

# Epidemiology of Obstructive Sleep Apnea Syndrome

Markku Partinen and Tiina Telakivi

*Department of Neurology, Kivelä Hospital, SF-00260 Helsinki, and  
Ullanlinna Sleep Disorders Clinic and Research Center, Helsinki, Finland*

**Summary:** Obstructive sleep apnea syndrome (OSAS) is the most common organic disorder of excessive daytime somnolence. In cross-sectional studies the minimum prevalence of OSAS among adult men is about one per cent. Prevalence is highest among men aged 40–65 years. The highest figures for this age group indicate that their prevalence of clinically significant OSAS may be 8.5% or higher. Habitual snoring is the most common symptom of OSAS (70–95%). The most significant risk factor for OSAS is obesity, especially upper body obesity. Other risk factors for snoring, and for OSAS, are male gender, age between 40 and 65 years, cigarette smoking, use of alcohol, and poor physical fitness.

Upper airway obstruction with snoring or sleep apnea are commonly seen in children of all ages. Snoring is very common among infants and children with Pierre Robin syndrome and among infants with nasal obstruction. Snoring and obstructive sleep apnea are also very common in men with acromegaly. Many other syndromes or diseases exist in which the upper airway is narrowed. Prevalence of snoring and sleep apnea is increased in all such situations.

It has been suggested that sleep apnea may be one mechanism contributing to sleep-related mortality. The prevalence of every night snoring seems to decrease after the age of 65. However, more than 25% of persons over 65 have more than five apneas per hour of sleep. It remains to be seen whether this finding has clinical significance.

Partial upper airway obstruction, even without apneas, may influence pulmonary arterial pressure and may cause daytime sleepiness and some health consequences. On the other hand, many healthy 60-year-olds have been snoring since childhood. Nightly snoring and sleep apnea are potential determinants of cardiovascular and cerebrovascular risk. The prevalence of sleep apnea among patients with essential hypertension is over 25%. Thus patients with arterial hypertension should always be queried about snoring history and possible sleep apnea. Snoring should be measured quantitatively in patients with indices such as the Basic Nordic Sleep Questionnaire. The quality of snoring should also be determined. Questioning about snoring and other symptoms of OSAS (with the right questions) combined with clinical examination is a good "screening method" for OSAS.

Obstructive sleep apnea (OSA) is characterized by repetitive apneas during sleep. Obstructive sleep apnea syndrome (OSAS) is defined by intermittent complete or partial upper airway obstruction during sleep, causing mental and physical effects. The main nocturnal symptoms of OSAS are loud and irregular snoring, breathing pauses, restless sleep and daytime sleepiness. Other symptoms include morning headache, irritability, problems of memory, nocturnal polyuria and excessive sweating at night. It must be noted that presence of sleep apnea (without mental and/or physical effects) is not equivalent to OSAS (1).

## PREVALENCE

Lavie in Israel (Table 1) has estimated in a cross-sectional study that the prevalence of the syndrome

among male industrial workers is at least 1% (2). In an epidemiologic study performed in Bologna, Italy, the authors estimated a minimum prevalence in this population of 2.7%, based on prevalence of nightly snoring and polysomnographic results, with an apnea-hypopnea index of 10 or more considered pathological. These figures are larger than those found in Scandinavian studies (3,4). The highest figure (8.5%) is from a recent Australian study by Bearpark and her collaborators (5). Their figures give a good estimate of OSA among Australian men but do not indicate how many of their subjects are symptomatic. Thus, further studies are needed to know the prevalence of symptomatic OSAS in Australia. Genetic differences may explain some of the differences in the figures.

Forty to 60% of patients with obstructive sleep apnea have arterial hypertension. In a case-controlled study, 30% of the hypertensive patients and none of the control patients had sleep apnea syndrome (6). This finding of a high prevalence of sleep apnea in hypertensive patients has been confirmed by other groups as well (7–9).

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Address correspondence and reprint requests to Markku Partinen, M.D., Department of Neurology, Kivelä Hospital, SF-00260 Helsinki, Finland.

**TABLE 1.** Prevalence (%) of obstructive sleep apnea syndrome (OSAS) in men

Author	Size	Age	Prevalence
Lavie, 1983 Israel	1,262	18-67	1.0-5.9
Peter et al., 1985 Germany	354	25-55	2.3
Telakivi et al., 1987 Finland	1,939	30-69	0.4-1.4
Gislason et al., 1988 Sweden	3,201	30-69	0.7-1.9
Cirignotta et al., 1989 Italy	355	30-39	0.2-1.0
	738	40-59	3.4-5.0
	417	60-69	0.5-1.1
Bearpark et al., 1991 Australia	309	40-65	8.5

Because of the high prevalence of sleep apnea in the elderly, sleep apnea has been suggested as one mechanism contributing to sleep-related mortality. The prevalence of nightly snoring seems to decrease after the age of 65. However, more than 25% of persons over 65 have more than 5 apneas per hour of sleep (10). This does not mean that 25% of them have sleep apnea syndrome. It remains to be seen whether the high frequency of sleep apnea among elderly people has clinical significance. In one study, a cohort of 198 noninstitutionalized elderly individuals (mean age at entry 66 years) were followed for periods up to 12 years after initial polysomnography (11). The mortality ratio for sleep apnea, defined as a respiratory disturbance index of over 10 events per sleep hour, was 2.7 (95% confidence interval 0.95-7.47). These results raise the possibility that "natural" death during sleep in the elderly may be associated with disordered breathing during sleep or with some other pathological events during sleep.

Snoring and sleep apnea are common in some specific groups of subjects, such as infants and children with Pierre Robin syndrome or infants with nasal obstruction. Snoring and obstructive sleep apnea are also very common in men with acromegaly. Many other syndromes or diseases exist in which the upper airway is narrowed. Prevalence of snoring and sleep apnea is increased in all such situations.

### OBSTRUCTIVE SLEEP APNEA AND CARDIOVASCULAR DISEASE

During obstructive apnea, pulmonary and systemic arterial pressures rise, straining the heart and especially its right side. The combination of bradycardia and hypoxemia increases the risk of arrhythmias, which are, in fact, rather common in patients with sleep apnea.

Cyclic variation of heart rate with repetitively occurring bradycardia (during apnea) and tachycardia (after each apnea) is a typical finding in OSAS patients. In a cross-sectional study at Stanford, 48% of the 400 patients had some kind of cardiac arrhythmia during the night (12). Increased frequency of ventricular ectopia has been observed in many other studies as well.

There are only a few reports on the incidence of myocardial infarction and the prevalence of coronary heart disease among patients with OSAS. Thirteen (76%) out of 17 patients with CHD had disordered breathing during sleep (13). In another study, five out of 13 patients with myocardial infarction and one of 13 controls exhibited a pathological number of apneas (14). A larger case-control study was done in Australia by Hung et al. (15), who investigated 101 male patients with myocardial infarction and 53 male controls. Men with >5.3 apneas per hour of sleep had 23.3 times (95%; CI 3.9-139.9) the risk of myocardial infarction of men with <0.4 apneas per hour of sleep. The association was independent of age, body mass index, arterial hypertension, smoking and cholesterol level. There was an increase in adjusted risk of myocardial infarction with increasing levels of sleep apnea (15).

An association between cerebral infarction and habitual snoring has been found (16,17). The odds ratio of brain infarction is 2.8 between self-rated frequent or habitual snorers and self-rated occasional or non-snorers. Between habitual snorers and occasional or nonsnorers the odds ratio of stroke may be as high as 10. Habitual snorers have a greater risk of cerebral infarction during sleep or early in the morning. However, their risk of cerebral infarction when awake during the day is not significantly different from the risk of nonsnorers (17).

In some studies, occurrence of sleep apneas tended to be more common in patients with multi-infarction dementia than in patients with Alzheimer's disease or aged controls (18,19). Sleep apneas may play some role in the development of multi-infarction dementia, whereas in Alzheimer's disease, and in normal aging, apneas are perhaps a consequence of central nervous system degeneration.

### EVOLUTION OF OBSTRUCTIVE SLEEP APNEA SYNDROME

Death during the night has been related to OSAS, but the morbidity and mortality associated with OSAS are still rather unclear. The modern treatments of OSAS include nasal continuous positive airway pressure (CPAP), several kinds of upper airway surgery, and controlled weight loss using behavioral techniques. Tracheostomy is used currently if other treatments do not work well, or in case of emergency. Tracheostomy

was the first efficient treatment, and the longest follow-ups thus concern patients treated with tracheostomy. Preliminary results from different sleep clinics suggests that nasal CPAP is about as effective as tracheostomy.

He et al. (20) found that those with an apnea index (AI) >20 had a greater mortality than those with AI ≤ 20. The probability of cumulative eight-year survival was  $0.96 \pm 0.02$  (SE) for AI ≤ 20 vs.  $0.63 \pm 0.17$  for AI > 20 ( $p < 0.05$ ). None of the patients treated with tracheostomy or nasal CPAP died during the follow-up. Eight of the 98 patients with uvulopalatopharyngoplasty (UPPP) died, and the cumulative survival of the group treated with UPPP-alone was not different from the survival of untreated patients with an AI > 20. Twenty of the 98 patients with UPPP did not have polysomnography after operation and six of them (30%) died.

At Stanford (21) the follow-up population consisted of 198 patients with a median age of 52 years. At entry, 112 patients (56.6%) presented with arterial hypertension. No statistically significant difference existed at entry for frequency of hypertension, coronary heart disease, cerebrovascular disease or chronic obstructive pulmonary disease between the tracheostomy and conservative treatment groups.

At 5-year follow-up, the mortality rate of the conservatively treated was 11 per 100 patients per 5 years. These patients also had a higher 5-year cardiovascular (CV) mortality rate: 8 per 100 patients per 5 years [95% confidence interval (CI) 1.1–15.0]. The age-standardized crude cardiovascular mortality rate was 5.9 per 100 patients per 5 years (95% CI 2.5–11.6) vs. 0 per 100 for the tracheotomized population (21). With a fictional adjunction of one possible death at five-year follow-up among the tracheotomized, the conservatively treated group had a five-year age-adjusted odds ratio of 4.7 for vascular death.

From the same Stanford population, 196 patients have been followed for up to 11 years (126 conservatively treated and 70 tracheotomized). The difference is statistically significant. The cardiovascular 11-year mortality of the conservatively treated was 13% (22,23).

Two hundred patients with OSAS seen at Montefiore between 1977–1982 have been followed for at least 5 years (24,25). Survival at 5 years for the tracheotomized was 96% and for the control group 92%. The difference is statistically not significant. At 10 years the survival was 88% vs. 76% (NS). There were 14 cardiovascular deaths (two among the tracheotomized). Their results support the view that active treatment of OSAS decreases cardiovascular mortality of OSAS patients. In addition, tracheostomy has a marked effect on daytime sleepiness. Active treatment reduces cardiovascular mortality, but it seems to be even more

efficient in reducing daytime sleepiness of the patients with OSAS.

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