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Chapter 7

Sleep disorders

The Most Effective Treatments for Snoring and Sleep Apnea

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Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a prevalent disorder characterized by intermittent cessation of airflow during sleep that results in excessive daytime sleepiness. Common presenting symptoms include habitual snoring, witnessed apneas, nighttime gasping or arousals, daytime sleepiness or fatigue, unrefreshing sleep, and morning headaches. The diagnosis of OSAHS is established if the patient has both an apnea-hypnea index (AHI) score \geq 5 on overnight monitoring, and evidence of excessive daytime sleepiness or fatigue. OSAHS is a prevalent disorder in Western society estimated to affect up to 5 percent of the adult male and 3 percent of the adult female population.¹ The focus of this review is the growing body of evidence that suggests that the burden of OSAHS is especially high in older adults.

Epidemiology of OSAHS in Older Adults

Several large population-based cohort studies have demonstrated that undiagnosed sleep apnea is higher in the \geq 60-year-old age group compared with younger age groups.² It is estimated that the rate of AHI \geq 5 is 50-60 percent in older men and 30-40 percent in older women, which is three times greater than the general adult population.³ Menopausal status appears to be a major determinant of the rate of OSAHS in women. In the large population-based Wisconsin Sleep Cohort Study, postmenopausal women were found to be 3.5 times more likely than premenopausal women to have AHI scores \geq 15.⁴ A number of theories have been proposed to explain the increased prevalence of OSAHS in older age groups, including ongoing loss of neuromuscular tone,⁵ decreased hormone levels,⁶ and changes in the ratio of fat to lean body mass.⁷

Although the prevalence of OSHAS appears to increase steadily with age, the overall prevalence of the disorder appears to level off after the age of 65 years.⁸ The reason for the plateau after age 65 is not clear but may be explained in several ways: (1) the incidence of new cases of OSAHS decreases after age 65; (2) the mortality rate of OSAHS cases increases after age 65; or (3) OSAHS remits at older age. Because there is little evidence to support death caused directly by OSAHS or spontaneous remission of OSAHS, a reduction in incidence after age 65 is the currently favored explanation;⁹ however, further investigation is needed to clarify this issue.

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Sleep Patterns in Older Adults

Changes in sleep duration and architecture occur as a normal part of the aging process.¹⁰ The duration and need for sleep appears to lessen with age. The average 70-year-old sleeps only 6 hours per night but may make up for loss of nocturnal sleep with daytime naps of 1 to 2 hours.¹¹ In addition, many older adults have increased difficulty falling to sleep once in bed (increased sleep latency), and have greater difficulty staying asleep (decreased sleep efficiency).¹² The number of co-morbid medical conditions that have an impact on sleep increases with age and includes depression, arthritis, gastroesophageal reflux, prostate hypertrophy, and renal and pulmonary disorders.¹³ In addition, older adults are more likely to take medications such as diuretics that cause nocturia. As a result, up to 40 percent of older individuals complain of sleep disturbance and undesired daytime fatigue.14

The challenge for the physician presented with complaints of sleep disturbance in older individuals is to determine the degree to which symptoms are related to normal age-related changes in sleep pattern versus an underlying medical disorder or a primary sleep disorder. A thorough review of the patient's past medical history, medications, and alcohol and stimulant (caffeine, nicotine) use is required. Improving the management of co-morbid conditions (for example, reflux, arthritic pain, or prostate hypertrophy) will often result in sleep improvement. A thorough sleep history (table 1) helps identify patients at risk of the primary sleep disorders seen most commonly in older adults: OSAHS, restless leg syndrome, insomnia, and sleep maintenance insomnia. It is important to note that sleep maintenance insomnia, which is characterized by early morning awakenings, is highly associated with either alcohol abuse or depression in older individuals.¹⁵ Patients suspected of a primary sleep disorder should undergo a fullnight polysomnography in order to establish the presence and severity of a sleep disorder. The diagnosis of OSAHS can be established in patients with daytime sleepiness or fatigue who are found to have an AHI \geq 5 on overnight polysomnography.

Table 1: Elements of sleep history

Time in bed Time of sleep Number of awakenings Rising time Presence of snoring, witnessed apneas, gasps, or choking spells Presence of involuntary leg motion or jerking Level of daytime sleepiness or fatigue (Epworth Scale Score) Time, duration, and number of daytime naps

Clinical Sequelae of OSAHS

Untreated OSAHS has been associated with reduced quality of life as well as a number of serious health conditions.

Reduced quality of life. Daytime sleepiness caused by OSAHS often results in decreased energy, loss of concentration,

poor job performance, and reduced social interaction. In addition, the loud snoring that often accompanies OSAHS may result in poor sleep for the sufferer's bed partner. An AHI \geq 5 has been associated with concentration difficulty but not memory tasks on self-assessment exams.¹⁶ OSAHS patients demonstrate significant improvement in daytime sleepiness,¹⁷ and in numerous quality-of-life parameters including physical functioning, social functioning, vitality, and general health perception after treatment.¹⁸ The relationship between sleepiness and level of AHI is not well defined, especially in older populations, with many people with AHI \geq 5 reporting minimal or no daytime sleepiness on self-reported examination.¹⁹

Cardiovascular morbidity and mortality. Evidence supports an increase in cardiovascular mortality five years after diagnosis in untreated patients with severe OSAHS patients compared with treated patients.²⁰ Another study found that men under age 60 with snoring and excessive daytime sleepiness were twice as likely to die over a 10-year study period compared with subjects without snoring or snoring without sleepiness.²¹ Two large prospective studies suggest that untreated, loud, habitual snorers have a 30-40 percent greater risk of myocardial infarction or stroke relative to nonsnorers.²² In contrast, a study by Jennum et al., which contained greater numbers of older individuals (age 54-74 years), failed to demonstrate a significant association between untreated snoring and cardiovascular morbidity or mortality.²³

Therefore, it has been hypothesized that the relationship between OSAHS and cardiovascular morbidity and mortality may be stronger in younger OSAHS patients compared with older patients.

Hypertension. It has been hypothesized that systemic arterial hypertension is the cause of the increased cardiovascular morbidity and mortality observed in patients with sleep-disordered breathing.²⁴ Laboratory evidence has demonstrated that sustained arterial hypertension can be induced in animal models subjected to intermittent airway occlusion during sleep.²⁵ The current epidemiological evidence shows a strong and consistent association between OSAHS and hypertension. Four large population-based cross-sectional studies found that the odds of hypertension were 1.4 to 2.5 times greater in patients with an AHI \geq 5 compared with controls.²⁶

A prospective analysis of the Wisconsin Sleep Cohort study found that even minimal elevation in AHI scores was associated with a 42 percent increased risk of developing hypertension over a four-year period.²⁷ Currently it is unclear the degree to which blood pressure can be lowered by treatment of OSAHS with CPAP or other methods.²⁸

Motor vehicle accidents. Epidemiological and laboratory evidence both suggest that patients with OSAHS are at greater risk for motor vehicle accidents (MVA). Patients with an AHI \geq 15 were 7.3 times more likely to have had multiple MVAs in the five years before their study, compared with those with lower or no apnea.²⁹ Hospitalized MVA victims were found to be 6.3 times more likely to have an AHI \geq 5 than community controls.³⁰ In addition, patients with severe OSAHS performed significantly worse on a driving simulator than controls without OSAHS.³¹

All patients \geq 65 years should be screened by history and physical examination for sleep-disordered breathing given the extensive prevalence of at least mild apnea (AHI \leq 15) in this age group and the severe potential health and quality-of-life consequences of undiagnosed sleep apnea. The evidence supports treatment of OSAHS in individuals with the following findings:

- AHI ≥ 5 with excessive daytime sleepiness
- AHI ≥ 5 with cardiovascular co-morbidities
- AHI \geq 15 with or without excessive daytime sleepiness

Patients with an AHI between 5 and 15 who do not have excessive daytime sleepiness are likely to be at low risk for cardiovascular sequelae and unlikely to be compliant with therapy, because they have no self-perceived sleepiness. Treatment selection depends largely on the level of sleep apnea, presence of medical co-morbidities, presence of anatomical deformities, and patient preference.

Quiz



1. Common primary sleep disorders in older adults include all of the following EXCEPT:

- a. Obstructive sleep apnea-hypopnea syndrome b.. Cataplexy c. Insomnia
- c. Insomnia
- d. Sleep maintenance insomnia e. Restless leg syndrome
- 2. Changes in sleep pattern with age include which of the following:
 - a. Increased sleep requirement b. Improved sleep efficiency c. Fewer nocturnal arousals d. Higher rates of insomnia e. Less need for napping

3. Which of the following statements is supported by current evidence:

a. OSAHS is a major cause of systemic hypertension.
b. OSAHS is a major cause of memory loss in older individuals .
c. Treatment of OSAHS significantly improves self-perceived quality of life.
d. The association between cardiovascular mortality and OSAHS is stronger in older individuals.
e. The treatment of OSAHS significantly lowers systemic high blood pressures.

Answers:

1. 2. 3.

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References

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1. Davies RJO, Stradling JR. The epidemiology of sleep apnea. Thorax. 1996;51:S65-S70.

2. See the following articles: Bixler E, Vgontzas A, ten Have T, Tyson K, Kales A. Effects of age on sleep apnea in men. Am J Respir Crit Care Med. 1998;157:144-148; Bixler E, Vgontzas A, Lin H, Ten Have T, Rein J, Vela-Bueno A, Kales A. Prevalence of sleep-disordered breathing in women. Am J Respir Crit Care Med. 2001;163:608-613; Duran J, Esnaola S, Rubio R, Iztueta A. Obstructive sleep apnea-hypopnea and related clinical features in a population-based sample of subjects aged 30 to 70 yr. Am J Respir Crit Care Med. 2001;163:685-689; and Duran CJ. Prevalence of obstructive sleep apnea-hypopnea and related clinical features in the elderly: A population-based study in the general population aged 71-100. World Conference 2001 Sleep Odyssey, October 21-26 2001, Montevideo, Uruguay.

3. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea. Am J Respir Crit Care Med. 2002;165:1217-1239.

4. Young T, Finn L, Austin D, Peterson A. Menopausal status and sleep-disordered breathing in the Wisconsin Sleep Cohort Study. Am J Respir Crit Care Med. 2003;167:1181-1185.

5. Klawe JJ, Tafil-Klawe M. Age-related response of the genioglossus muscle EMG-activity to hypoxia in humans. J Physiol Pharmacol. 2003;54:14-19. 6. See Young, Finn, Peterson, 2003, note 4.

7. See Young, Peppard, Gottlieb, 2002, note 3.

8. Young T, Shahar E, Nieto FJ, et al. Predictors of sleep-disordered breathing in community dwelling adults: The Sleep Heart Health Study. Arch Intern Med. 2002;162:893-900. Also see Bixler et al., 1998, and Bixler et al., 2001, note 2.

9. See Young, Peppard, Gottlieb, 2002, note 3.

10. Piani A, Brontini S, Dolso P, et al. Sleep disturbances in elderly: A subjective evaluation over 65. Arch Gerontol Geriatr Suppl. 2004;9:325-331.

11. Cauter EV, Leproult R, Plat L. Age-related changes in slow wave sleep and REM sleep in relationship with growth hormone and cortisone levels in healthy men. JAMA. 2000;284:861-868.

12. Boselli M, Parrino L, Smeireri A, Terzano MG. Effect of age on EEG arousals in normal sleep. Sleep. 1998;21:351-357.

13. Foley D, Ancoli-Israel S, Britz P, Walsh J. Sleep disturbances and chronic disease in older adults: Results of the 2003 National Sleep Foundation Sleep in America Survey. J Psychosom Res. 2004;56:497-502.

14. Bliwise DL. Sleep in normal aging and dementia. Sleep. 1993;16:40-81.

15. Barthlen GM. Obstructive sleep apnea syndrome, restless legs syndrome, and insomnia in geriatric patients. Geriatrics. 2002;57:34-39.

16. Jennum P, Sjol A. Self-assessed cognitive function in snorers and sleep-apneics: An epidemiological study of 1504 females and males age 30-60 years. The Dan-MONI-CA II Study. Eur Neurol. 1994;34:204-208.

17. Ballester E, Badia JR, Hernandez L, et al. Evidence of the effectiveness of continuous positive airway pressure in the treatment of sleep apnea/ hypopnea syndrome. Am J Respir Crit Care Med. 1990;159:495-501. Also see Engelman H, Kingshott RN, Wraith PK, et al. Randomized-placebo controlled crossover trial of continuous positive airway pressure for mil sleep apnea/ hypopnea syndrome. Am J Respir Crit Care Med. 1999;159:461-467.

18. Pichel F, Zamarron C, Magan F, et al. Health-related quality of life in patients with obstructive sleep apnea: Effects of long-term positive airway pressure treatment. Respir Med. 2004;98:968-976.

19. See Young, Peppard, Gottlieb, 2002, note 3.

20. He J, Kryger MH, Zorick FJ, et al. Mortality and apnea index in obstructive sleep apnea: Experience in 385 male patients. Chest. 1988;94:9-14. Also see Partinen M, Jamieson A, Guilleminault C. Long-term outcome for obstructive sleep apnea syndrome patients: Mortality. Chest. 1988;94:1200-1204. 21. Lindberg E, Christer J, Svardsudd K, et al. Increased mortality among sleepy snorers: A prospective population based study. Thorax. 1998;53:631-637.

22. Hu FB, Willett WC, Manson JE, et al. Snoring and the risk of cardiovascular disease in women. J Am Coll Cardiol. 2000;35:308-313. Also see Koskenvuo M, Kaprio J, Telakivi T, et al. Snoring as a risk factor for ischemic heart disease and stroke in men. Br Med J. 1987;294:16-19.

23. Jennum P, Hein HO, Suadicani P, Gyntelberg F. Risk of ischemic heart disease in self-reported snorers. Chest. 1995;108:138-142.

24. Lavie P, Herer P, Peled R, et al. Mortality in sleep apnea patients: A multivariate analysis of risk factors. Sleep. 1995;18:149-157.

25. Tilkian AG, Guilleminault C, Schroeder JS, et al. Hemodynamics in sleep-induced apnea: Studies during wakefulness and in sleep. Ann Intern Med. 1976;85:714-719.

26. See Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, Sleep apnea, and hypertension in a large community-based study. JAMA. 2000;283:1829-1836; Bixler EO, Vgontzas AN, Lin HM, et al. Association of hypertension and sleep-disordered breathing. Arch Intern Med. 2000;160:2289-2295; Young TE, Peppard P, Palta M, et al. Population-based study of sleep-disordered breathing as a risk factor for hypertension. Arch Intern Med. 1997;157:1746-1752; and Duran et al., 2001, note 2. 27. Peppard PE, Young TE, Palta M, et al. Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med. 2000;342:1378-1384. See also Peppard PE, Young T. Sleepdisordered breathing and hypertension: Reply. N Engl J Med. 2000;343:967.

28. See Young, Peppard, Gottlieb, 2002, note 3.

29. Young TB, Blustein J, Finn L, Palta M. Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. Sleep. 1997;20:608-613.

30. See Davies and Stradling, 1996, note 1; Young, Peppard, Gottlieb, 2002, note 3; and Young et al., 1997, note 29. Also see Teran-Santos J, Jimenez-Gomez A, Cordero-Guevara J. The association between sleep apnea and the risk of traffic accidents. Cooperative Group Burgos-Santander. N Engl J Med. 1999;340:847-851.

31. Findley FJ, Fabrizio MJ, Knigth H, et al. Driving simulator performance in patients with sleep apnea. Am Rev Respir Dis. 1989;140:529-530.

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