Editorial

Snoring, a Vexing Night Symphony or Clinical Red Flag?

Mohammad Nami[1,2,3]

[1]Department of Neuroscience, School of Advanced Medical Sciences and Technologies, Shiraz University of Medical Sciences, Shiraz, Iran
[2]Sleep Disorders Laboratory, Namazi Hospital, Shiraz University of Medical Sciences, Shiraz, Iran
[3]Shiraz Neuroscience Research Center, Shiraz University of Medical Sciences, Shiraz, Iran

Corresponding author:
MT Nami
Department of Neuroscience, School of Advanced Medical Sciences and Technologies, Shiraz University of Medical Sciences, Shiraz, Iran
torabinami@sums.ac.ir

Received: 18.06.2016
Accepted: 01.07.2016

Keywords: Sleep-disordered breathing, Snoring, Apnea, Sleep medicine

Abstract

Sleep disordered breathing (SDB) is widely known to negatively affect productivity due to the resultant sleepiness. Meanwhile, the significance of snoring, upper airway resistances and sleep apnea has not been well appreciated by the general public. There has not been enough understanding about this common symptom as a major cause of various cardiovascular or neurological diseases. To properly diagnose SDB, the understanding of current development in technology and devices in the field is deemed mandatory. This brief perspective paper has been an attempt to touch on the significance of snoring as a clinically noticeable symptom especially when becomes intense and persistent. The negative impact of SDB on the so-called lung-brain axis has turned to an evolving area of experimental research in the field of sleep medicine.

https://doi.org/10.18869/nrip.jamsat.2.2.197

Snoring results from airflow-induced flutter of soft tissues of the nasopharynx, particularly the soft palate. As in any fluttering physical structure, flutter in the nasopharynx develops depending on interacting factors, including the mass, stiffness, and attachments of the fluttering element and the velocity and direction of airflow. The fact that people do not snore while awake suggests that sleep-induced muscular relaxation is at least part of the etiology because muscle tone is the only component of flutter that can change during sleep, where tissue mass and attachments do not change. Furthermore, if pharyngeal dilators cannot keep the airway open in response to the negative intraluminal pressure induced by inspiration, the upper airway narrows and increases the local airflow velocity (for a given inspiratory volume). The increased flow velocity promotes flutter directly and decreases intraluminal pressure, further enhancing airway closure and thus promoting flutter and snoring (1).

Primary snoring is snoring that is not accompanied by awakening or excessive arousals, limitation of airflow, oxygen desaturation, or arrhythmias during sleep which occurs in people who do not have excessive daytime sleepiness (EDS). Arousals are brief transitions to lighter sleep or awakenings that last < 15 sec and are usually not noticed (2).

If we snore loud, we might assume that our partner or roommate is perhaps the one mostly affected by the vexing night-time symphony and he/she would keep awake all night long for the noise we make while we, for the most part, remain blissfully rested and unaware.

The truth can however be far different. Snoring is way beyond just a bother to someone else resting

next by, and may be regarded as a serious red flag raised for ones health. As a possible sign of upper airway resistance or associated pauses in breathing upon sleep, the condition may give rise to frequent drops in blood oxygen saturation during sleep and subsequent daytime symptoms including an increased risk for heart diseases and stroke (3).

Fluctuating oxygen levels throughout the night causes stress and oxidative damage to cells within the body. They also force the brain to be on high-alert all night and to deliver a rush of adrenaline to the heart every time an apnea occurs, when the body and brain are ideally supposed to be resting and recovering (4).

In fact, relaxation of the muscles in the back of the throat and roof of the mouth partially or even totally block the airflow when we are asleep. Wheezing, snorting and snuffling at the middle of the night can happen for a different reasons, while they all have to do with narrowing or blockade of a person's airway (5).

This may happen more frequently when we sleep on our backs instead of our sides, or we have nasal congestion due to allergies or a cold. Though snoring can be a detrimental sign, it should be noted that one out of two adults habitually snore which is usually considered not dangerous. When snoring turns to a medical concern, daytime symptoms including daytime sleepiness, morning headaches, fatigues, muscle soreness and neurocognitive lapses may become evident (5).

A key question encountered in the practice of sleep medicine is “when to take snoring seriously”? When we have run-of-the-mill snoring heard by others on a regular basis or periodically witnessed stops in breathing for several seconds at a time, it can be the red flag mentioned above.

Risk factors for significant snoring and possible obstructive sleep apnea-hypopnea syndrome (OSAHS) are older age, obesity, use of alcohol or other sedatives, chronic nasal congestion or blockage, a small or posteriorly displaced jaw, male sex, postmenopausal status, pregnancy and abnormal structures which can block airflow (for instance large tonsils, a deviated nasal septum or nasal polyps).The clinical evaluation is not completely reliable for diagnosis of OSAHS but can be suggestive. Red flag findings clearly correlate with OSAHS. However, all of these findings occur along a continuum, and there is no widespread agreement on cut-off points and relative weighting. Nonetheless, the more red flag findings a patient has and the more severe they are, the greater is the likelihood of OSAHS (5).

OSAHS is quite treatable and accurately diagnosed through an overnight sleep test done either at home or a sleep laboratory (5). Lifestyle modifications including weight loss or avoid sleeping on the back, may help some patients. If not, many cases may get sufficiently treated using a device called “continuous positive-air pressure”, or CPAP machine. This smart device is designed to send air through a tube and a mask, into a patient's nose and mouth while he/she is a sleep, keeping the airway patent (6).

Treating any nasal congestion (e.g. with decongestant and/or corticosteroid sprays or with elastic strips that hold the nares open) may help some patients. Other measures include the use of oral appliances (including mandibular advancement devices), sleep position trainers, etc. (3).

Typical clinical risk factors such as loud snoring and gasping sounds, witnessed sleep apneas, daytime sleepiness and obesity help identify patients at risk of OSAHS who are therefore in need for sleep testing (polysomnography). While only some snorers have OSAHS, most patients who have OSAHS snore. When our snore is found to be clinically significant by our physician (preferably sleep specialists), a set of recommendations need to be followed.

General measures to follow to reduce snoring include avoiding alcohol and sedating drugs, sleeping with the head elevated and losing weight. Specific measures including but not limited to mandibular advancement devices, uvulopalatopharyngoplasty (throat surgery), palate-altering procedures, and CPAP to treat snoring due to OSAHS may be considered by the treating physicians (7).

References

1. *Acar M, Yazici D, Bayar Muluk N, Hanci D, Seren E, Cingi C. Is There a Relationship Between Snoring Sound Intensity and Frequency and OSAS Severity? The Annals of otology, rhinology, and laryngology. 2016;125(1):31-6.*
2. *Kalchiem-Dekel O, Westreich R, Regev A, Novack V, Goldberg M, Maimon N. Snoring intensity and excessive daytime sleepiness in subjects without obstructive sleep apnea. The Laryngoscope. 2016.*
3. *Nami MT. Evaluation of the sleepy patient. Australian family physician. 2012;41(10):787-90.*
4. *Jahn C, Gouveris H, Matthias C. Systemic inflammation in patients with compromised upper airway anatomy and primary snoring or mild obstructive sleep apnea. European archives of oto-rhino-laryngology : official journal of the European Federation of Oto-Rhino-Laryngological Societies. 2016.*
5. *Torabi-Nami M, Mehrabi S, Borhani-Haghighi A, Derman S. Withstanding the obstructive sleep apnea syndrome at the expense of arousal instability, altered cerebral autoregulation and neurocognitive decline. Journal of integrative neuroscience. 2015;14(2):169-93.*

6. *Ansarin K, Attaran D, Jamaati H, Masjedi MR, Abtahi H, Alavi A, et al. Approach to Patients with Severe Asthma: a Consensus Statement from the Respiratory Care Experts' Input Forum (RC-EIF), Iran. Tanaffos. 2015;14(2):73-94.*
7. *Graf I, Schumann U, Neuschulz J, Hofer K, Ritter L, Braumann B. Sleep-disordered breathing in orthodontic practice : Prevalence of snoring in children and morphological findings. Journal of orofacial orthopedics = Fortschritte der Kieferorthopadie : Organ/official journal Deutsche Gesellschaft fur Kieferorthopadie. 2016;77(2):129-37.*