

Is Snoring a Health Risk?

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What is Snoring?

Snoring, one of the most prevalent obnoxious human habits, can be a social nuisance and may well be a medical problem. No one who has slept beside a snorer needs any description of what is snoring, or any convincing that a remedy would be very much welcome. Snoring is a sound produced during sleep by vibrations of the soft tissues of the pharyngeal walls, soft palate and the uvula¹. Although snoring is generally thought to be an inspiratory noise, recent studies showed that in chronic heavy snorers, an expiratory noise can be recorded and audible as well². It has been suggested that the sounds of snoring are caused by three mechanisms: (1) floppy pharyngeal muscles with poor tone; (2) airflow limitation in the nose; and (3) space-occupying masses in the airway, including enlarged tonsils or tumours^{3,4}.

During normal sleep the tone of the pharyngeal airway dilator muscles, the genioglossus, levator palati, and palatopharyngeus is decreased with resultant partial upper airway collapse and narrowing of the lumen giving rise to turbulent flow^{5,6}. The turbulence leads to vibration of the soft tissues in the collapsible part of the upper airway where there is no rigid support. This extends from the choanae to the epiglottis and involves the soft palate, the uvula, the tonsils, the tonsillar pillars, the tongue base and the muscles of the pharynx with their overlying mucosa. Even in normal, non-snoring individuals upper airway narrowing increases the resistance to airflow at sleep onset⁷. The nose accounts for approximately half of the total airway resistance to airflow and a semi-blocked nose requires increased inspiratory effort; this lowers intrapharyngeal pressures and tends to suck the pharyngeal walls together causing increased snoring⁸. Many patients experience a worsening of their snoring when they

suffer from an upper respiratory tract infection or rhinitis.

Who Snores?

Snoring is extremely common and has been estimated to occur in 19% to 37% of the general population⁹. It is more common in men than women at all ages¹. The prevalence of snoring increases with age and at the age of 60 years 60% of men and 40% of women snore⁹. Snoring is more common in the obese than in those of normal body weight¹. Nasal obstruction and increased nasal resistance may aggravate snoring by increasing the negative intrapharyngeal pressure and facilitating the conditions leading to inspiratory flow limitation and fluttering of the upper airway walls¹. Consumption of alcohol, sedatives and antihistamines may contribute to, or even cause snoring.

Snoring to some extent, is hereditary. In one study¹⁰ there is a strong association between self-reported habitual snoring and family history of snoring among grandparents, parents, siblings and children. As the majority of individuals who snore are unaware of their snoring¹¹, questions about snoring should be answered by the bed partner or other cohabiting relatives and not by the snorer.

The Clinical Significance of Snoring

Is snoring merely an acoustic nuisance? Snoring and the disturbance it causes is frequently regarded as a joke. That a large number of people snore would not be a reason for concern except that there have been reports suggesting the possible relationship between snoring and increased cardiovascular morbidity and mortality. For many years it was thought that the only danger of

snoring is to the listener, who is kept awake and irritated by the disruptive noise produced by the snorer. Snoring itself was considered harmless and without any adverse consequences to the snorer. We now know that snoring may be a pointer to abnormalities with breathing at night which may be harmful to the health of the snorer. When sleep apnoea was described more than two decades ago¹², snoring suddenly emerged as an important symptom of a disorder with potentially serious consequences. Obstructed breathing during sleep may be viewed as a continuum - from pure snoring to severe obstructive sleep apnoea with daytime respiratory failure and cor pulmonale. The most benign extremity of this continuum is represented by pure snoring. A step further is the occurrence of occasional complete pharyngeal collapse which causes obstructive apnoea which is the cessation of airflow despite persistent inspiratory effort. When these apnoeic episodes occur repeatedly throughout the night during sleep, the full blown sleep apnoea syndrome is constituted. The sleep apnoea syndrome is the occurrence of repetitive apnoeas during sleep associated with daytime somnolence. Obstructive sleep apnoea is usually preceded by years of snoring¹³. For many individuals, snoring represents an intermediate state in the continuum between normal subjects and patients with full blown sleep apnoea syndrome. Habitual snoring is the most common symptom of obstructive sleep apnoea syndrome (OSAS), being present in 70% to 95% of such patients¹⁴. The most significant risk factor for OSAS is obesity, especially upper body obesity^{15,16}. Other risk factors for OSAS include male gender, age between 40 and 65 years, cigarette smoking and alcohol consumption.

In obstructive sleep apnoea, snoring is cyclical, with loud snoring noises alternating with periods of silence when the snorer struggles to reopen his/her airway. Finally, a loud snort - the so-called 'resuscitative snort' - occurs and the patient's airway is opened and breathing resumes. The boundary between so-called benign snoring and snoring associated with obstructive sleep apnoea syndrome is not very distinct. While non-apnoeic snoring is associated with circumferential narrowing of the velopharyngeal lumen during inspiration, there is inspiratory collapse in the velopharyngeal or oro/hypopharyngeal region in apnoeic snoring.

Non-apnoeic snoring or continuous snoring is thought to be of little consequence because it is associated with few measurable cardiorespiratory changes¹⁷. However, heavy snoring without classical sleep apnoea may cause sleep fragmentation, as evidenced by recurrent transient electroencephalographic (EEG) arousals, related to increased respiratory effort in response to raised upper airway resistance.^{2,18} The fragmentation of sleep consequent upon these EEG arousals results in daytime symptoms of tiredness and drowsiness with diminished alertness and performance⁵. Excessive daytime sleepiness is recognised as an important cause of motor vehicle¹⁹ and industrial accidents, decreased productivity, interpersonal difficulties, and cognitive dysfunction with memory and concentration problems¹.

Patients with untreated obstructive sleep apnoea have increased morbidity and mortality from vascular causes²⁰⁻²². Successful treatment of patients with obstructive sleep apnoea syndrome has been shown to reduce morbidity and mortality due to cardiovascular causes^{21,23}. Why is snoring implicated in the pathogenesis of vascular disease? The reason is the association between snoring and partial upper airway obstruction that leads to hypoventilation, hypoxaemia, more negative intrathoracic pressure, and arousals from sleep. All these factors lead to increased sympathetic activity during sleep. It is logical to postulate that these transient periodic increases in nocturnal sympathetic activity present for many years will eventually lead to sustained daytime elevation in blood pressure, episodic spasm of the coronary arteries and therefore predisposition to vascular disease - hypertension, angina pectoris and stroke.

The Association Between Snoring and Hypertension

Several epidemiological studies have shown that the prevalence of arterial hypertension is increased in snorers^{9,24,25}. Since snoring is a very common symptom of obstructive sleep apnoea,²⁶ and the association between hypertension and obstructive sleep apnoea is well established,²⁷⁻³¹ a direct relationship between snoring alone and hypertension has been questioned³². Furthermore, patients with snoring and obstructive sleep apnoea are often overweight, a condition that is

also known to be associated with arterial hypertension. Although arousals induced by snoring can cause systemic blood pressure elevations similar to those of obstructive sleep apnoea,³³ these elevations are transient and there is no evidence that they lead to sustained daytime hypertension¹.

The evidence published so far does not support a direct link between snoring and hypertension. In a review³⁴ of 19 studies that were published in 1980 to 1995, almost all the studies reported a higher prevalence of hypertension among snorers than among nonsnorers. When confounding factors such as age, obesity, smoking, and alcohol consumption were taken into account, 14 of the studies did not find snoring to be an independent risk factor for hypertension. Even in the 5 so-called positive studies, doubt was raised whether there was an independent relationship between snoring and hypertension because either confounding variables were not taken into account, study variables were self-reported and not measured, or the sample population was contaminated by patients with sleep apnoea. The relationship between snoring and daytime hypertension can be explained by the confounding variables such as obesity, age, smoking, alcohol consumption and sleep apnoea; snoring is only an indirect determinant of blood pressure, via its association with such confounders³⁴.

Snoring and Cardiovascular and Cerebrovascular Morbidity and Mortality

The possible association of snoring with increased cardiovascular and cerebrovascular morbidity and mortality has been a topic of numerous investigations^{9,24,32,35-40} with seemingly divergent results, mainly because of differences in methodology employed in various studies, such as inclusion of confounding factors, type of population studied, methods of measurements and analysis, etc. In a recent review³⁴ of 15 studies

published in 1980 to 1995 that investigated the association between snoring and vascular disease (i.e. angina pectoris, ischaemic heart disease, myocardial infarction and stroke), 9 studies concluded in favour of positive association and 6 studies concluded against it. A detailed review of the studies showing a positive association between snoring and vascular disease revealed that in some of the studies unsuspected sleep apnoea might have accounted for the increased risk. Since snoring is very frequently present in patients with sleep apnoea, and since in none of the studies linking snoring with coronary artery and cerebrovascular disease was nocturnal polysomnography performed, there is a possibility that the observed association between snoring and coronary and cerebrovascular disease is due to sleep apnoea. Without nocturnal monitoring one is never entirely certain whether the effect attributed to snoring is in fact due to snoring or to unrecognised sleep apnoea. Several confounding factors (age, gender, systemic hypertension, obesity, smoking, alcohol consumption) can be involved in the association between snoring and stroke because many potential risk factors for cerebral infarction are also associated with snoring and sleep apnoea syndrome. None of three prospective studies⁴¹⁻⁴³ where snorers and nonsnorers were followed for 5 or 6 years found an independent association between snoring and vascular disease.

Because snoring is such a cardinal symptom of obstructive sleep apnoea, it is sometimes difficult to distinguish the independent effect of snoring from that of apnoeas. There is no convincing evidence that non-apnoeic snoring is a risk factor for cardiovascular and cerebrovascular diseases¹. However, it is probably not totally correct to dismiss snoring as being only a social nuisance because it can be a precursor of obstructive sleep apnoea in those individuals who acquire other risk factors for this condition, and because, it may disrupt sleep and cause daytime dysfunction.

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MCQs on Is Snoring a Health Risk?

1. Snoring
 - A. is produced by vibration of the soft tissues of the upper airway
 - B. occurs in about a third of the general population
 - C. is more common in women
 - D. prevalence decreases with age
 - E. has been conclusively linked to increased cardiovascular and cerebrovascular morbidity and mortality

2. Snoring is aggravated by
 - A. weight gain
 - B. nasal decongestants
 - C. alcohol
 - D. sedatives
 - E. lying on the side

3. Features of obstructive sleep apnoea syndrome are
 - A. habitual smoking
 - B. non-refreshing sleep
 - C. daytime somnolence
 - D. lower body obesity
 - E. systemic hypertension

4. Excessive daytime sleepiness related to obstructive breathing during sleep
 - A. is due to sleep fragmentation as a result of recurrent arousals
 - B. does not occur in non-apnoeic loud snorers
 - C. predisposes to motor vehicle accidents
 - D. interferes with concentration
 - E. affects work performance

5. Nocturnal polysomnography
 - A. is the monitoring and recording of various physiological parameters during sleep
 - B. is usually performed in a hospital-based sleep laboratory
 - C. is a facility available in most Malaysian hospitals
 - D. detects sleep arousals by electroencephalographic changes
 - E. cannot differentiate non-apnoeic snorers from snorers with obstructive sleep apnoea