NEW RESEARCH

SCIENTIFIC INVESTIGATIONS

JCSM
Journal of Clinical Sleep Medicine

Does Snoring Intensity Correlate with the Severity of Obstructive Sleep Apnea?

Nimrod Maimon, M.D.[1]; Patrick J. Hanly, M.D.[2]

[1]*Department of Medicine, Respirology Unit, Soroka University Medical Center, Ben-Gurion University, Beer-sheva, Israel;*
[2]*Department of Medicine, University of Calgary, Alberta, Canada*

Study Objectives: It is commonly believed that louder snoring is associated with more severe obstructive sleep apnea (OSA). We evaluated the association between snoring intensity and the severity of OSA to better understand this clinical correlation. We also investigated the relationships between body mass index (BMI), neck size, sleep stage, and body position with the intensity of snoring.

Methods: Overnight polysomnography, including objective measurement of snoring intensity, in 1643 habitual snorers referred for evaluation of sleep apnea.

Results: Sixty-five percent of patients were male; the cohort had a mean age of 48.7 ± 13.7 y and BMI of 30.9 ± 8.8 kg/m^2. The mean apnea-hypopnea index (AHI) was 28.2 ± 26. The severity of OSA was graded as no OSA (AHI < 5), mild (AHI 5 to 15), moderate (AHI 15 to 30), severe (AHI 30 to 50), and very severe OSA (AHI > 50). Snoring intensity increased progressively across all 5 categories of AHI frequency and ranged from 46.3 ± 3.6 db in patients with AHI < 5 to 60.5 ± 6.4 db in those with AHI > 50. Furthermore, there was a positive correlation between the intensity of snoring and the AHI (r = 0.66, $p < 0.01$).

Conclusions: The intensity of snoring increases as OSA becomes more severe.

Keywords: Obstructive sleep apnea syndrome, snoring, polysomnography, obesity

Citation: Maimon N; Hanly PJ. Does snoring intensity correlate with the severity of obstructive sleep apnea? *J Clin Sleep Med* 2010;6(5):475-478.

Snoring is common in the general population, with up to 25% of women and 45% of men reporting habitual snoring.[1,2] It is the most common symptom of obstructive sleep apnea (OSA), occurring in 70% to 95% of patients.[3,4] However, snoring is a poor predictor of OSA because of the high prevalence of snoring in the general population.[5] Contrary to non-apneic snoring, OSA is closely associated with a number of serious illnesses, including arterial hypertension, cardiovascular disease, stroke, and metabolic syndrome.[6,7]

The sound of snoring in patients with OSA is produced when air flows through the narrowed upper airway which closes and reopens rapidly causing increased turbulence, palatal flutter, and hence loud respiratory sounds.[8] Hence, it is reasonable to assume that more frequent obstructive events are associated with louder snoring. Consequently, it is commonly thought that louder snoring is associated with more severe OSA. However, the relationship between snoring intensity and the severity of OSA has not been systematically investigated; the majority of studies have been limited by small sample size and lack of objective monitoring of snoring intensity.[9]

The primary objective of this study was to assess the correlation between the severity of OSA, as reflected by the apnea-hypopnea index (AHI), and the intensity of snoring which was objectively monitored during sleep. The secondary objectives included the evaluation of the association between snoring intensity and other factors known to effect OSA severity, including body position, sleep stage, gender, body mass index (BMI), age, and neck size.

BRIEF SUMMARY

Current Knowledge/Study Rationale: It is commonly thought that louder snoring is associated with more severe OSA. Previous studies have been limited by small sample size and lack of objective monitoring of snoring intensity.

Study Impact: Louder snoring can be an indication of more severe OSA.

METHODS

Patient Recruitment

We conducted a prospective study over 2 years. All patients who underwent overnight polysomnography (PSG) between October 2003 and September 2005 were considered for enrollment. Patients who were ≥ 18 years of age and spent ≥ 30 min in both NREM and REM sleep on the night of PSG were eligible. Patients were excluded if they had undergone previous upper airway or neck surgery, or if they were using continuous positive airway pressure (CPAP) or an oral appliance during the night. We used only the initial diagnostic PSG for patients who had multiple PSGs during the 2-year period. We confined our assessment to patients who described themselves as snorers on a sleep history questionnaire that they completed on the night of PSG. The severity of obstructive sleep apnea was determined from the AHI for total sleep time (AHI_{TST}). OSA was defined as an AHI ≥ 5 events per hour. The study was approved by the research ethics board of St. Michael's Hospital.

Table 1—Patient demographics

Patient characteristic	All patients	Male	Female
Number	1,643	1,120 (68%)	523 (32%)**
Age, y	48.7 ± 13.7	50 ± 13	45 ± 13**
BMI, kg/m^2	30.9 ± 8.8	32 ± 9.7	28.6 ± 6**
Neck circumference, cm	39.9 ± 4.1	40.9 ± 4.1	37.8 ± 3.6**

BMI, body mass index. Data are mean ± standard deviation
*p < 0.01 vs male, **p < 0.001 vs male

Study Protocol

Full-night sleep studies were performed on all patients using Sandman 5.0 software (NPB-Melville, Ottawa, Canada). The recording montage included the following: 2-channel electroencephalography (C3-A2, C4-A1); submental and bilateral anterior tibialis electromyography; electrooculography; airflow, monitored using nasal pressure (Ultima Dual Airflow Pressure Transducer, Braebon Medical Corporation, Kanata, ON); electrocardiography; thoracic and abdominal respiratory efforts measured by respiratory inductance plethysmography (Respitrace; Ambulatory Monitoring Inc., White Plains, NY); and finger pulse oximetry (Mallinckrodt/Nellcor Puritan Bennett, Hazelwood, MO). Body position was assessed continuously through a body position sensor (Vitalog; Respironics Inc., Redwood City, CA). Finally, sound intensity was monitored synchronously with other PSG recordings (CEL-231 Digital Sound Survey Meter, CEL Instruments Bedford, England). Height and weight were assessed on the evening of the study and used to calculate BMI.

Sleep stage was scored manually according to the criteria of Rechtschaffen and Kales.[10] Total recording time was defined as the time from "lights out" in the evening to "lights on" the following morning. Total sleep time (TST) was defined as the duration of sleep from the beginning to the end of the polysomnographic recording. Sleep efficiency was defined as TST expressed as a proportion of the total recording time. Sleep was scored as NREM and REM. NREM sleep was further scored as stage 1, 2, and slow wave sleep. An arousal was defined as a sudden increase in electroencephalogram frequency to that of α waves or higher, accompanied by electromyogram activation and eye movements that lasted for 3 to 15 sec. Obstructive apnea was defined as absence of airflow > 10 sec in the presence of continued respiratory effort. Central apnea was defined as the absence of airflow > 10 sec due to loss of respiratory effort. Hypopneas were defined as reduction in airflow > 10 sec associated with an arousal and/or reduction in oxygen saturation > 3% and were classified as central or obstructive events, based on the presence of synchronous or paradoxical breathing, respectively. The AHI was defined as the number of apneas and hypopneas per hour of sleep, and was calculated separately during total sleep time (AHI_{TST}), REM sleep (AHI_{REM}), and NREM sleep (AHI_{NREM}). The AHI was also calculated with the subject in the supine and non-supine sleeping positions.

Snoring Intensity

All patients completed a self-answered questionnaire to determine whether they snored, and to rate the perceived loudness of their snoring from 1 (mild) to 4 (severe). The intensity of snoring was measured objectively during PSG by using a digital sound meter. The maximum decibel level recorded on the sound meter during each 30-sec epoch of the polysomnogram was identified and the mean value of this measurement (mean maximum decibel level) during different sleep states and body positions was used to determine snoring intensity. We used the mean maximum decibel level to classify snoring as mild (40-50 db), moderate (50-60 db), or severe (> 60 db). This classification was based on a validation study (unpublished) in a separate group of patients in which we correlated the sleep laboratory technologist's subjective assessment of snoring intensity with the recorded decibel level.

OSA Severity

The severity of OSA was determined by the AHI_{TST} and was classified as no OSA (AHI < 5), mild (AHI 5 to 15), moderate (AHI 15 to 30), severe (AHI 30 to 50), or very severe OSA AHI (> 50). This arbitrary classification, although consistent with a previously published recommendation,[11] was primarily used to aid the presentation of our data over a wide range of AHI values.

Statistical Analysis

Descriptive statistics were used to describe patient demographics. Differences in proportions of categorical variables between study groups were assessed by the χ^2 test. Differences in means of continuous variables were assessed by Student *t* test. Pearson correlation coefficient was used to measure the strength of association between continuous variables. A p value < 0.05 was considered significant. All data were analyzed with the use of SPSS version 12 software for Windows (SPSS, Inc., Chicago, IL). Continuous data are reported as mean ± SD.

RESULTS

We performed 3,303 PSGs between October 2003 and September 2005. Of these, 1,330 (41%) were excluded from the study for the following reasons: 981 (30%) were CPAP titration studies; 301 (9%) were follow-up PSGs; 34 (1%) were done to assess the response to an oral appliance; 12 (< 1%) were done to assess the impact of upper airway surgery; and 2 (< 1%) were done in patients < 18 years old. Of the remaining 1,973 diagnostic PSGs, 1,643 (83%) were done on patients who reported habitual snoring; and they form the cohort for this study.

The demographics of patients studied are shown in **Table 1**. There were more males than females; men were older, heavier, and had larger neck circumferences than women. PSG findings are summarized in **Table 2**.

The indices of snoring intensity are outlined in **Table 3**. Men had louder snoring than women (54.1 ± 6.4 versus 47.4 ± 4 db, p < 0.001); this was consistent for all sleep stages, body positions, and weight. Snoring was louder among those with BMI > 30 than those with BMI < 30 (54.8 ± 6.7 versus 49.5 ± 5.3 db, p < 0.001); it was also louder among those with neck circumference > 40 cm than those < 40 cm (54.9 ± 6.8 versus 49.5 ± 5.2 db, p < 0.001). Snoring was louder in the supine than the non-supine position (53.4 ± 6.5 versus 50.9 ± 6.6 db, p < 0.01), and during NREM than REM sleep (52.2 ± 6.7 versus 50.5 ± 6.1 db, p < 0.001).

Table 2—Polysomnography

	All patients	Male	Female
Total sleep time (TST), h	5.7 ± 1.2	5.6 ± 1.2	5.9 ± 1.2
Sleep efficiency, %	79.4 ± 14	78.3 ± 14.7	81.7 ± 14
Stage 1 NREM (%TST)	11.3 ± 10.3	12.9 ± 11.3	7.8 ± 6.5
Stage 2 NREM (%TST)	56.6 ± 12.4	56.9 ± 12.8	55.9 ± 11.4
Slow wave sleep (%TST)	16 ± 13	14.8 ± .7	18.6 ± 6.7
REM (% TST)	16 ± 7.2	15.3 ± 7.1	17.6 ± 7
AHI, /h	28.2 ± 26	38.7 ± 26.1	22 ± 13.2**
AHI (supine), /h	41.5 ± 35.6	54.1 ± 34.5	30.5 ± 20**
AHI (non-supine), /h	20.5 ± 26	28.5 ± 28	17.2 ± 11**

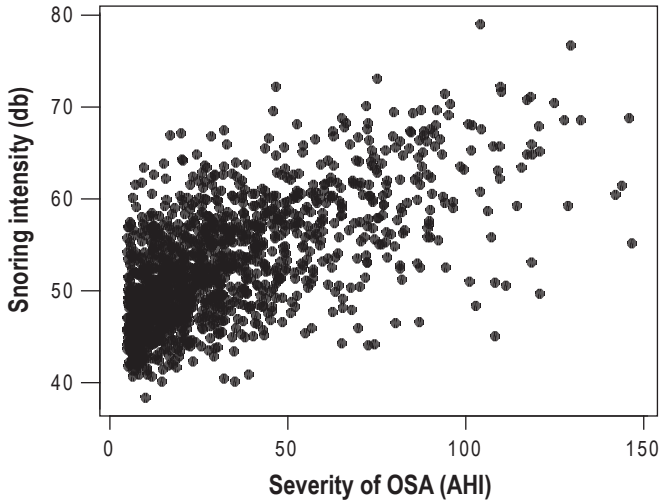
AHI, apnea-hypopnea index. Data are mean ± standard deviation.
*p < 0.01 vs male, **p < 0.001 vs male.

Table 3—Snoring intensity (decibels) in all subjects

	All patients	Male	Female
Total sleep time	52.0 ± 6.5	54.1 ± 6.4	47.4 ± 4**
NREM sleep	52.2 ± 6.7	54.4 ± 6.5	47.6 ± 4.2**
REM sleep	50.5 ± 6.1	52.3 ± 6	46.4 ± 3.9**
Supine	53.4 ± 6.7	55.4 ± 6.5	48.7 ± 4.7**
Non-supine	50.9 ± 6.6	52.8 ± 6.6	46.7 ± 4**
BMI < 30 kg/m²	49.5 ± 5.3	51.6 ± 5.4	46.6 ± 3.4**
BMI ≥ 30 kg/m²	54.8 ± 6.7	56.1 ± 6.4	49.3 ± 4.6**
Neck circumference < 40 cm	49.5 ± 5.2	51.7 ± 5.3	46.6 ± 3.5**
Neck circumference ≥ 40 cm	54.9 ± 6.8	55.9 ± 6.6	49.4 ± 4.9**

*p < 0.01 vs male, **p < 0.001 vs male.

Figure 1—Correlation between severity of OSA and snoring intensity



Snoring Intensity and Severity of OSA

The mean maximum snoring intensity during sleep in the study group was 52 ± 6.5 db (mean ± SD). Eighty-seven percent (1435/1643) of these patients had OSA (AHI > 5). The mean maximum snoring intensity in the OSA group was significantly higher than in the non-apneic snorers (52.7 ± 5.5 versus 46.3 ± 5.1 db, p < 0.01).

Patients were further divided into 5 groups based on AHI (< 5, 5-15, 15-30, 30-50, > 50). These groups comprised 208, 475, 414, 277, and 269 patients respectively. These cutoff points represent thresholds commonly used to diagnose and classify the severity of OSA. A progressive increase in the mean maximum snoring intensity was seen across all 5 groups (46.3 ± 3.6, 48.6 ± 4.4, 51.6 ± 4.8, 54.2 ± 5.2, and 60.5 ± 6.4 db, respectively. Since the decibel units are expressed on a logarithmic scale, a difference of 14.2 db between those with an AHI < 5 and those with an AHI > 50 represents an increase in snoring intensity of 1420%. This relationship was further evaluated by the Pearson correlation test which showed a relatively strong association between the severity of OSA, reflected by the total AHI, and snoring intensity, reflected by the mean maximum decibel level (r = 0.66, p < 0.01) (**Figure 1**). Snoring intensity was also positively correlated with neck circumference (r = 0.46, p < 0.01) and BMI (r = 0.49, p < 0.01). There was no correlation between snoring intensity and age.

DISCUSSION

We observed a significant positive correlation between the severity of the OSA and snoring intensity. Previously published data on this association were based upon self-report or family member report of snoring. The current study is the first to use an objective measurement of snoring intensity in a large group of patients to assess the relationship between OSA and snoring severity.

Several small previous studies recorded and analyzed snoring in an attempt to differentiate non-apneic snorers from patients with OSA. Pastercamp et al.[12] recorded the tracheal sound of 8 known OSA patients while awake, and showed increased intensity in low-frequency, medium-frequency, and high-frequency sounds compared to normal subjects. Fiz et al.[13] compared the sound of snoring in 7 non-apneic snorers and 10 patients with OSA and demonstrated significant differences between the two groups in terms of the quality and spectrum of noise. However, neither study was designed to correlate snoring intensity and OSA. Perez-Padilla[14] further evaluated the quality of snoring and showed that in patients with OSA, one can identify post-apnea snores which have different characteristics from non-apneic snoring. This difference may help differentiate OSA patients from habitual snorers. Finally, Liistro et al. reported that snoring is characterized by high-frequency oscillations of the soft palate, pharyngeal walls, epiglottis, and tongue, and that the pattern of snoring is different in OSA compared to habitual snorers.[15] The present study extends our current understanding of the relationship between OSA and snoring intensity by demonstrating, in a large cohort of adults referred for PSG, a relatively strong positive association between snoring intensity and the presence of OSA

Our findings have a number of clinical implications. Firstly, it appears that bed partners of patients with OSA are exposed to considerable environmental noise at night. The average sound intensity of our OSA patients exceeded the limits of nocturnal environmental noise pollution levels recommended by government agencies.[16] Furthermore, in some patients, the peak sound

intensity was above that considered necessary to avoid hearing loss, and our results support recent studies which showed an association between sleep disordered breathing and the bed partner's quality of life.[17,18] Secondly, the study supports the clinical observation that there is an association between snoring intensity and OSA. Although this may add to our current risk stratification for OSA, the wide variability of snoring intensity between subjects precludes this as a reliable solitary measure to diagnose sleep apnea, which continues to require more direct measurements of respiratory physiology such as airflow, respiratory effort, and oxygen saturation.

Potential limitations of this study should be addressed. First, our study was based upon a single PSG in each patient; although the monitoring was objective and used standard and calibrated measurements, it is possible the single studies were not representative of the patient's sleep. Secondly, the external validity of this study is limited; it is not known if a similar relationship exists between OSA and snoring intensity in the general population. Notwithstanding the strength of the association, we found one should be cautious in predicting the presence of OSA by snoring intensity alone.

ABBREVIATIONS

AHI, Apnea hypopnea index
BMI, body mass index
CPAP, continuous positive airway pressure
CSA, central sleep apnea
OSA, obstructive sleep apnea
PSG, polysomnography

REFERENCES

1. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993;32:1230-5.
2. Ohayon MM, Guilleminault C, Priest RG, et al. Snoring and breathing pauses during sleep: telephone interview survey of a United Kingdom population sample. *BMJ* 1997;314:860-3.
3. Whyte KF, Allen MB, Jeffrey AA, et al. Clinical features of the sleep apnoea/hypopnoea syndrome. *Q J Med* 1989;72:659-66.
4. Hoffstein V, Mateika S, Anderson D. Snoring: is it in the ear of the beholder? *Sleep* 1994;17:522-6.
5. Flemons WW, Whitelaw WA, Brant R, et al. Likelihood ratios for a sleep apnea clinical prediction rule. *Am J Respir Crit Care Med* 1994;150:1279-85.
6. Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study. *JAMA* 2000;283:1829-36.
7. Peppard PE, Young T, Palta M, et al. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med* 2000;342:1378-84.
8. Remmers JE, deGroot WJ, Sauerland EK, et al. Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol* 1978;44:931-8.
9. Gottlieb DJ, Yao Q, Redline S, Ali T, Mahowald MW. Does snoring predict sleepiness independently of apnea and hypopnea frequency? *Am J Respir Crit Care Med* 2000;162 (4 Pt 1):1512-7.
10. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. National Institutes of Health U.S. Government Printing Office, Washington, DC; Publication No. 204, 1968.
11. McNamara SG, Cistulli PA, Sullivan CE, Strohl KP. Clinical aspects of sleep apnea. In: Saunders NA, Sullivan CE, eds. Sleep and breathing: lung biology in health and disease, 2nd ed. New York: Marcel Dekker. 1994: 493-528.
12. H Pasterkamp, Schafer J, Wodicka GR. Posture-dependent change of tracheal sounds at standardized flows in patients with obstructive sleep apnea. *Chest* 1996;110:1493-8.
13. Fiz JA, Abad J, Jané R, et al. Acoustic analysis of snoring sound in patients with simple snoring and obstructive sleep apnoea. *Eur Respir J* 1996 9:2365-70.
14. Perez-Padilla JR, Slawinski E, Difrancesco LM, Feige RR, Rammers JE, Whitelaw WA. Characteristics of the snoring noise in patients with and without occlusive sleep apnea. *Am Rev Respir Dis* 1993;147:635-44.
15. Liistro G, Stanescu DC, Veriter C, Rodenstein DO, Aubert-Tulkens G. Pattern of snoring in obstructive sleep apnea patients and in heavy snorers. *Sleep* 1991;14:517-25.
16. Public Law No. 92-574, 86 Stat. 1234 (1972). Noise Pollution and Abatement Act of 1972, codification amended at 42 U.S.C. 4901-4918 (1988)
17. Breugelmans JG, Ford DE, Smith PL, Punjabi NM. Differences in patient and bed partner-assessed quality of life in sleep-disordered breathing. *Am J Respir Crit Care Med* 2004;170:547-52.
18. Sharief I, Silva GE, Goodwin JL, Quan SF. Effect of sleep disordered breathing on the sleep of bed partners in the Sleep Heart Health Study. *Sleep*. 2008;31:1449-56.

ACKNOWLEDGMENTS

Research performed at St. Michael's Hospital Sleep Laboratory.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication January, 2010
Submitted in final revised form February, 2010
Accepted for publication February, 2010
Address correspondence to: Nimrod Maimon, M.D., Division of Respirology, St. Michael's Hospital, 30 Bond St, Suite 6049, Toronto, ON, Canada M5B 1W8; Tel: (416) 783-6331; E-mail: nimrod.maimon@gmail.

DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.