

The Snoring Spectrum*

Acoustic Assessment of Snoring Sound Intensity in 1,139 Individuals Undergoing Polysomnography

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Study objectives: To quantify the snoring sound intensity levels generated by individuals during polysomnographic testing and to examine the relationships between acoustic, polysomnographic, and clinical variables.

Design: The prospective acquisition of acoustic and polysomnographic data with a retrospective medical chart review.

Setting: A sleep laboratory at a primary care hospital.

Participants: All 1,139 of the patients referred to the sleep laboratory for polysomnographic testing from 1980 to 1994.

Interventions: The acoustic measurement of snoring sound intensity during sleep concurrent with polysomnographic testing.

Measurements and results: Four decibel levels were derived from snoring sound intensity recordings. L_1 , L_5 , and L_{10} are measures of the sound pressure measurement in decibels employing the A-weighting network that yields the response of the human ear exceeded, respectively, for 1, 5, and 10% of the test period. The Leq is a measure of the A-weighted average intensity of a fluctuating acoustic signal over the total test period. L_{10} levels above 55 dBA were exceeded by 12.3% of the patients. The average levels of snoring sound intensity were significantly higher for men than for women. The levels of snoring sound intensity were associated significantly with the following: polysomnographic testing results, including the respiratory disturbance index (RDI), sleep latency, and the percentage of slow-wave sleep; demographic factors, including gender and body mass; and clinical factors, including snoring history, hypersomnolence, and breathing stoppage. Men with a body mass index of > 30 and an average snoring sound intensity of > 38 dBA were 4.1 times more likely to have an RDI of > 10 .

Conclusions: Snoring sound intensity levels are related to a number of demographic, clinical, and polysomnographic test results. Snoring sound intensity is closely related to apnea/hypopnea during sleep. The noise generated by snoring can disturb or disrupt a snorer's sleep, as well as the sleep of a bed partner. (CHEST 1999; 115:762-770)

Key words: acoustic; obstructive sleep apnea; polysomnography; snoring; spectrum

Abbreviations: BMI = body mass index; CI = confidence interval; dB = decibel; dBA = sound pressure measurement in decibels employing the A-weighting network that yields the response of the human ear; L_1 = measure of the dBA level exceeded for 1% of the test period; L_5 = measure of the dBA level exceeded for 5% of the test period; L_{10} = measure of the dBA level exceeded for 10% of the test period; Leq = a measurement of the A-weighted average energy of a fluctuating acoustic signal over a specific measurement period; LS = light sleep; MPCA = Minnesota Pollution Control Agency; OSHA = Occupational Safety and Health Administration; RDI = respiratory disturbance index; SWS = slow wave sleep

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Snoring is an acoustic phenomenon generated by vibrating tissue structures in the upper airway during sleep. Several studies have identified the anatomic structures involved in snoring using radiographic cephalometry,^{1,2} CT scanning,³ MRI,⁴ and videoendoscopy.⁵

Previous investigations have centered around observations that snoring may be a risk factor in the development of systemic arterial hypertension,⁶ coronary artery disease,⁷ and stroke.⁸ Recent literature suggests that increased upper airway resistance associated with snoring can lead to a transient sleep disturbance resulting in daytime somnolence.⁹ The

pathogenic mechanisms leading to these transient events during sleep have been described¹⁰ and subsequently reported as the upper airway resistance syndrome.¹¹

Snoring is known to be an important symptom of obstructive sleep apnea syndrome or the upper airway resistance syndrome.^{12,10} However, data suggest that a reliable diagnosis of a clinically significant breathing abnormality during sleep cannot be made based solely on a patient's history of noisy respiration during sleep.^{13,10}

It may be that "simple" snoring does not exist because when the sleep of a bed partner is disturbed snoring constitutes a social nuisance.¹⁴ The sleep disturbance to the bed partner has been linked to chronic insomnia and its consequences. Previous research also suggests that snoring constitutes excessive bedroom noise exposure and may cause hearing problems.¹⁵

Regulatory standards have been established by the Occupational Safety and Health Administration (OSHA) to limit noise exposure in the workplace.¹⁶ Federal and state agencies have set standards for acceptable noise levels in commercial and residential areas.^{14,17,18} Regulatory agencies have set building standards to ensure acoustic damping of environmental ambient sound to preserve a peaceful environment inside the home. No systematic study has attempted to relate the amount of sound generated by snoring to noise levels that have been deemed by regulatory agencies to cause hearing problems,¹⁶ or to levels that may disrupt the sleep of a bed partner.^{14,17,18}

Many issues regarding the overall health impact of nonapneic snoring have not been resolved. A recently published review of this topic has emphasized that although many investigators have measured snoring as part of their study protocols, there is little agreement as to the details of measurement, signal analysis, and interpretation. Perhaps the most important drawback of all of the measurements is the lack of biological validation. To date, there are no studies validating the electronic measurement of a sound scored as a snore by a polysomnographic technologist or by a computer against its perception as snoring by the listeners.¹⁹

This study is a systematic investigation to quantify the sound pressure levels of snoring generated during polysomnographic testing, and to explore the associations between snoring sound intensity and a variety of demographic and clinical factors. All of the study participants underwent polysomnographic monitoring with acoustic measurement of sound intensity. Sound measurements were performed using a standard technique, so that snoring sound intensity measures could be related to polysomno-

graphic and clinical measures, as well as to governmental and regulatory acoustic standards. The study was conducted with the approval of the HealthEast Institutional Review Board and in accordance with HealthEast research policies.

MATERIALS AND METHODS

Study Population

All of the patients referred to a sleep laboratory for polysomnographic testing from 1980 to 1994 were investigated for snoring sound intensity levels using a method that has previously been reported.²⁰ Sound level recordings were available for the 1,139 patients who comprised the study population.

Data Collection

Acoustic Data: Sound intensity levels during sleep were prospectively measured during polysomnographic testing using a consistent methodology. The acoustic data acquisition and display methods, and the sonographic block diagram for conducting the acoustic measurement have been described previously.²⁰ The testing room had a background noise level below 35 dBA (sound pressure measurement in decibels employing the A-weighting network that yields the response of the human ear), with an ambient noise level always ≥ 10 decibels (dB) below the measured signal, and a reverberation time of < 1.5 s. These parameters ensured that the sound that was measured was generated by the patient. The microphone used to record the snoring sounds was suspended 24 inches above the surface of the patient's bed. This arrangement allowed a noninvasive recording approach and, more importantly, a standardized distance that approximated the distance between sleeping bed partners. Based on acoustic measurements, it was estimated that a variation of not more than ± 3 dBA would occur if a patient changed position by rolling side to side. The recording equipment had a frequency response of ± 10 dB from 20 to 12,000 Hz, and a 70-dB dynamic range. The system was acoustically calibrated with a known test tone before each study. A noise exposure analyzer (model NA850A; Illbruck USA Inc; Minneapolis, MN) was used for all snoring sound measurements. This instrument examined the sound pressure level of a random noise source at 1-s intervals and classified it into 1-dB steps according to the amplitude exceeded. The sound generated by the subject during sleep was identified as snoring by the monitoring technical personnel.

The following patient information was entered into a computer database that was part of measurement apparatus: name, hospital medical record number, test date, duration of testing, distribution of A-weighted dB values over time, and the average A-weighted dB level (L_{eq}). Three peak dB levels were selected for analysis from the resulting continuous distribution of the A-weighted dB values over time: the dBA level exceeded for 1% of the test period that represents the maximum peak level measured during the testing period (L_1), the dBA level exceeded for 5% of the test period (L_5), and the dBA level exceeded for 10% of the test period (L_{10}).

Polysomnography: The polysomnographic results for each patient were retrospectively abstracted from hospital medical records. Standard polysomnographic recordings consisted of 2 EEG derivations (O2-A1 and C3-A2), electro-oculogram receiver operating characteristics (ROC-A1 and LOC-A2), an electromyogram of submental and tibialis anterior muscles, and a modified V2-lead ECG. Respiration was monitored using an oronasal

thermistor, and thoracic and abdominal movements were monitored with inductive plethysmography. Arterial oxygen saturation (SaO₂) was recorded using finger pulse oximetry. Sleep was scored in 30 s epochs according to the criteria of Rechtschaffen and Kales.²¹ Sleep efficiency was defined as the total sleep time divided by the total time in bed. Sleep latency was defined as the time between lights out and the onset of sleep.

Apnea was defined as a complete cessation of oronasal air flow for at least 10 s. Apneas were classified as obstructive in the presence of thoracic or abdominal movements. Hypopnea was defined as a reduction of $\geq 50\%$ in the amplitude of the airflow waveform from a preceding stable baseline. The respiratory disturbance index (RDI) was calculated as the sum of the apneas and hypopneas divided by the total sleep time.

Clinical Information: Clinical information was retrospectively abstracted from medical records according to a standard protocol: demographics, medical history, chief complaint leading to sleep laboratory referral, polysomnogram and other related procedure information, test results, and concluding diagnosis. Positive or negative clinical history information was recorded only if specific documentation was found in the medical record. Data elements that were not specifically documented were treated as missing.

Study Database: A final computer database was developed by combining the sound intensity level data (Leq, L₁, L₅, and L₁₀) with all available polysomnographic and clinical data abstracted from the medical record. The accuracy and completeness of the study database were verified by conducting range, missing value, and consistency checks for each data element. Sound level measurements were prospectively recorded at the time of testing and, therefore, are complete for all patients. There are missing clinical data and, to a lesser extent, missing polysomnographic results because this information was abstracted from medical records.

Statistical Analysis: The results were expressed as percentages and means (\pm SD) or 95% confidence intervals (CIs). Hypothesis tests were used to evaluate the presence of statistical significance among the groups. The likelihood ratio χ^2 test and the proportion Z test were used for categorical data analysis. Nonparametric testing was used for continuous variables with a nongaussian distribution. Multivariate logistic regression analysis and partial correlation coefficients were used to determine the independent effects of selected variables on the dependent variables. A p value of < 0.05 was considered statistically significant.

RESULTS

Study Population

A total of 1,139 patients were included in the study, with an average age (\pm SD) of 48.1 ± 13.3 years old. Seventy-eight percent of the patients were men, and 22% were women. The study population was substantially heavier than the general adult population of the United States. Of the 654 patients with a known weight and height, the median body mass index (BMI) was substantially greater than the median BMI of the general adult population of the United States in 1995, respectively: 34.2 vs 25.5 kg/m². Obesity is defined in this study as a BMI of ≥ 30 ; thus, 62% of the patients were considered obese. Descriptive statistics for each variable measured are shown in Table 1.

The chief complaints leading to referral for poly-

Table 1—Summary of Study Variables

Variables	Valid %, Mean (SD)	Valid, N	Missing, N
Demographics			
Age, yr	48.1 (13.3)	1,131	8
Weight, lb	225.8 (58.4)	729	410
Height, inch	68.6 (5.1)	664	475
BMI, kg/m ²	33.4 (8.1)	654	485
Gender		1,138	1
Male	78.3		
Female	21.7		
Snoring sound intensity measures			
Leq	46.2 (7.9)	1,138	1
L ₁	56.7 (7.2)	1,135	4
L ₅	51.2 (6.7)	1,071	68
L ₁₀	48.8 (6.1)	959	180
Polysomnographic test results			
SWS	9.4 (10.4)	1,129	10
LS	76.0 (12.7)	1,131	8
Sleep efficiency	78.9 (14.2)	1,131	8
Sleep latency, min	12.2 (18.2)	1,126	13
RDI	31.0 (33.5)	1,094	45
< 10	38.6		
≥ 10	61.4		
Obstructive sleep apnea diagnosis		943	196
Yes	682 (72.3)		
No	261 (27.7)		
Clinical information			
Chief complaint leading to the referral for a polysomnographic test		682	457
Snoring	29.6		
Hypersomnolence	36.2		
Night time breathing stoppage	18.0		
Narcolepsy	2.6		
Other	13.5		
History of breathing stoppage		305	834
Yes	89.8		
No	10.2		
History of systemic hypertension		199	940
Yes	48.2		
No	51.7		
Duration of snoring symptoms		245	894
Recent onset	11.4		
A few years	27.4		
Many years	61.2		
Nasal chambers		249	890
Normal	57.0		
Narrow	43.0		
Oropharynx		319	820
Normal	26.6		
Narrow	73.4		

somnographic testing were categorized into five groups: snoring (29.6%), hypersomnolence (36.2%), nighttime breathing stoppage (18.1%), narcolepsy (2.6%), and other (13.5%).

Snoring Sound Intensity During Sleep

Four measures of snoring sound intensity during sleep were tabulated for each subject: L₁, L₅, L₁₀,

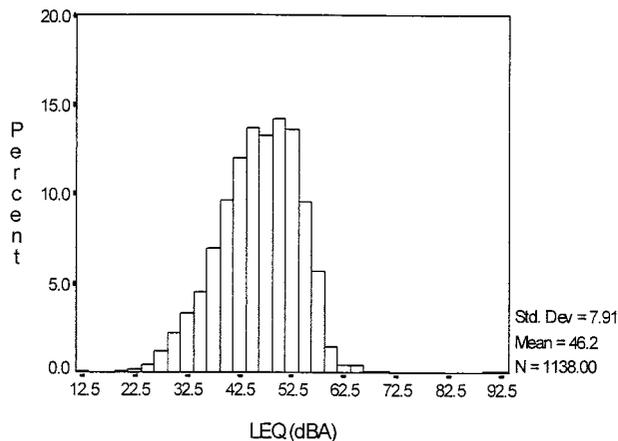


FIGURE 1. Distribution of the average A-weighted decibel level Leq.

and Leq. The distributions of these values in the study population are presented in Figures 1 through 4. An L_{10} value of 40 dBA was exceeded by 78.7% of the patients, and an L_{10} value of 50 dBA was exceeded by 34.4% of the patients. An Leq value of 38 dBA was exceeded by 84.7% of the patients. Forty-eight percent of the patients with breathing stoppage as their chief complaint had an Leq between 50 and 70 dBA. The regulations of the Minnesota Pollution Control Agency (MPCA) limit the maximum acceptable outdoor nighttime noise levels to L_{10} levels of 55 dBA. This level was exceeded by 12.3% of the patients tested (12.8% of the men and 10.5% of the women).

The relationships between snoring sound intensity and demographic factors, the BMI, gender, and age were examined. Univariate analyses revealed that the BMI and gender were significantly related to all sound intensity levels (Table 2). The BMI had a positive association with snoring sound intensity levels, with a Pearson correlation coefficient for the Leq, L_1 , L_5 , and L_{10} values, respectively, of 0.30,

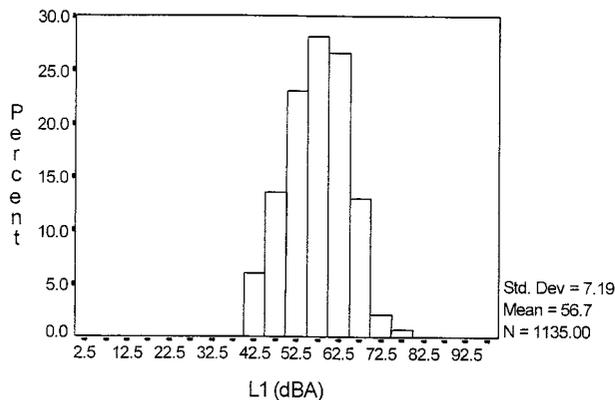


FIGURE 2. Distribution of the peak decibel level L_1 .

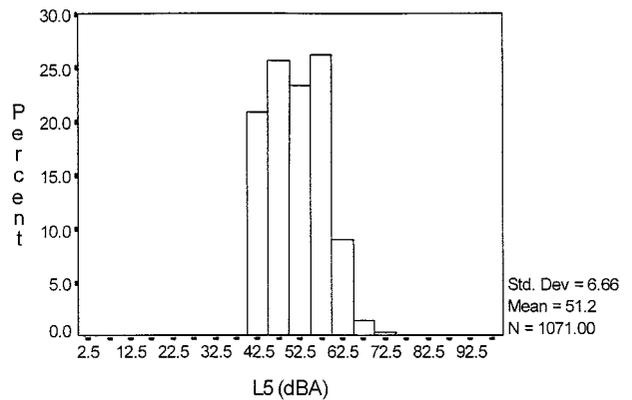


FIGURE 3. Distribution of the peak decibel level L_5 .

0.29, 0.28, and 0.24 ($p < 0.0001$). Men were significantly louder snorers than women. The relationship between snoring sound intensity levels and the BMI remained statistically significant after controlling for gender ($p < 0.001$). No correlation was found between snoring sound intensity levels and age for the Leq, L_1 , L_5 , and L_{10} values, respectively: 0.08, 0.05, 0.01 and 0.06.

Snoring Sound Intensity During Sleep and Polysomnographic Results

The potential relationship between snoring sound intensity levels and the RDI was investigated. Patients with apneic snoring, defined as an RDI of ≥ 10 , generated significantly higher snoring sound intensity levels than patients with nonapneic snoring, defined as an RDI of < 10 (Table 3). The relationship between snoring sound intensity levels and the RDI was analyzed as a function of the reasons for polysomnographic referral. Patients referred with breathing stoppage as their chief complaint had significantly higher snoring sound intensity levels and RDI values than patients referred because of

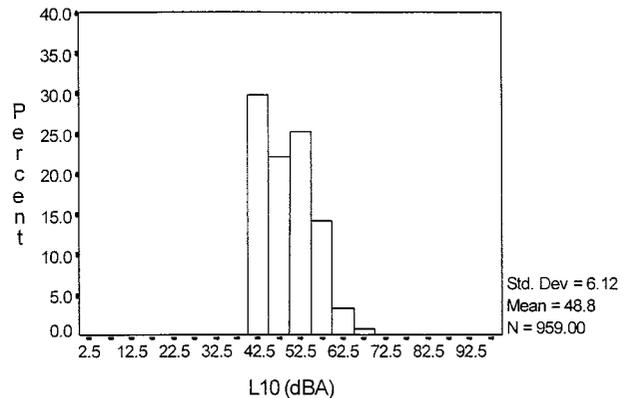


FIGURE 4. Distribution of the peak decibel level L_{10} .

Table 2—Snoring Sound Intensity Measures by Gender

Sound Intensity Measure	Male*	Female*	p Value
Leq, dBA	46.8 (46.2–47.3)	43.8 (42.8–44.9)	< 0.0001
No. of patients	891	247	
L ₁ , dBA	57.3 (56.9–57.8)	54.6 (53.6–55.6)	< 0.0001
No. of patients	889	245	
L ₅ , dBA	51.6 (51.1–52.0)	49.6 (48.7–50.4)	< 0.0001
No. of patients	849	221	
L ₁₀ , dBA	49.1 (48.6–49.5)	47.9 (47.0–48.8)	< 0.0001
No. of patients	768	190	

*Values are expressed as mean (95% CI).

snoring or hypersomnolence (Table 4). Multivariate logistic regression analysis showed that snoring sound intensity levels were strongly associated with the RDI, even after controlling for demographic and clinical factors (Table 5).

The relationships between snoring sound intensity and measures of sleep quality were analyzed (Table 6). Nonrapid eye movement sleep stages 3 and 4 were combined as slow wave sleep (SWS), and nonrapid eye movement sleep stages 1 and 2 were combined as light sleep (LS). The percentage of SWS for patients with an L₅ value of > 55 dBA (high L₅) was significantly higher than for patients with an L₅ value of ≤ 55 dBA (low L₅). The percentage of LS for patients with an L₅ value of > 55 dBA was similar to that found in patients with an L₅ value of ≤ 55 dBA. The percentage of SWS and LS for patients with an Leq of > 55 dBA (high Leq) was similar to that found in patients with an Leq of ≤ 55 dBA (low Leq). Patients with high Leq or L₅ values had significantly shorter periods of sleep latency. No significant differences in sleep efficiency were found between patients having high or low Leq or L₅ values.

Table 3—Sound Intensity Measures for Subjects With Apneic Snoring (RDI ≥ 10) and Non-apneic Snoring (RDI < 10)

Sound Intensity Measure	Nonapneic Snoring* (RDI < 10)	Apneic Snoring* (RDI ≥ 10)	p Value
Leq, dBA	42.7 (42.0–43.4)	48.8 (48.7–49.3)	< 0.0001
No. of patients	422	672	
L ₁ , dBA	53.4 (52.8–54.1)	59.2 (58.7–59.7)	< 0.0001
No. of patients	419	672	
L ₅ , dBA	48.0 (47.4–48.6)	53.2 (52.7–53.7)	< 0.0001
No. of patients	382	655	
L ₁₀ , dBA	46.0 (45.5–46.5)	50.4 (49.9–50.9)	< 0.0001
No. of patients	327	606	

*Values are expressed as mean (95% CI).

Snoring Sounding Intensity During Sleep and Clinical Factors

The clinical information regarding the chief complaint was available for 682 of the study participants (Table 1). Table 4 shows the sound intensity levels for three chief complaint categories: snoring, hypersomnolence, and breathing stoppage. Patients with breathing stoppage had significantly higher snoring sound intensity levels than patients with snoring or hypersomnolence ($p < 0.001$). Patients with snoring or hypersomnolence had similar Leq, L₁, and L₅ values ($p > 0.283$).

A complete clinical history record of the duration of snoring symptoms was available for 245 patients (Table 1). Patients with a recent onset or a snoring duration of only a few years had significantly lower average Leq values than those who had snored for many years, respectively: 45.5 (95% CI, 44.0 to 47.0) vs 48.2 (95% CI, 46.8 to 49.7) dBA ($p < 0.02$). Similar results were obtained for patients with a positive or negative clinical history of breathing stoppage. A complete clinical history record of breathing stoppage was available for 305 patients. Patients ($n = 31$) with a documented absent history of breathing stoppage during sleep had significantly lower average Leq values than those ($n = 290$) with a known history of breathing stoppage, respectively: 44.3 (95% CI, 41.2 to 47.4) vs 47.7 (95% CI, 46.7 to 48.6) dBA ($p = 0.028$).

A complete clinical history record of the presence or absence of systemic hypertension was available for 199 patients (Table 1). Patients with a history of systemic hypertension had significantly higher snoring sound intensity levels than did patients without a history of hypertension (Table 7). However, the relationships between hypertension and snoring sound intensity levels were lost after adjusting for effects of gender and obese status ($p > 0.251$).

Some patients received a visual investigation of the nasal ($n = 249$) and oropharyngeal ($n = 319$) regions by their referring physicians. Based on this investigation, patients with “narrow” nasal or oropharyngeal dimensions were put into one group, and patients with “normal” dimensions were put into another group. Patients with a narrow oropharynx had significantly higher Leq, L₁, and L₅ values than patients with a normal oropharynx. However, there was no statistical difference in snoring sound intensity levels between patients having narrow or normal nasal chambers (Table 8).

Multivariate Logistic Regression Analyses

Multivariate logistic regression analyses measured the relative strength of the associations among the RDI categories (an RDI of ≥ 10 or < 10), the

Table 4—Sound Intensity Measures and RDI For Three Categories of Chief Complaint

Sound Intensity Measure	Chief Complaint			p Value†	p Value‡
	Snoring*	Hypersomnolence*	Breathing Stoppage*		
Leq, dBA	46.2 (45.3–47.2)	45.5 (44.4–46.6)	48.7 (47.3–50.0)	0.001	0.608
No. of patients	202	247	123		
L ₁₀ , dBA	47.4 (46.6–48.2)	48.9 (48.0–49.8)	50.9 (49.6–52.1)	0.000	0.033
No. of patients	182	197	112		
L ₅ , dBA	50.3 (49.4–51.1)	50.9 (50.0–51.8)	53.7 (52.5–55.0)	0.000	0.444
No. of patients	198	226	118		
L ₁ , dBA	56.8 (55.9–57.6)	56.0 (55.1–57.0)	59.3 (57.9–60.6)	0.001	0.283
No. of patients	201	247	123		
RDI	25.4 (21.6–29.2)	31.8 (27.4–36.2)	46.7 (39.5–53.9)	0.000	0.101
No. of patients	195	232	122		

*Values are expressed as mean (95% CI).

†The p value for the comparison of sound intensity levels associated with snoring and breathing stoppage.

‡The p value for the comparison of sound intensity levels associated with snoring and hypersomnolence.

snoring sound intensity levels, and a variety of demographic and clinical factors (Table 5). Independent variables were selected for the model based on their univariate relationship with the RDI categories, the dependent variable. Independent variables with strong colinearity with other independent variables were taken out of the model. Regression results show that among the factors included in the model, snoring sound intensity levels, obesity status, and gender had the strongest associations with the RDI categories. Patients with Leq values of ≥ 38 dBA were 3.44 (95% CI, 1.99 to 5.95) times more likely to have an RDI ≥ 10 after controlling for gender, age, BMI, SWS, and sleep efficiency. Based on the regression results, it was found that obese male patients with an Leq value of ≥ 38 dBA were 4.1 (95% CI, 2.15 to 6.43) times more likely to have an RDI of ≥ 10 than any other group.

DISCUSSION

Using a large data set of clinical variables and polysomnographic results in conjunction with standardized snoring sound measurements, we have

attempted to describe the full range of snoring sound intensity. We realize that the acoustic measurement technique used in this study has certain limitations. It did not allow for the linking of acoustic data to polysomnographic variables in time. It assessed frequency by employing the A-weighting network that yields a frequency response curve similar to that of the human ear. Recent studies suggest that frequency information may be important for distinguishing between snoring produced by individuals with and without obstructive sleep apnea.^{22–24}

As pointed out by Hoffstein,¹⁹ many questions about the clinical significance of snoring are unanswered largely because of the lack of standardized procedures for measuring snoring sounds. The measurement methodology used in this study provided accurate and consistent information on snoring sound intensity.

The same equipment was used to measure and record snoring sound intervals for all study patients. While each recording was made, technicians subjectively verified that the noisy breathing sounds being recorded were made by snoring. The acoustic analyses of the snoring sound intensity data were per-

Table 5—Multivariate Logistic Regression Statistics*

Independent Variables	Regression Coefficient	Standard Error	Significance, p Value	Odds Ratio (95% CI)
Intercept	-2.14	0.97	0.000	
Leq ≥ 38	1.24	0.28	0.000	3.44 (1.99–5.95)
Obese, BMI > 30	1.01	0.20	0.000	2.76 (1.87–4.06)
Gender, male	0.89	0.23	0.000	2.43 (1.54–3.83)
Age	0.03	0.01	0.000	1.03 (1.01–1.04)
Percentage of SWS	-0.02	0.01	0.071	0.98 (0.96–1.00)
Sleep efficiency	-0.01	0.01	0.492	0.99 (0.98–1.01)

*Dependent variable is coded as 1 for RDI ≥ 10 and also for RDI < 10; -2 log likelihood = 667.56; degree of freedom = 6.

Table 6—Sleep Architecture Measures

Sleep Architecture Measure	Leq, dBA			L ₅ , dBA		
	0–55*	> 55*	p Value	0–55*	> 55*	p Value
Sleep latency, min	12.6 (11.4–13.8)	9.5 (7.3–11.7)	0.025	13.2 (11.8–14.7)	9.8 (8.3–11.3)	0.004
No. of patients	1001	125		748	311	
Sleep efficiency, min	78.8 (77.9–79.7)	79.4 (77.1–81.7)	0.642	78.6 (77.5–79.7)	79.7 (78.4–81.1)	0.821
No. of patients	1006	138		753	311	
Percentage of SWS	9.5% (8.8–10.1)	8.6% (6.8–10.4)	0.327	10.1% (9.3–10.8)	7.5% (6.5–8.5)	0.011
No. of patients	1004	125		752	310	
Percentage of LS	76.3% (75.5–77.0)	74.6% (72.2–77.1)	0.242	75.8% (74.9–76.7)	76.7% (75.3–78.1)	0.191
No. of patients	1006	125		753	312	

*Values are expressed as mean (95% CI).

formed using methods that are used by OSHA¹⁶ to determine the intensity of noise pollution in the workplace, and by other governmental agencies, including the MPCA, to monitor environmental noise. These methods had not been used previously for sleep study purposes. Government and industrial hearing conservation programs based on OSHA standards recommend that hearing protection be used if the average sound intensity reaches 85 dBA for ≥ 8 h, and mandate that hearing protection be used at levels of ≥ 90 dBA for ≥ 8 h.¹⁶ Without hearing protection, the amount of exposure allowed is 4 h at 95 dBA, and the time decreases by half for each additional 5 dBA. Our data does not support the proposition that hearing loss may result in a significant number of individuals as a consequence of sleeping with a snoring bed partner, as previously suggested.¹⁵

On the other hand, there is no doubt that many snoring men and women regularly disturb the sleep of their bed partner. The results of this study are consistent with this observation. The MPCA regulatory objective of a nighttime internal home L₁₀ value of 35 to 40 dBA is equivalent to an outdoor residential L₁₀ value of 55 dBA, presuming approximately 20 dBA of structural damping. When sound stimuli

reach intensities of 40 to 50 dBA, changes in EEG can be observed; at 70 dBA, arousal or awakening are imminent.²⁵ Previous studies^{26,27} have shown that noise events between the Leq values of 36 to 56 dBA during sleep can lead to an increase in cardiac arrhythmias and excretion of urinary catecholamines. Another recent study²⁸ showed that in two hospitals with environmental Leq values, respectively, of 54 and 46 dBA, the subjective sleep quality of men was significantly better in the quieter hospital. In our study, an L₁₀ value of 40 dBA was exceeded by 78.7% of the patients, and an L₁₀ value of 50 dBA was exceeded by 34.4% of the patients. An Leq value of 38 dBA was exceeded by 84.7% of the patients. Patients with breathing stoppage as their chief complaint had significantly higher snoring sound intensity levels, and 48% percent of these patients had Leq values ranging from 50 to 70 dBA.

We have shown that women do not snore as loudly as men. The difference between the mean Leq value for men and women was 3.0 dBA, which translates into a substantially higher sound intensity perception for the listener. This gender effect on snoring sound during sleep was independent of the severity of sleep-disordered breathing and body mass. Women also had lower peak intensity values. For example, men presented L₁ values that were 2.7 dBA louder than those for women. This finding stands in contrast to data from Metes et al,²⁹ who reported that there was no difference in the maximal snoring sound intensity levels between 77 women and 293 men who were studied prospectively during sleep. It is not clear what accounts for this difference in results. Clearly, there were differences in the methods used to measure snoring sound intensity. Our data were based on the complete distribution of sound intensity over time, and Metes et al²⁹ used the maximal snoring sound intensity reported during the recording period.

Similar to Metes et al,²⁹ we did not find a strong relationship between age and snoring sound intensity

Table 7— Sound Intensity Measures and Systemic Hypertension

Sound Intensity Measure	Systemic Hypertension*		p Value
	Yes	No	
Leq, dBA	49.6 (48.0–51.1)	46.9 (45.5–48.3)	0.018
No. of patients	96	103	
L ₁ , dBA	60.1 (58.8–61.4)	57.2 (55.8–58.5)	0.005
No. of patients	95	103	
L ₅ , dBA	53.3 (52.0–54.6)	51.3 (50.0–52.6)	0.022
No. of patients	94	100	
L ₁₀ , dBA	50.1 (48.8–51.4)	48.6 (47.3–49.9)	0.074
No. of patients	84	93	

*Values are expressed as mean (95% CI).

Table 8—The Relationship Between Snoring Sound Intensity Measures and Anatomical Characteristics of Nasal Chambers and Oropharynx at Visual Inspection

Sound Intensity Measure	Nasal Chambers			Oropharynx		
	Narrow*	Normal*	p Value	Narrow*	Normal*	p Value
Leq, dBA	48.2 (46.4–50.0)	46.2 (45.0–47.4)	0.103	47.6 (46.6–48.7)	44.9 (43.2–46.7)	0.016
No. of patients	107	142		234	85	
L ₁ , dBA	57.8 (56.5–59.2)	57.1 (55.9–58.2)	0.377	57.9 (57.0–58.8)	55.7 (54.1–57.3)	0.017
No. of patients	106	142		233	85	
L ₅ , dBA	51.9 (50.6–53.2)	51.1 (50.0–52.2)	0.313	51.6 (50.7–52.5)	50.0 (48.4–51.6)	0.058
No. of patients	104	135		228	78	
L ₁₀ , dBA	49.4 (48.1–50.6)	48.3 (47.2–49.4)	0.146	48.8 (48.0–49.7)	47.8 (46.3–49.4)	0.183
No. of patients	96	126		213	68	

*Values are expressed as mean (95% CI).

levels. Stoohs et al³⁰ showed that snoring sound intensity levels can be a measure of respiratory effort. One may hypothesize that age would have a negative effect on maximal negative inspiratory pressure generated during the obstructed respiratory effort associated with snoring.³¹ This hypothesis was not adequately tested with the analyses reported here. Further analysis of the relationship between the loudest observed sound intensity levels and age is needed.

A very significant difference in sound intensity was noted between apneic and nonapneic snoring patients. The Leq and the peak values for L₁ and L₅ were more than 5 dBA louder for apneic snoring patients with an RDI of ≥ 10 than they were for nonapneic snoring patients with an RDI of < 10 . This observation corresponds to data from Hoffstein and colleagues³² who studied pharyngeal function and snoring characteristics in apneic and nonapneic snoring subjects. This finding is consistent with the increased negative inspiratory pressures generated in patients who exhibit complete upper airway obstruction during sleep. Higher intraluminal negative pressures present upon the resumption of breathing in apneic snoring patients lead to higher inspiratory flow rates, turbulent flow, and higher forces on the vibrating structures in the upper airway, resulting in higher sound intensity levels.^{28,33}

From our study sample, two other important anatomical determinants were associated with higher snoring sound intensity levels: a higher BMI and narrow pharyngeal passages.

We hypothesized that snoring sound intensity would have a negative association with sleep quality. Our data partially supports this hypothesis. Varying degrees of snoring sound intensity levels resulted in significant differences in the polysomnographic markers of sleep quality. Specifically, the percentages of SWS were significantly lower in patients with peak L₁ or L₅ values of ≥ 55 dBA,

compared to patients with L₁ or L₅ values of < 55 dBA. Sleep latency was significantly lower in patients with L₁, L₅, and Leq values ≥ 55 dBA, compared to patients with L₁, L₅, and Leq values of < 55 dBA. These results indicate that louder snoring has an adverse association with the sleep quality of the snorer. Our data do not provide the answer to the mechanisms by which sleep architecture is altered. Although previous work¹⁰ suggests that increased respiratory effort associated with periods of flow limitation during sleep plays an important role, the direct effects of snoring sound on the sleep architecture of the snoring individual need further evaluation.

We were unable to show a significant independent relationship between the intensity of snoring sounds and a clinical history of arterial hypertension. Significant differences in snoring sound intensity levels between subjects with or without a history of arterial hypertension were explained by the BMI and gender, an observation previously reported.^{34,35}

Finally, after entering relevant variables into a logistic regression model with an RDI of ≥ 10 as the dependent variable, we were able to determine that an Leq of ≥ 38 , a BMI of > 30 , gender, and age were significantly associated with the presence of apneic sleep.

We conclude that a snoring sound intensity level can be accurately measured and that it has demonstrated associations with other sleep parameters. Snoring sound intensity should be included in the assessment of patients with sleep-disordered breathing in order to characterize both the patient's physiologic condition and the acoustic impact on the patient's bed partner. To further delineate the impact of snoring on the snorer and his/her bed partner, future research is needed that incorporates the measurement of snoring intensity and frequency variables, and links these acoustic variables to polysomnographic variables in time.

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