



CLINICAL REVIEW

Simple snoring: Not quite so simple after all?

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SUMMARY

Simple snoring (SS), in the absence of obstructive sleep apnoea (OSA), is a common problem, yet our understanding of its causes and consequences is incomplete. Our understanding is blurred by the lack of consistency in the definition of snoring, methods of assessment, and degree of concomitant complaints. Further, it remains contentious whether SS is independently associated with daytime sleepiness, or adverse health outcomes including cardiovascular disease and metabolic syndrome. Regardless of this lack of clarity, it is likely that SS exists on one end of a continuum, with OSA at its polar end. This possibility highlights the necessity of considering an otherwise ‘annoying’ complaint, as a serious risk factor for the development and progression of sleep apnoea, and consequent poor health outcomes. In this review, we: 1) highlight variation in prevalence estimates of snoring; 2) review the literature surrounding the distinctions between SS, upper airway resistance syndrome (UARS) and OSA; 3) present the risk factors for SS, in as far as it is distinguishable from UARS and OSA; and 4) describe common correlates of snoring, including cardiovascular disease, metabolic syndrome, and daytime sleepiness.

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Introduction

Snoring is a common problem, yet prevalence estimates vary widely. The lack of clarity around snoring prevalence is in part a reflection of unresolved issues concerning its definition(s). Most authors agree on what snoring is: “... a fluttering sound created by the vibrations of pharyngeal tissues ...” [1]; or more generally “... a sound produced by the upper aerodigestive tract during sleep ...” [2]. There is less agreement as to what counts as clinically significant snoring, as well as the nature of the distinctions, qualitative or quantitative, between simple snoring (SS), upper airway resistance syndrome (UARS) and obstructive sleep apnoea (OSA). This picture is further confused by the variety of actual and proposed assessment methods for each of these disorders, and the variety of putative physiological and anatomical correlates within each snoring classification. Finally, the degree of associated daytime dysfunction is often, but not consistently, used as a distinguishing diagnostic marker and this in turn has (confusing) implications for clinicians and treatment decisions.

Prevalence

In one of the largest prevalence studies to date, a Hungarian population survey of 12,643 people, 50% self-reported being loud snorers [3]. The sample was subdivided into loud and habitual snorers, and further by gender. Thirty-seven percent of males self-identified as being loud snorers with breathing pauses and 23% habitual snorers; whereas in females 21% identified as loud snorers with breathing pauses and 21% as habitual snorers. Several factors may influence prevalence variation across studies, and between nations, including socio-demographic characteristics of study populations; health behaviours and variation in assessment methods and classification categories. For these reasons, the authors, like other researchers, concluded that cross-nation comparisons are not possible. Prior prevalence estimates in other epidemiological studies varied between 2% and 85%, depending on measurements and population variables [4]. A similar large population based study of 4533 Latin Americans, reported a prevalence of ~60% in four Latin American cities, with approximately 10% higher prevalence in males than females [4]. There have been other recent international prevalence estimates. Adewole and colleagues estimate a 32% habitual snoring prevalence in a small sample of 370 adults in Nigeria [5]. In one of the first studies in a Pakistani population, Hussain and colleagues surveyed 2497 adults and reported

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Abbreviations

AHI	apnoea–hypopnea index
BMI	body mass index
CFI	craniofacial angle
CFS	chronic fatigue syndrome
CT	computerised tomography
EDS	excessive daytime sleepiness
MPH	mandibular plane and hyoid distance
OR	odds ratio
OSA	obstructive sleep apnoea
PAS	posterior airway space
PSG	polysomnography
REM	rapid eye movement
RERAs	respiratory event related arousals
SS	simple snoring
UARS	upper airway resistance syndrome

an average prevalence of self-reported snoring of 32%, yet in middle age (>35 y of age) prevalence was 46% [6]. In a sample of 8583 Japanese adults (35–79 y of age) prevalence rates were 24% for men and 10% for women [7]. This sex difference is perhaps not surprising given that being male is one of the risk factors for snoring (see section below). As well as there being prevalence variation between studies, there is also variation within studies which have looked at the ethnic mix of their populations. Among 1611 Malaysian adults with an overall habitual snoring prevalence of 47%, Indian and Chinese individuals were significantly more likely to report snoring than Malaysian [8]. Further, in a survey of 2298 adults of Indian, Chinese and Malaysian origin, for Indian individuals the odds of reporting snoring were 1.5 times greater than in Chinese individuals [9]. European estimates tend to be around 20–40% [1]. Possible reasons for ethnic differences in snoring will be considered when we discuss risk factors below.

A major limitation of these studies is the definition of habitual snoring. Hussain and colleagues distinguished between habitual and occasional snoring (5.4% vs. 26.9%) with the former being defined as “daily” [6]. Other studies have defined habitual snoring as “often”; or more than three, four or five times per week. This is highlighted in one of the few meta-analyses in the epidemiology of snoring [10]. This systemic review and meta-analysis of 63 studies reporting on gender differences in the prevalence of snoring identified significant methodological heterogeneity in population, age, sampling frames and assessment methods. Furthermore they found that ~62% of studies did not specify definitional criteria, and 81% did not ask about the loudness of snoring. As such estimates of snoring are somewhat piecemeal, and with continuing lack of internationally agreed definitions, classifications and measurement tools, are likely to remain so.

A continuum of snoring?

Most authors support a continuum of snoring from SS through UARS up to and through degrees of OSA (for example, see [11,12]). OSA has the clearest definition and diagnostic criteria. Objectively it is marked by partial or complete collapse of the upper airway during sleep which leads to total (apnoea) or substantial (hypopnea) decrease in inspiration which lasts for at least ten seconds [13]. The number of these events per hour – the apnoea/hypopnea index (AHI) – is taken as a measure of the severity of the condition. Conventionally people are classified as mild OSA if they have

between 5 and 15 events an hour, moderate if they have between 15 and 30, and severe if they have >30 [14], as measured by polysomnography (PSG). However, even within the relative objectivity of these criteria, there are still variations among researchers in diagnostic thresholds for airflow reduction, oxygen desaturation and cortical arousal [15].

Patients who snore but have an AHI less than five tend to be classed as primary or habitual snorers [16]. OSA is relatively rare compared to snoring, and is generally estimated to affect 2–4% of the population, though estimates suggest that at least 15% of snorers have an AHI >15 [17]. This large excess of snorers to OSA patients suggests that the vast majority of snorers are simple or non-apnoeic snorers (also variously called primary, habitual and socially disruptive snorers). One of the main concomitants of snoring is daytime sleepiness, which has been used as a diagnostic proxy, in the absence of PSG, for distinguishing OSA from SS. The justifying hypothesis is that disordered breathing in OSA disturbs sleep. However, as Svensson and colleagues [18] note, linkage between OSA and daytime sleepiness is not clear-cut. Many people with apnoea do not report daytime sleepiness, while many non-apnoeic snorers do. Guilleminault and colleagues [19] attempted to address some of these inconsistencies by suggesting that there is a distinct, third clinical entity between SS and OSA, marked by non-apnoeic, non-hypopnoeic changes in respiratory effort and associated cortical arousal – respiratory effort-related arousals (RERAs) – which are associated with daytime sleepiness. This postulated clinical entity, upper airway resistance syndrome (UARS) is still disputed [17] (see below for further discussion of UARS).

Given the blurred and disputed boundaries between SS, UARS and OSA, it is worth reviewing what is done in practice to distinguish and define them. Several criteria have been proposed.

Definitions and distinctions of snoring

Distinctions by anatomical and neurological markers

Several authors have studied the structure and ultra-structure of the palate, following the hypothesis that snoring is a marker of pathology or abnormality of upper aerodigestive tract anatomy. The most popular version of this theory is the obstructive theory of snoring, which hypothesises that hypertrophy of uvular and palatal structures causes narrowing and collapse of airways. Karakoc and colleagues attempted to distinguish 133 SS and 131 OSA patients on an anatomical basis, although found no difference between groups in nasal obstruction [16]. However, grouping patients according to their AHI category revealed significant differences in Fujita classification. The Fujita classification is a method of describing the location of any airway obstruction as seen by visual and endoscopic examination during sleep [20]. SS patients were much more likely to be classified as type 1 (upper pharyngeal) and OSA as type 2 (hypopharyngeal) (80% and 61%, respectively). Differences in AHI based on Mallampati classification – which approximates to the tongue size relative to palate and pharynx [21] – were also significant: the greater the relative tongue size, the higher the AHI category. Finally a measure of collapsibility of the pharyngeal walls was also positively and significantly related to AHI. All of this would suggest that there are at least quantitative differences between SS and OSA, although there was no normal control group for comparison. By contrast, in a battery of similar measures comparing 20 SS with 32 mild and 22 moderate OSA patients, Balsevicius and colleagues found only clinical assessment of tonsil size distinguished SS from the other two groups: SS had a higher Friedman's score of palatal tonsils [22]. In a radiographic (computed tomography (CT) scanning) comparison 34 SS patients had, predictably,

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