime sleepiness is less characteristic in this age group, limiting the sensitivity of the scale. The STOP-BANG questionnaire may be more useful, although it also fails to capture all aspects of OSA in older adults and should not be used by patients as a stand-alone diagnostic tool (Table 2). In addition to questionnaire findings, patients should be actively asked about disorders that may reflect repeated nocturnal apneas, such as permanent atrial fibrillation, nocturnal arrhythmias, and established heart failure. These findings should increase the index of suspicion for OSA [11].

Table 2. STOP-BANG questionnaire [7].

Item	Question / criterion	Meaning
S	Do you snore loudly?	Snoring
T	Do you often feel tired?	Tiredness
O	Has anyone observed you stop breathing during sleep?	Observed apneas
P	Do you have, or are you being treated for, high blood pressure?	Blood pressure
B	BMI >35 kg/m^2	Body mass index
A	Age >50 years	Age
N	Neck circumference >16 inches (40 cm)	Neck circumference
G	Male sex	Gender

When OSA is suspected on the basis of history, a screening questionnaire, and physical examination, the patient should be referred for a sleep study. Although polysomnography is less available than polygraphy, it is generally preferable in older patients because EEG recording permits differentiation of coexisting sleep disorders and laboratory supervision avoids the need for the patient to apply sensors independently. Nevertheless, limited access to polysomnography and adequate patient or caregiver ability to perform a home study may justify diagnosis based on polygraphy so that treatment is not delayed. Sleep studies are performed substantially less often in older adults with a justified suspicion of OSA than in younger patients, which contributes to underdiagnosis in clinical practice. Home polygraphy also has important limitations, particularly in patients with concomitant insomnia, complex cardiopulmonary disease, suspected central sleep-disordered breathing, or marked sleep fragmentation [7, 11].

Another important issue is whether OSA severity as defined by AHI accurately reflects the degree of hypoxemia and functional impairment in older patients. Severe OSA is more common in older adults, but they also spend significantly more time with oxygen saturation below 90% regardless of AHI. This does not always correlate with daytime symptoms but may translate into greater chronic tissue hypoxia. Longer apneas and hypopneas and a weaker ventilatory response to hypoxemia and hypercapnia have also been described. Polysomnography allows calculation of the sleep apnea-specific hypoxic burden (SASHB), which measures the depth and duration of oxygen desaturations caused specifically by breathing events during sleep and thus reflects cumulative hypoxic exposure. This metric may predict cardiovascular risk more accurately than AHI alone, which is particularly relevant in older patients [18]. Positional OSA is also substantially more frequent in older adults. In this phenotype, the supine AHI is at least twice the AHI recorded in other sleep positions. Positional OSA has been reported in up to 80-90% of older patients, possibly because aging increases the tendency of the pharyngeal soft tissues to collapse in the sagittal plane [7, 11, 19]. Finally, multimorbidity is common in this age group. In particular, overlap of OSA with chronic obstructive pulmonary disease (COPD) may produce marked nocturnal hypoxemia and substantially increase the long-term risk of chronic respiratory failure and pulmonary hypertension [6].

Treatment of OSA in older adults

The first goal of OSA treatment in older adults is to reduce night- and daytime symptoms, including arousals, sleep fragmentation, insomnia, chronic fatigue, cognitive decline, and progression of frailty. The second goal is to reduce the risk of serious cardiovascular and cerebrovascular events, including arrhythmias, uncontrolled blood pressure elevations, and stroke [11].

Given the complexity of OSA in older adults, which reflects multimorbidity and the consequences of aging, therapeutic decisions should move beyond a simple AHI-based model. Clinicians should consider how OSA affects daily functioning, the risk of falls, cognitive capacity, independence, manual dexterity, and overall quality of life

of a patient. A functional approach that identifies the dominant OSA phenotype in an individual older patient and defines the main treatment target is particularly useful in geriatric practice. Such personalized management better reflects real patient needs than classification based solely on conventional OSA severity.

The main treatment options include lifestyle modification, continuous positive airway pressure therapy (CPAP), oral appliances, positional therapy, and selected surgical procedures [6]. Lifestyle modification is recommended for all patients and includes reducing excess body weight or maintaining a healthy weight, avoiding alcohol and hypnotic or sedative medications, and smoking cessation. In older adults, these measures should be tailored to physical capacity and nutritional status. Regular exercise, walking, and appropriate rehabilitation help preserve mobility. In this age group, neck and visceral fat distribution may be more relevant to OSA risk than obesity alone. In patients with overweight or obesity, often accompanied by type 2 diabetes, treatment with GLP-1 receptor agonists (e.g. semaglutide and related drugs) or the dual GIP/GLP-1 receptor agonist (tirzepatide) can produce substantial weight loss and reductions in AHI of up to 51-59% [11, 20]. Some of these agents have got officially approved indications for weight management and may therefore contribute to OSA treatment. These effects are promising, especially for patients unable to tolerate CPAP, although evidence specifically in older populations remains limited [7]. Myofunctional therapy, consisting of exercises for the oral and pharyngeal muscles, may be a useful adjunct and can be incorporated into a daily routine [21]. Restoration of the regular sleep-wake cycle is also important and may reduce overuse of hypnotic medications [2].

Because positional OSA is common in older adults, positional therapy should be considered, particularly in patients who are not candidates for CPAP or decline it. The aim is to avoid supine sleep or to sleep in a semi-upright position. Traditional approaches include placing a rubber or tennis ball in the back of the upper part of the pajamas to discourage supine sleep. More modern options include smartphone applications, specially designed pillows, and vibratory devices that detect supine position and prompt the patient to turn onto the side. Semi-upright sleep with the head and trunk elevated approximately 30-60 degrees is another option [6]. Positional therapy is generally less effective than CPAP but may be better tolerated and therefore used more consistently [22].

For most older adults with moderate or severe OSA, CPAP remains the treatment of choice. By mechanically maintaining upper-airway patency, CPAP can rapidly improve sleep quality, reduce snoring, and improve or normalize AHI. Effectiveness depends on disease severity, patient cooperation, device tolerance, and the dominant clinical phenotype. Studies have reported longer sleep duration, improved sleep architecture, less daytime fatigue, and subjective mood improvement. Some publications have also described improved cognitive function after 2-12 months of CPAP use or a slower progression of dementia [23, 24], although other studies have not confirmed a clear protective effect against progressive cognitive decline [7, 11, 13, 15].

With regard to cardiovascular and cerebrovascular outcomes, some studies have reported a protective effect of CPAP against stroke, heart failure, and related mortality in patients older than 65 years with severe OSA compared with untreated patients [11, 25]. A lower risk of recurrent stroke and death has also been observed in patients with at least moderate OSA treated with CPAP [2, 11]. Other studies, however, have produced less consistent results and suggest that clinical effectiveness may be more variable in older than in younger patients [3, 13]. The greatest benefit appears most likely in older adults with prominent daytime symptoms, a high hypoxic burden, and severe OSA, whereas effects may be less clear in very old, minimally symptomatic patients. Possible adaptation to chronic intermittent hypoxia has been proposed as one explanation. Treatment burden and adverse effects - such as nasal and oral dryness, mask pressure, claustrophobia, and air leakage - must also be considered [3, 11].

Adherence is a key issue in older patients. CPAP adherence is commonly defined as device use for at least 4 hours per night on at least 70% of nights [26]. Some studies report adherence in older adults comparable to that in younger populations, reaching 71-78% over 12 months, whereas others report poorer acceptance and discontinuation rates of 37-74% during the first year. Regular use declines particularly after the age of 80 years, partly because of reduced manual dexterity and progressive cognitive impairment. Patient education, caregiver

support, and appropriate mask selection can substantially improve CPAP tolerance and adherence [3, 7, 11]. The decision to use CPAP should therefore be individualized after balancing expected benefits against practical limitations and treatment burden.

Oral appliances can be used for habitual snoring and mild or moderate OSA, as an adjunct to positional therapy, or when CPAP is contraindicated or not accepted. Mandibular advancement devices (MADs) are the most widely used, but they generally require at least eight teeth in the dental arch and the ability to advance the mandible by at least 5 mm. Their use in older adults may therefore be limited by dental disease, temporomandibular joint disorders, restricted mandibular protrusion, and edentulism [3, 6].

Surgical treatment of OSA includes upper-airway procedures, hypoglossal nerve stimulation, and bariatric surgery. In older patients, surgical decisions require particular caution because perioperative risk and complication rates are higher than in younger groups in retrospective studies [6]. Comorbid medical conditions, polypharmacy, and the realistic magnitude of expected benefit must be considered. Surgery may be appropriate in selected non-obese patients with moderate OSA who decline CPAP, particularly when a clear anatomical obstruction is present in the nose, pharynx, or tongue base. Drug-induced sleep endoscopy (DISE) may be useful in uncertain cases, including in older adults. Poor nasal patency can increase the tendency of the pharyngeal soft tissues to collapse. Nasal blockage can also contribute to more severe OSA, and reduce tolerance of CPAP and oral appliances [27, 28]. Therefore, in carefully selected older patients without major medical contraindications, septoplasty, conchoplasty, or endoscopic sinus surgery may be considered. Patent's health condition and anesthetic/surgical risk, rather than age alone, should guide qualification. Nasal surgery has been associated with improved sleep quality, better CPAP tolerance, and longer CPAP use [27].

Soft-palate procedures should likewise be selected according to realistic potential benefit. Minimally invasive techniques such as radiofrequency or laser palatoplasty performed under local anesthesia may be reasonable in selected patients with mild OSA or with moderate disease who reject other options. These procedures aim to stiffen the soft palate and widen the oropharyngeal isthmus, potentially reducing soft-tissue collapse, but studies specifically evaluating their effectiveness in older adults are lacking. More extensive uvulopalatopharyngoplasty procedures are associated with higher complication rates and require general anesthesia, and are therefore rarely indicated in older patients [5, 29]. Hypoglossal nerve stimulation may represent a useful surgical alternative. A multicenter study from the United States and Germany reported a greater reduction in AHI and higher adherence in patients older than 65 years than in younger patients [30]. Bariatric surgery is highly effective for OSA and may lead to disease remission in up to 70% of patients, but candidates are predominantly younger adults [31].

Conclusions

OSA is a common but underdiagnosed disorder in older adults. Untreated disease may accelerate frailty and loss of independence and increase the risk of cardiovascular attacks and cerebrovascular events. Because OSA may have a less typical presentation in older than in younger people, greater diagnostic vigilance is required. Polysomnography remains the diagnostic reference standard. Treatment should be individualized according to the dominant symptoms, comorbidities, everyday functioning, and the patient's ability to cooperate with therapy. CPAP remains the treatment of choice for moderate and severe OSA, with the overarching aim of preserving independence and good physical condition of a patient for as long as possible.

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