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REVIEW

Sleep Apnea in the Elderly

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Abstract

Purpose of Review The population of the world is aging and increasing age has been associated with increased prevalence of obstructive sleep apnea (OSA). However, OSA in the elderly has not received much attention. The purpose of the present narrative review is to provide a comprehensive overview on what is known about the specific features of OSA, its consequences and treatment in the elderly.

Recent Findings The number of studies regarding OSA in the elderly is very limited. The clinical presentation of OSA in the elderly differs from the clinical presentation of OSA in middle-aged people. Furthermore, recent studies suggest that the risks caused by untreated OSA in the elderly differ from the risks in middle-aged patients with less focus on sleepiness, hypertension and cardiovascular health and more concerns on cerebrovascular health, cognition and perhaps even the risk of falls. However, the treatment of especially severe symptomatic OSA seems to have many benefits also in the elderly. Several studies suggest that CPAP adherence may also be good among the elderly. There is also emerging evidence of cost effectiveness of CPAP treatment in the elderly. Ongoing studies will reveal the effect of CPAP on cognitive decline and the risk of falls.

Summary Taking into consideration that the population is aging, there is an urgent need for larger studies especially on the phenotypes of OSA in the elderly and identifying groups benefiting from and adhering to CPAP therapy.

Keywords Sleep Apnea · Prevalence · Symptoms · CPAP · Cost-Effectiveness · Older Adults

Introduction

The population of the world is aging. The number of people aged ≥ 60 years is estimated to increase from 1.1 in 2023 to 1.4 billion by 2030, i.e. 1 in 6 people in the world will be aged ≥ 60 years [1]. Increasing age has been associated with increased prevalence of obstructive sleep apnea (OSA), but most studies have been performed in middle-aged men [2]. Further, studies focusing in OSA in the elderly use different definitions for "elderly". The first systematic review of original studies and expert consensus statement addressing the treatment of OSA in older or frail patients was published ten years ago [3]. The expert panel recommended that CPAP should be used routinely for the treatment of OSA in older persons and in the frail elderly, particularly in those with stroke but without major heart failure with an ejection fraction ≤ 45%. They also stated that patients with Alzheimer's or Parkinson's disease, as well as other frail elderly, do tolerate CPAP and treatment should be considered. They also strongly encouraged more and larger studies in elderly OSA patients. However, data on elderly OSA is still limited. There is no consensus, whether "elderly" should be applied to individuals aged over 60 or over 65 or over 70 years of age or something else. In this narrative review, we provide a comprehensive overview of OSA in the elderly.

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Prevalence and Incidence

In community dwelling elderly individuals of age ≥ 60 years, prevalence estimates of AHI ≥ 15/h range from 15.4% to 48.7% [2]. In studies, reporting prevalence estimates separately for sexes, the prevalence rates of AHI ≥ 15/h range from 24.2.% to 84.7% and from 15.9%

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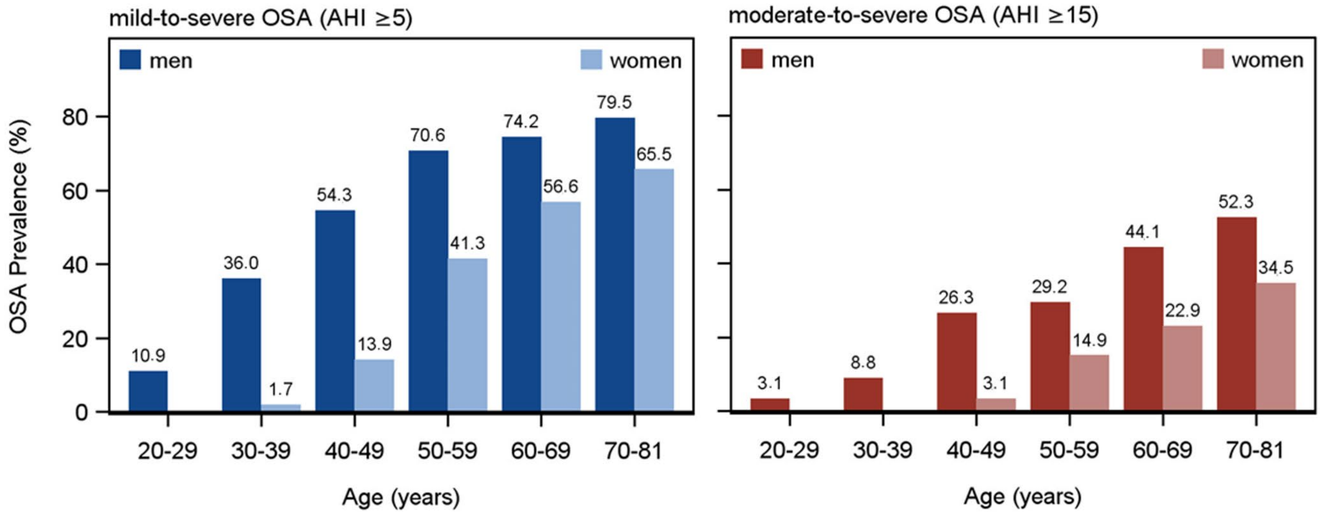

Fig. 1 Estimated OSA prevalence for age by 10-year categories, OSA severity groups and gender. With permission from Fietze I, Laharnar N, Obst A, Ewert R, Felix SB, Garcia C, et al. Prevalence and association analysis of obstructive sleep apnea with gender and age differences - results of SHIP-Trend. J Sleep Res. 2019;28(5):e12770

Table 1 Possible factors related to greater upper airway collapsibility in elderly. ↑ = increase, ↓ = decrease

Factor	Effect of aging
Upper airway dimensions (fat deposition, structural changes, loss of teeth)	↓
Airway resistance during sleep	↑
Upper airway muscle activity	↓
Lung elastic recoil affecting lung volume and tracheal traction	↓
Respiratory muscle strength	↓
Sleep fragmentation	↑
Respiratory instability during sleep	↑

to 36.2% in men and women, respectively [2]. A German population-based study reported estimated prevalences of AHI ≥ 5/h in 79.5% for men and 65.5% for women aged 70–81 years, and AHI ≥ 15/h in 52.3% and 34.5% in men and women, respectively [4]. In those aged 70–81 years, median AHI was 15.2/h and 8.5/h in men and women, respectively (Fig. 1.). AHI ≥ 5 combined with an Epworth Sleepiness Scale (ESS) score > 10 was found in 6.4% and AHI ≥ 15 combined with ESS score > 10 in 4.0% of individuals aged ≥ 60 years. Type of sleep study, inclusion of those with downstream organ dysfunction, asymptomatic individuals or overlooking other symptoms than daytime sleepiness, will have a significant impact on prevalence rates [4]. Of note, the wide array of prevalence estimates is affected by different age ranges, scoring criteria, and the type of a sleep study used.

According to a recent Finnish nationwide registry-based study, OSA incidence in 2020 was 7.6 per 1000 men and 4.5 per 1000 women in those aged ≥ 60 years [5], which is in line with a Canadian study reporting an incidence of 7 per 1000 individuals aged ≥ 60 years [6].

Pathophysiology

Evidence on potential underlying pathophysiologic mechanisms that may contribute to OSA in the elderly are limited or inconsistent. There are overlapping and possibly bidirectional effects between aging and OSA. Aging may cause physical and neurological changes that predispose to OSA and vice versa. Aging and OSA share common symptoms such as excessive daytime sleepiness (EDS), insomnia and impaired memory and cognition. Both aging and OSA are associated with cardiovascular and cerebrovascular diseases. Aging and OSA may have synergistic or cumulative effects on comorbidities, quality of life and functional capacity. It may be difficult to separate treatable consequences of OSA from the afflictions of old age.

Abnormal upper airway anatomy is considered a prerequisite for OSA. However, during the last 15 years, also other pathophysiological factors have been paid attention: collapsibility of the passive airway (Pcrit or closing pressure), responsiveness of upper airway muscles to respiratory stimuli, arousability, and loop gain (response of the respiratory controller to a transient disturbance) [7]. Aging may affect all of these pathophysiological factors. Moreover, other sleep disorders, chronic lung diseases, Alzheimer´s diseases, or medication with CNS effects may have an impact on those pathophysiological factors. Table 1 shows possible factors related to greater upper airway collapsibility in the elderly [8, 9]. Upper airway collapsibility has been suggested to be stronger both in elderly OSA [10] and non-OSA [11] individuals compared to younger ones.

In population based studies, elderly women have less severe OSA in terms of AHI [4, 12, 13], which might be explained by shorter and therefore less collapsible upper airway. However, the length of upper airway has not been compared between sexes in elderly OSA patients. Contrary to sex differences in OSA severity in general population, no difference was found in a clinical cohort in moderate to severe OSA patients aged 70–80 years (n=2010) or >80 years (n=457) [14].

Clinical Presentation

As is the case also in many other aspects of OSA, published data on the clinical presentation or symptoms of OSA in the elderly is scarce. OSA, however, seems to be underdiagnosed in the elderly largely due to their different symptom profile compared to middle-aged OSA-patients. Making clinical decision even more challenging, their symptoms are similar to and can therefore be confused with some of the normal signs and impairments of aging. For example, fatigue, nocturia, unintentional napping, and cognitive dysfunction may be ascribed to the aging process or to other disorders [15] and retirement or living alone may allow more opportunities to compensate for the consequences of sleep disruption [16]. Polypharmacy further complicates the process by often offering a possible cause for symptoms.

Elderly patients less often complain of snoring as a chief complaint of OSA [17]. Furthermore, the association between sleepiness and OSA seems to reduce with advancing age [18]. In addition, elderly OSA-patients are less obese and have more symptoms of insomnia compared to the middle-aged [19]. Elderly women with OSA seem to be more obese, have more depressive symptoms and mental distress than elderly men with OSA [14].

Among the older and more frail patients the atypical symptoms of OSA may indeed stand out, and in elderly people with new neurocognitive symptoms such as impaired attention and vigilance [20], or symptoms of impaired episodic memory [21], the possibility of OSA should be considered. However, it must be remembered that elderly people comprise a very heterogenous group and while the symptoms of a frail and multimorbid elderly patient depending on the help of others may be atypical, a functionally independent elderly patient may present with more classical symptoms.

In addition to neurocognitive symptoms, nocturia [22] or nocturnal polyuria [23] may also be a leading symptom. Moreover, as OSA has been linked to impaired balance or gait [24] and among the elderly, even with dizziness [25], and it has been proposed that recurrent falls may also be a symptom of OSA. This is an important topic for future research, as recurrent falls is a major cause of hospitalisations and can lead to a functional decline, loss of independence and frailty.

Comorbidity

Comorbidity and multimorbidity are common in elderly OSA patients [26]. This chapter focuses on cardiovascular diseases (CVDs), comorbid insomnia (COMISA), impaired cognition and dementia, and risk of falls.

Cardiovascular and Cerebrovascular Diseases

The role of OSA as a risk factor for CVDs in older patients seems to be less prominent than in younger patients, possibly due to other contributing factors. Adults from the Penn State Adult Cohort without cardiovascular or cerebrovascular diseases or severe OSA at baseline were followed-up after nine years. Mild-to-moderate OSA was not associated with incident hypertension, cardiovascular or cerebrovascular diseases in adults aged ≥ 60 years, although it was associated in younger adults [27, 28]. The risk of developing hypertension across each decade from 20 to 80 years of age was higher in OSA compared to the young and middle-aged individuals (20–60 years) but became non-significant after the age of 60 [27]. Contrary, no age effect on OSA and incident hypertension was found in the Wisconsin Sleep Cohort Study [29]. Further, the Sleep Heart Health Study (SHHS) found no association between mild to moderate OSA and cardiovascular and cerebrovascular diseases, which may be explained by that the population consisted primarily of older adults with an average age of 63 years (range 40–79 years) [30].

Several hypotheses have been suggested to explain the lack of association between OSA and incident CVD in older adults. First, chronic OSA exposure may have a protective effect (ischemic preconditioning) [31]. Second, older individuals may represent a more resilient group of survivors due to genetic and environmental factors (selection bias) [32]. Third, the lack of effect of OSA on CVD may be due to the detrimental effects of aging masking the effects of OSA (ceiling effect) [32]. Fourth, OSA in older adults may represent a distinct phenotype with altered physiological response to OSA than in young and middle-aged individuals.

Contrary to above mentioned studies, a Spanish prospective observational study of consecutive patients aged ≥ 65 years with suspected OSA, found that untreated severe OSA is an independent risk factor in elderly patients for incident stroke during the median follow-up of six years [33]. However, the incidence of coronary heart disease was not increased. The different impact of OSA on cerebrovascular and coronary heart disease in the elderly could be explained by different risk profiles linked to OSA. Another possible explanation could be ischemic preconditioning due to long-term intermittent hypoxia, which may trigger the formation of collateral vessels in the heart, but not in the brain [34]. Also, differences in disease exposure time may explain the controversy in cardiovascular outcomes in elderly OSA patients.

Recently, it has been drawn attention to the role of hypoxic burden in cardiovascular risks. In two large community-based samples of middle-aged and older adults, the SHHS and Osteoporotic Fractures in Men (MrOS), hypoxic burden, but not AHI, predicted incident heart failure in men but not in women [35]. These associations persisted after adjusting for multiple potential demographic factors, smoking, and co-morbidities, and possible mediators such as diabetes, hypertension, and stroke.

OSA and OSA-induced hypoxia may correlate with the severity of myocardial infarction (MI), increase the occurrence of heart rhythm disorders, and worsen their short-term outcomes. In a prospective study with 252 acute MI patients (mean age 68 years) undergoing revascularization had a sleep study during hospitalization. OSA patients (AHI ≥ 15/h) had higher BMI and lower $MinSaO_2$, greater proportion of multivessel coronary artery disease, and higher incidence of arrhythmias. After a median follow-up of six months, OSA combined with $MinSaO_2$ ≤80% was independently associated with the incidence of major adverse cardiac and cerebrovascular events (MACCEs) after adjusting for traditional risk factors (HR 4.54). Those findings suggest that OSA associated with hypoxia may lead to instability of autonomic nervous system function, triggering the incidence of ischemia-related arrhythmia in elderly subacute MI patients and further worsening their prognosis [36].

Depression is common in elderly patients with OSA [14]. In elderly OSA patients without history of myocardial infarction, heart failure, or hospitalization for unstable angina, depression was an independent risk factor of major adverse cardiovascular events (MACE) [37]. In a previous study depression predicted CVD in males with OSA [38]. This gender effect may be explained by the fact that men are more socially isolated and less socially supported than women, and both isolation and social support are associated with CVD [39]. Further, the mechanism linking depression to CVD in male patients may be linked to arterial stiffness [40]. Of note, lower nocturnal SpO_2 was associated with increased prevalence of depressive symptoms in a clinic-based study of OSA patients aged ≥ 70 years [14]. Therefore, depression might contribute to CVD risk via nocturnal desaturation.

These findings suggest that OSA in older adults may be a distinctly different phenotype than in young and middle-aged adults, which may have implications for the management of OSA. The findings are inconsistent but suggest the association of OSA-related hypoxemia instead of AHI as a risk factor and highlight the impact of different OSA phenotypes as a risk factor for cardiovascular and cerebrovascular diseases. Further, contribution of OSA may differ among cardiovascular and cerebrovascular diseases.

Insomnia

Co-occurrence of insomnia and OSA (COMISA) is common due to high prevalence of both disorders. Moreover, insomnia symptoms are a major part of the symptom burden in OSA [41]. A 3% increase in the odds of COMISA has been reported with each additional year of age being 51.9%, 74.6% and 78.1% in OSA age categories of < 50 years, 50–69 years and ≥ 70 years, respectively [42]. In 110 older adults aged 65–83 years with overnight polysomnography diagnosed OSA and Insomnia Severity Index score ≥ 11, COMISA was associated with significantly worse verbal memory performance in women but not in men. Moderation analysis was adjusted for age, BMI, APOE4 status, and education. Post hoc analyses revealed that women with COMISA showed reduced REM sleep and increased slow wave sleep (SWS) compared to men with COMISA [43].

Cognitive Impairment and Dementia

Recent reports have shown that OSA is a risk factor for the pathology and progression of Alzheimer´s disease (AD). There is a link between OSA and AD biomarkers of neurodegeneration [44]. It has been hypothesized that sleep impairment and sleep deprivation, SWS disruption, glymphatic system dysfunction, intermittent hypoxemia, and increased intrathoracic and intracranial pressure due to apneic events could be triggering factors for AD neuropathological changes [45]. Several studies have found an association between nocturnal hypoxemia and cognitive decline or dementia in the elderly OSA patients [46–49]. In a prospective study of 298 women without dementia (mean age 82 years), the women with AHI ≥ 15/h or oxygen desaturation index (ODI) ≥ 15 events/h and high percentage (> 7%) of sleep time with apneic or hypopneic events, were more likely to develop mild cognitive impairment or dementia during a five-year follow-up compared with women without OSA [46]. Arousal index, wake after sleep onset (WASO) or total sleep time were not associated with risk of cognitive impairment. In the MrOS Sleep Study 2,636 community-dwelling men (mean age 76 years) without mild cognitive impairment were followed for three years [47]. Men with ≥1% of sleep time with SaO_2<90% had an adjusted annualized decline of 0.43 points in Modified Mini-Mental State examination test (MMSE) compared to 0.25 for in the control group. For each 5-point increase in ODI, there was an average annualized decline of 0.36 points. The association between AHI and cognitive decline was not significant.

In the HypnoLaus Study cohort of community-dwelling elderly individuals without dementia (mean age 71 years), AHI and indices of nocturnal hypoxemia were associated with steeper cognitive decline over 5 years, whereas sleep fragmentation had no association. The cognitive decline was observed in global cognitive function, processing speed, executive function and episodic verbal memory, but not in language or visuospatial function. Participants with mean SpO_2≤92.5% during sleep had a steeper decline in MMSE, processing speed and episodic verbal memory. Time spent with SpO_2 lower than 90% ≥4.5% associated with a steeper decline in processing speed. Moderation analysis showed that AHI and ODI associated with a steeper decline in global cognitive function, processing speed and executive function only in older participants, men and ApoE4 carriers (Fig. 2.) [49].

The large population-based Health and Retirement Study (n=18,815) found a sex-specific dementia risk in individuals with known or suspected OSA during a ten-year follow-up [50]. Known or suspected OSA associated with higher cumulative incidence of dementia across ages 60–84 years for women and men. By age 80, relative to adults without known or suspected OSA, the cumulative incidence of dementia was 4.7% higher for women and 2.5% for men. Adjusted associations between age-specific OSA and cumulative incidence of dementia attenuated for both women and men but remained significant.

To summarize, in elderly individuals, cross-sectional and longitudinal associations between OSA and cognition are highly variable, depending on the sleep study type, setting, and OSA criteria as well as type of neurocognitive tests. In cross-sectional studies, OSA is more prevalent among older individuals with AD and/or dementia than in those with normal cognitive function.

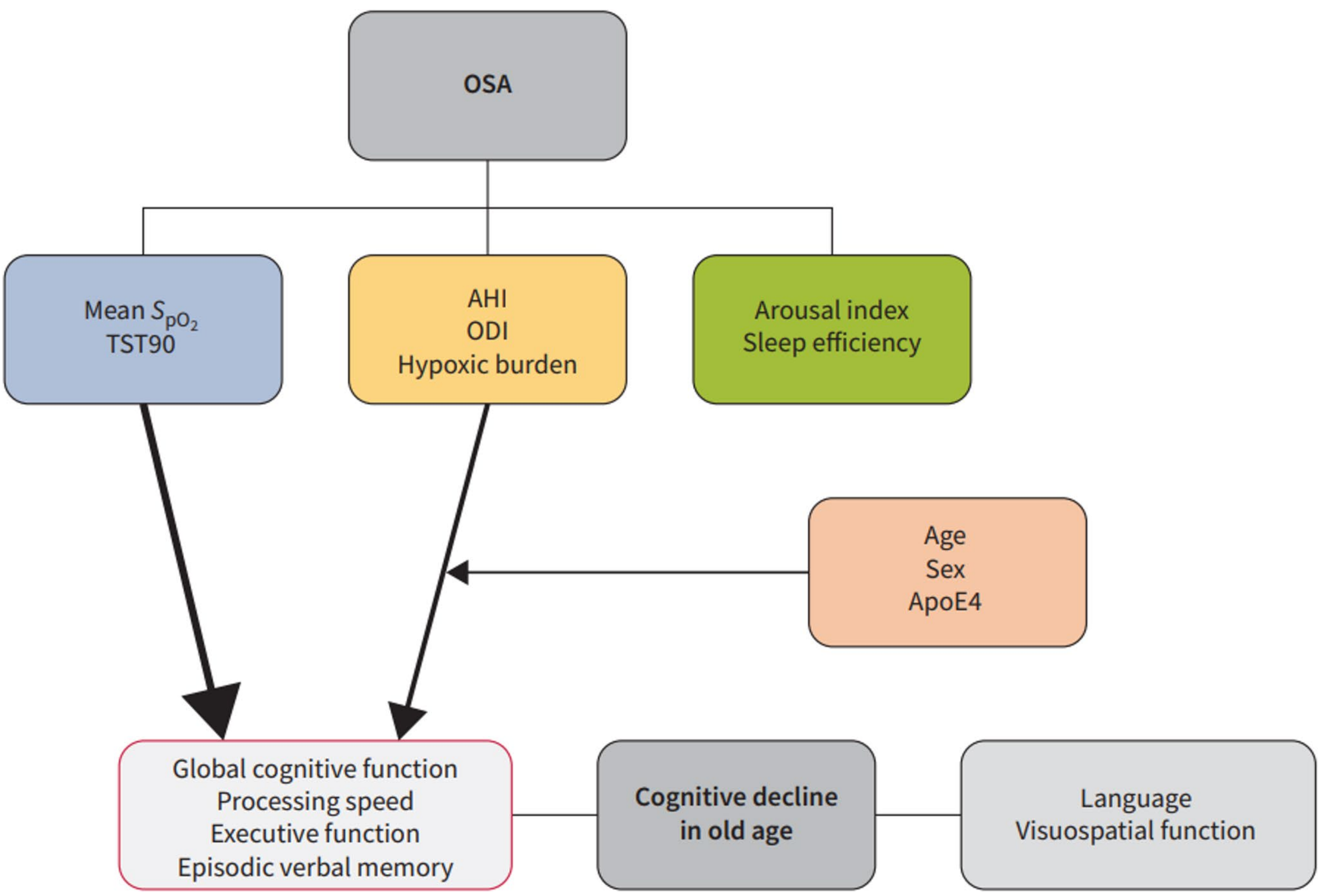

Fig. 2 Overview of the factors contributing to cognitive function in elderly patients with obstructive sleep apnea. The thickness of the arrows indicates the strength of the associations. SpO2=mean peripheral oxygen saturation, TST90=sleep time with SpO2 <90%. With permission from Marchi NA, Solelhac G, Berger M, Haba-Rubio J, Gosselin N, Vollenweider P, et al. Obstructive sleep apnoea and 5-year cognitive decline in the elderly. Eur Respir J. 2023;61(4):2201621

Risk of Falls

The overall control of balance relies on a complex interaction between several physiological functions including vestibular, muscle, visual, and cognitive functions [24]. OSA-related hypoxemia and sleep fragmentation may possibly affect these different systems. The few studies on OSA show evidence of an increased risk for falls. Information regarding sleep habits in individuals aged ≥ 60 years (n = 1,952) was collected during the 10-year follow-up. After controlling for variables associated with falls, OSA, but not snoring, witnessed apnea, or an ESS score above 10, was associated with a doubling of the risk of two or more falls during the previous 12 months. OSA was also associated with a higher Geriatric Depression Scale score, and inclusion of the depression variable into the model attenuated the relationship between OSA and falls. Including the MMSE score did not markedly change these results. OSA-induced hypoxemia and sleep fragmentation are associated to daytime functional impairments in executive function, vigilance, alertness, fine motor coordination, and psychological disturbance, which may explain the findings [51]. In the prospective observational MrOS Sleep Study of 3,101 community-dwelling men ≥ 67 years of age, actigraphically measured sleep efficiency was also associated with increased risk of falls, as was nocturnal hypoxemia (≥ 10% of sleep time with SaO_2 < 90%), but not the AHI [52]. Further, in a middle-aged cohort nocturnal oxygen saturation level had the strongest independent association with impaired daytime postural control [53]. A recent large study utilized Taiwan's National Health Insurance Research Database from 2000 to 2015 with 8,901 individuals diagnosed with OSA, and finally including 6,915 participants into the study cohort. OSA patients aged ≧ 65 years OSA had a two-fold risk for overall injuries compared to non-OSA individuals. The risk of injury in patients with OSA increased with concomitant comorbidities, including DM, hypertension, hyperlipidaemia, CVD, stroke, obesity, anxiety, and depression [54].

Mortality

The relationship between severe OSA and all-cause mortality seems to be influenced by age. Data from 146,148 individuals of the Veterans Health administrative electronic medical records in 1999–2022 reported an increased rate and odds of all-cause mortality among younger population, but this association reversed with increasing age [55]. Similar findings have been reported from smaller cohorts [28, 31, 56]. The impact of OSA on all-cause mortality in different age groups might be due to survivor bias. Further, it has been hypothesized that OSA patients successfully adapt to the nightly apneic events by an unknown mechanism, possibly by ischemic preconditioning [31] or adapt to nightly use of CPAP. However, in a study of 289 participants (age > 65, no dementia or depression at baseline) with an average follow-up of 13.8 years, the presence of AHI ≥ 20/h and EDS was associated with an increased all-cause mortality risk in older adults even when adjusting for sleep duration > 8.5 h, self-reported angina, male gender, African American race, and age. In those who had OSA without EDS, or EDS without OSA, there was no increased all-cause mortality rate [57]. There may also be gender differences in mortality risk in the elderly OSA patients but the data are scarce. Ancoli-Israel et al. reported nearly forty years ago that elderly female OSA patients in nursing home had higher mortality rate compared to males [58].

Effect of CPAP Treatment

CPAP Adherence

As the prevalence of OSA increases in older patients, the proportion of very elderly patients treated with CPAP is expected to become even higher [9]. However, there is evidence that despite similar severity of OSA, fewer of the patients aged ⩾80 years were prescribed CPAP compared to those of 65–79 years [59]. CPAP adherence studies of OSA patients over 65 years of age are scarce, and the results are still controversial. Some studies have found lower CPAP adherence in elderly patients [59] or declining adherence after 80 years of age [60] or as good or even better CPAP adherence as in middle-aged CPAP users [61, 62].

In a Spanish cohort study of 939 consecutive patients aged ⩾65 years referred for suspicion of OSA and CPAP users were stratified into four age groups, namely 65–69, 70–74, 75–79 and over 80 years [59]. They were followed for a median of 69 months. The adherence to CPAP showed a progressive decrease with advancing age groups, especially after 75 years. They hypothesized that lower adherence observed in older OSA patients probably has many causes, including a greater number of comorbidities, neurocognitive impairment, difficulties with self-fitting the mask caused by osteoarthritis, lack of family support or decrease in the quality or quantity of sleep. All these factors are associated with advanced age [63].

In a large French eQUALISAS cross-sectional study of CPAP treated OSA patients, adherence data from 26,343 patients including 1,656 patients aged 80–85 years and 639 patients aged ≥ 85 years was analysed [60]. They demonstrated that adherence gradually increased with age until 80 years and thereafter slightly decreased (Fig. 3.). No significant relationship was found between adherence after 80

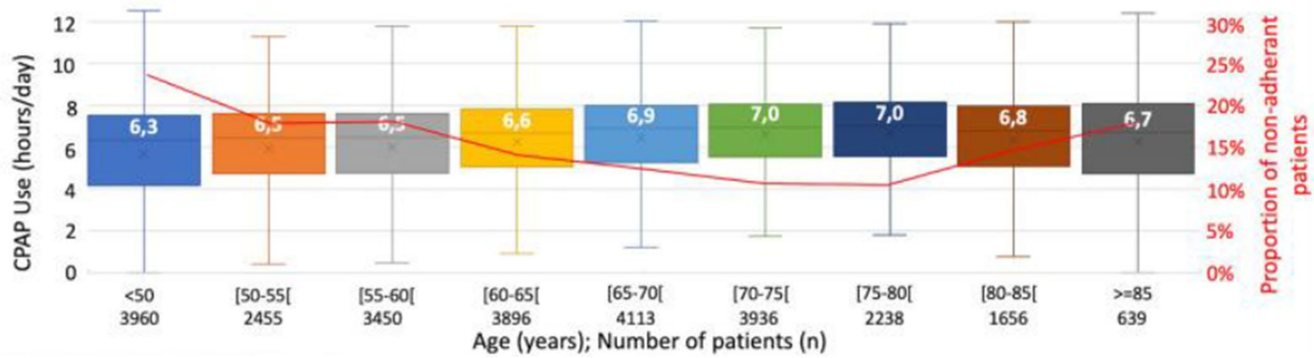

Fig. 3 Age-category–specific adherence to CPAP therapy in middle-aged and elderly patients and proportion of non-adherent patients (Red curve). Modified with permission from Prigent A, Blanloeil C, Serandour AL, Barlet F, Gagnadoux F, Jaffuel D. A biphasic effect of age on CPAP adherence: a cross-sectional study of 26,343 patients. Respir Res. 2023;24:234

years old and gender, leaks and residual AHI. As an alternative hypothesis, they suggested that the decrease in CPAP adherence after 80 years might be explained by the age-related increase in wake after sleep onset or changes in sensory perception of autonomy that may make CPAP use more difficult. Also, chronic sleep problems, loneliness, lack of social support, or increased prevalence of heart failure and stroke with age are associated with poorer sleep quality and hence may cause difficulties in CPAP use. Another large French real-life data (ALASKA) investigated long-term CPAP therapy termination rates in different age groups, focusing on the contribution of comorbidities [64]. The risk of termination of CPAP therapy was higher in older patient groups (71–80 years and over 80 years) with one or more comorbidities compared to middle aged group.

In a US study of 789,260 patients initiated on CPAP including age groups of 18–30, 31–40, 41–50, 51–60, 61–70, 71–80, 81–90 years, older patients (up to 71–80 years) showed a gradual increase in usage within the 90 days follow-up [61]. Overall adherence was 72.6%, being highest 80.6% in men aged 71–80. They concluded that tailored strategies are needed to address the specific challenges faced by different age groups to enhance adherence and maximize the benefits of CPAP therapy.

The European Sleep Apnea Database (ESADA) study on 6,547 patients on CPAP is one of the largest studies including 1,766 patients aged 60–69 and 687 patients aged 70–79 years [19]. They found good CPAP adherence in elderly and even better among them than in younger patients (Fig. 4.). Age, gender, EDS, OSA severity, or clinical phenotypes based on EDS and insomnia symptoms did not affect CPAP adherence in the elderly group but higher rating of Clinical Global Impression scale by the clinician, predicted lower CPAP adherence. This was confirmed in a recent ESADA study although more confounding factors, basic clinical characteristics, polypharmacy (reflecting multimorbidity), the Baveno classification, the use of medications acting through the central nervous system, or the general health status of the patient, were taken into account [65].

In the two large studies from the US and France where they included also patients over 80 years of age, female OSA patients had lower CPAP usage than male patients in all age groups [60, 61]. As OSA has been viewed as a male disease, women may be less willing to accept CPAP treatment, which can be seen unattractive as well. Claustrophobia may be more common in women than in men prohibiting CPAP usage [66]. Other reasons for lower CPAP adherence in women may be gender differences in OSA pathophysiology and more common insomnia and fatigue symptoms in women, which may be less responsive to CPAP therapy.

CPAP Effect on OSA Symptoms and Findings

In symptomatic OSA, CPAP therapy is the mainstay of treatment [67]. Earlier studies about CPAP effects have included mainly up to middle-aged OSA patients and studies on patients over 65 years of age are few. Studies of CPAP effect after three or twelve months of use on daytime sleepiness measured with ESS show improvement in EDS in patients over 65 years [68–74]. The improvement was greater in ESS score when CPAP usage was more than 4 h per day [69, 72]. Although a dose-dependent effect of CPAP therapy has been found, there is evidence that as little as 2 h of usage might be enough to improve OSA- related symptoms and reduce health care utilization whereas device usage of 4–6 h/night appears to be necessary to reduce blood pressure and cardiovascular risk [75]. In a multicentre French study of adherent CPAP patients (mean use > 4 h/night), the impact of six months' use of CPAP on fatigue and ESS was stronger in younger patients compared to patients over 50 years [76]. This is in line with the ESADA study on elderly CPAP patients where ESS score decreased proportionally more in the 40–49-year-olds than in the 70–79-year-olds following

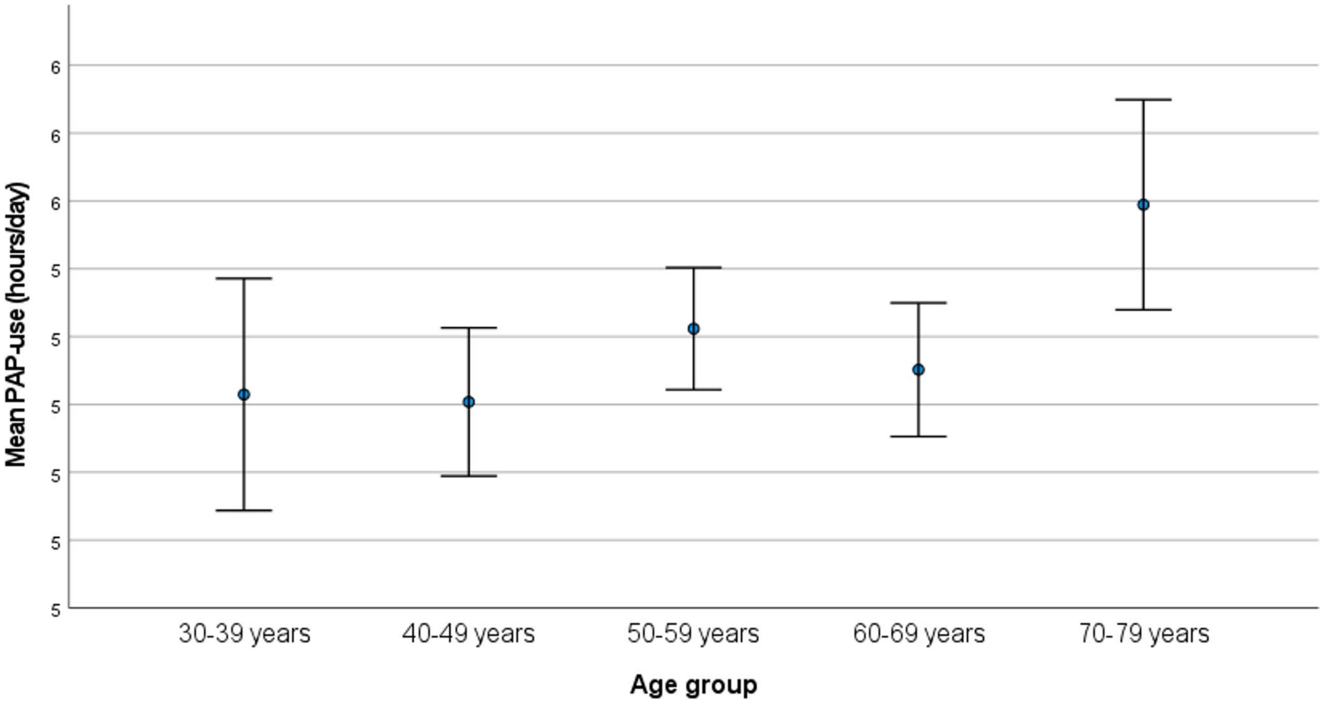

Fig. 4 Mean CPAP use of sleep apnea patients in the ESADA database according to ten-year age groups. The error bars represent 95% CIs. P-value (for the differences across all groups) 0.035. With permission of Lammintausta A, Anttalainen U, Basoglu ÖK, Bonsignore MR, Gouveris H, Grote L, et al. Clinical characteristics and positive airway pressure adherence among elderly European sleep apnoea patients from the ESADA cohort. ERJ Open Res. 2023;9:00506–2022

CPAP therapy [19]. One reason for this was the lower baseline level of sleepiness in older age groups in both studies. It is also noteworthy that ESS is not validated with elderly and therefore ESS seems to reflect daytime sleepiness less well among the elderly [77].

Three recent reviews on CPAP effect on elderly OSA patients have been published [78–80] looking at changes in neurocognitive tests, sleep-related quality of life, mood, mortality and ESS as well. They included 4 to 13 studies on patients over 65 years of age using CPAP therapy and compared to untreated OSA patients. In these reviews, CPAP therapy improved EDS and reduced anxiety, depression, and mortality, while at least partially improved neurocognitive function and sleep-related quality of life. These benefits were most pronounced in patients who maintained consistent device usage of more than four hours per night. Therefore, larger and well-structured randomized controlled trials involving compliant patients are essential to provide more robust evidence on the effects of CPAP therapy and to better identify the appropriate elderly OSA phenotype for CPAP treatment. However, OSA should already be accepted as an independent risk factor in the elderly population, and they should be granted with a CPAP trial.

CPAP Effect on Cardiovascular Comorbidities and Mortality

RCTs evaluating the CPAP effects of treating OSA on cardiovascular events and all cause death have not demonstrated any beneficial effect [81–83]. These RCTs have been criticised by several factors including non-sleepy patient selection with low adherence to CPAP, short follow-up time and the low number of total mortality events. Those issues may limit the statistical power to detect between-group differences and may not be representative of real-world conditions. In the recent systematic review and meta-analysis of RCTs and confounder-adjusted, non-randomised controlled studies of CPAP and all-cause and cardiovascular mortality in people with OSA, a potentially beneficial effect of CPAP therapy was found, and it was more pronounced as nightly CPAP use increased [84].

In three non-RCT but controlled studies including only OSA patients over 65 years, mortality was significantly reduced with CPAP therapy compared to no CPAP with median follow-up time of 4–5 years [67, 85, 86]. In line with these studies and contrary to RCTs, the large French real-world data including 88,007 OSA patients (mean age 60 years), continuation of CPAP therapy was associated with a significantly lower risk of all-cause death compared with CPAP therapy termination and the results were similar in men and women [87]. Incident heart failure also was less common in patients who continued vs. terminated CPAP therapy.

Given the high prevalence of OSA and CVD especially among older population, only a few studies have evaluated the effect of CPAP therapy on cardiovascular (CV) outcomes [88]. In the PREDICT trial of OSA patients over 65 years of age after 12 months follow-up of CPAP therapy vs. medical treatment alone, no significant difference in the incidence of CV events were between the groups [68]. One explanation to the negative results was poor CPAP adherence in the study. From ten years back in a prospective cohort study of 130 elderly OSA patients with or without CPAP therapy with mean follow-up of five years, incidence of CV events was 13.9% in the CPAP group and 55.7% in the untreated group [85]. In a large retrospective study of more than 5,000 OSA patients over 65 years initiating CPAP treatment, good CPAP adherence was associated with reduced risk of new CVD events [89]. This risk reduction was consistent across race, sex, and socioeconomic subgroups and was seen at 6-, 12- and 25-month but not at 3-month follow-up after CPAP initiation. This is supported with a recent prospective observational study of 420 OSA patients aged over 65 years with or without CPAP therapy with 22 months' follow-up where optimal CPAP adherence combined with usual medical care for CVDs associated with a lower incidence of MACE and recurrent atrial fibrillation [90]. These results point out that in older OSA patients, especially with CVD, the efforts to maximize the adherence to CPAP should be prioritized [89, 90].

CPAP Effect on Falls

There is only emerging evidence suggesting that treating OSA with CPAP can improve gait or risk of falls [91–93] but also a small RCT study with negative results has been published [94]. The largest randomised controlled clinical study on this subject with 140 OSA patients over 60 years after six months of CPAP therapy is still ongoing [95]. The inconsistency in previous findings may relate to small number of participants, short follow-up period of CPAP therapy or that recruited participants were from sleep clinics and therefore not generalizable to populations who may be at higher risk of falls.

Cost-Effectiveness

Studies on cost-effectiveness of CPAP treatment in elderly adults are scarce. Recent data suggest savings via reduction of MACE [96] and other comorbidity-related costs in CPAP-users [26]. Among nearly 900,000 Medicare beneficiaries with OSA (median age 73 years; 44% women, median follow-up 3.1 years), those with evidence of PAP initiation had significantly lower all-cause mortality and MACE incidence risk [96]. In over 28,000 Medicare beneficiaries aged ≥ 65 years with newly diagnosed OSA, 45% were adherent to PAP, 10% were non-adherent, and 44% did not initiate PAP. Relative to non-initiators, beneficiaries who initiated PAP displayed $195 reduced per-member per-month costs over 24 months. This finding remained consistent across all seven subgroups (patients with comorbid hypertension, diabetes, COPD, obesity, depression, chronic heart failure, or stroke), as well as among individuals with multimorbidity [26]. In a Danish nationwide, register-based case-control study with 21,555 OSA patients aged ≥ 65 years (29.1% females) and 86,212 matched controls, public transfer payments increased over time for both groups [97]. OSA patients received significantly higher total public transfers than controls from index year − 15 to + 1. Further, the difference in total healthcare costs per patient-year increased in a similar manner before the diagnosis, peaking at the index year, where the net cost difference was €5219. While costs declined slightly thereafter in the OSA group, they remained elevated compared to controls throughout the post-index period. Elevated costs in OSA patients were consistently observed across all healthcare sectors, including somatic inpatient and outpatient care, psychiatric services, primary care, and prescription medications [97]. These findings suggest that earlier detection and treatment of OSA may reduce or delay societal and healthcare expenditures. Therefore, diagnosis and treatment in a timely manner may turn out to be cost-effective.

Effect of Non-CPAP Therapies

A recent review has comprehensively explored the possibilities of pharmacological therapies in OSA treatment [98]. Old age especially among men is associated with higher upper airway collapsibility. Pharmacotherapeutic agents might be beneficial in this group, but such treatment in older men is likely to be contraindicated [99]. Glucagon-like peptide-1 (GLP-1)/glucose-dependent insulinotropic polypeptide (GIP) -receptor agonists may be beneficial in obese patients with metabolic comorbidities. Tirzepatide is the first FDA-approved and EMA-sanctioned pharmacologic treatment in obese OSA patients [100]. However, the effects on elderly OSA patients are not known. Also alerting drugs might alleviate daytime sleepiness and thereby improve functioning but data in elderly OSA patients are not available.

COMISA is common particularly among elderly patients [42]. Melatonin and z-drugs might provide beneficial effects. However, adverse effects of z-drugs have to be weighted against benefits. Cognitive behavioral therapy

Table 2. Proposal of CPAP treatment indications for OSA patients aged > 65 years. The strength of treatment indication is depicted a follows: ■ = very weak, ■ = weak, ■ = moderate, ■ = strong, ■ = very strong. T90 = time spent under oxygen saturation of 90% during the diagnostic sleep study. Based on [26, 97, 109, 111–114]

Cardio-metabolic comorbidity* or COPD	Asymptomatic			Symptomatic**		
	AHI 5-14.9/h	AHI 15-29.9/h	AHI≥30/h or T90≥15 min	AHI 5-14.9/h	AHI 15-29.9/h	AHI≥30/h or T90≥15 min
None or well controlled						
In-suffciently controlled						

* Systemic hypertension, atrial fibrillation, ischemic heart disease, heart failure, stroke, diabetes, obesity

** Daytime sleepiness, non-restorative sleep, morning headache, drowsy driving, reduced work performance, snoring, witnessed apneas, nocturia, nocturnal dyspnea, sweating, dry mouth, insomnia, nocturnal awakening

(CBT-I) increases the use of CPAP therapy in middle-aged patients [101] but data in elderly patients are lacking.

Data on mandibular advancement devices (MAD) [102] and myofunctional therapy [103] are scarce in the elderly OSA patients although those treatment modalities may be beneficial in selected patients. Loss of teeth and gingivitis may prevent use of MAD. Data on positional treatment of OSA in the elderly are scarce, although there is a report of beneficial effects in nine elderly patients [104].

Who Should Be Treated?

Clinical presentation of the elderly OSA patients differs from the younger ones. Also risks of untreated OSA are different, e.g. risk of falls. It has to be emphasised that the AHI per se is not sufficient to decide if the OSA is clinically relevant or not. Moreover, as AHI increases with aging [4], the traditionally applied cut-off values to classify OSA severity are unlikely to be relevant in the elderly patients. According to the meta-analysis of Boulos et al. [105] the normative AHI in the age group of 65–79 years is 15.5/h and in those of age ≥ 80 years 30.3/h. However, as the authors acknowledge the findings of the meta-analysis are statistically underpowered for older adults (≥ 65 years) due to the low numbers of studies and high level of clinical heterogeneity in this age group. Of note, the AHI in the oldest age group was derived from a single study with only ten male subjects. Recently, measures of hypoxic burden during sleep have been proved to be better predictors for risk and symptoms [106]. OSA treatment improves a variety of daytime and sleep related symptoms irrespective of patients age and AHI at baseline [107] although the impact on fatigue and depressive symptoms may be weaker in older patients [76]. This implicates a moderate to very strong indication for treatment in symptomatic OSA patients. On the other hand, treatment indication is weak in asymptomatic, otherwise healthy elderly OSA patients. However, in any case, an informed discussion is of importance between a health care professional and a patient regarding the advantages and disadvantages of different treatment options. Of importance, the recent data from the US and Denmark suggest that earlier detection and treatment of OSA in elderly patients is likely to be cost-effective [26, 97].

Treatment goals in the elderly OSA patients should be driven more by symptoms and functional status than AHI. There is lack of data on clinical phenotypes in elderly OSA patients. It is important to take into consideration the elderly patients' functional status when considering OSA diagnostics and treatment. The CGI-S [65] is an easy subjective measure of health and functional status. However, we urgently need for future OSA studies and everyday clinical decision making more specific measures of functional capacity, for example, the easy-to use 7-point Clinical Frailty Scale [108] as predictors of treatment efficacy and adherence in the elderly. Recent Swedish Guidelines for OSA treatment [109] utilized the data from the national Swedish Sleep Apnea Registry (SESAR), Baveno classification [110], literature search, and a Delphi round amongst experts from all Swedish regions. Based on those Swedish guidelines [109] and studies from Marin et al. [111, 112], Matthes et al. [113], and Jorquera et al. [114], we propose CPAP treatment indications for OSA patients aged > 65 years (Table 2). This proposal considers AHI, time spent under oxygen saturation of 90% during the diagnostic sleep study, symptoms (not only EDS), cardio-metabolic comorbidity, COPD, and whether the comorbidities are well-controlled or not.

Conclusion and Future Directions

In elderly OSA patients, clinical presentation and risks differ between sexes and compared with young and middle-aged patients. Treatment of especially severe symptomatic OSA seems to be beneficial also in the elderly. CPAP adherence varies between studies but is generally about 70% and as good or even better than in younger patients. Further, recent studies suggest cost-effectiveness of CPAP treatment in elderly OSA patients.

There is urgent need for studies defining diagnostic and treatment recommendations for different elderly age groups. To achieve these goals requires research on phenotypes in the elderly, validation of specific questionnaires, controlled studies of effectiveness of CPAP and other treatment modalities on quality of life, functional capability, and use of health care resources related to comorbidities. Moreover, we should form a consensus of the term “elderly” or stratification of elderly individuals, when reporting results of future studies.

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Declarations

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