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Expiratory Breathing Sounds Characteristics During Wakefulness and Sleep in Mild and Severe Apneic Groups

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ABSTRACT

We investigated plausible changes in spectral and phasic properties of tracheal respiratory sounds from wakefulness to sleep in relation to obstructive sleep apnea (OSA). Data were tracheal expiratory breathing sounds of 30 subjects during wakefulness and sleep, both recorded in supine position. Subjects were divided into 2 groups of mild and severe OSA (15 in each group) based on their apnea/hypopnea index (AHI). Power spectral density (PSD) and phase spectrum, were estimated from each normalized expiratory sound; their characteristics were compared within and between the groups. Spectral analysis during wakefulness showed an opposite pattern between mild and severe groups in low and high frequencies. The mild group's PSD on average was higher than that of the severe group in lower frequencies, while this pattern was reversed in high frequencies. During sleep, however, the PSD of the severe group was higher than that of mild group across the spectrum. On the other hand, during sleep, the average phase spectrum of the severe group showed larger delay than that of the mild group, especially at higher frequencies. The physiological and pathological interpretations of these findings are discussed.

INTRODUCTION

Obstructive sleep apnea (OSA) syndrome is due to partial or complete collapse of the upper airway (UA) [1]. An individual with OSA, despite having persistent effort to breathe, may experiences periods of cessation of breathing (apnea) and/or more than 50% reduction in airflow (hypopnea), which last at least 10s and are associated with a minimum of 4% drop in oxygen saturation level in blood [2].

OSA affects the health of both children and adults; It is associated with major comorbidities including increased risk of cardiovascular disease, daytime sleepiness, reduced concentration and increased risk of car accidents [2]. Current gold standard for diagnosis of OSA is full-night polysomnography (PSG). It is used to count the number of apnea/hypopnea events per an hour of sleep (AHI). It also records many physiological signals such as heart and muscles signals, respiratory effort, respiratory flow, and brain waves (EEG) to have a full physiological picture of the patients' UA collapsibility and sleep quality. PSG assessment is time-consuming, cumbersome and expensive with long waiting list. Therefore, designing simpler assessment modalities of unsupervised monitoring such as portable home monitoring devices is an alternative way to detect sleep apnea and to overcome the drawbacks of PSG. The goal of this study is explore the breathing sounds potential to understand changes of upper airway from wakefulness to sleep in people with mild and severe OSA. The outcome of this study may lead to physiological understanding of the mechanism of UA collapse during sleep with only breathing sounds analysis.

The underlying pathophysiology of OSA is due to the interaction of various features of airway anatomy and neuromuscular control [3]; therefore deficits in each trait may vary considerably between individuals. Recent studies using MRI/CT imaging on the UA of subjects with OSA, have shown that OSA individuals have a narrower UA compared to non-OSA individuals during wakefulness [4], [5]. In addition, their UA showed more collapsibility during sleep [6].

Our team and a few others around the world have been using tracheal respiratory sounds to monitor and assess OSA. It is known that structural and physiological properties of

the UA would affect sound generation mechanism [7]. Thus, our team focused on analyzing respiratory sound during wakefulness for OSA assessment and classification [8], [9]. In this study, however, we focused on changes in spectral and phasic characteristics of respiratory sounds from wakefulness to sleep in relation to OSA, and whether respiratory sound analysis can represent pathophysiological changes in OSA population.

METHOD

Data

We used tracheal respiratory sounds of 30 individuals with OSA. Tracheal breathing sounds were recording during both wakefulness and sleep in supine position with head resting on a pillow. During wakefulness, subjects were instructed to breathe at their normal rate in two maneuvers: first through their nose with mouth closed, and second through their mouth and a nose clip in place. After wakefulness recording, subjects were prepared for PSG assessment. Breathing sounds recording during sleep were done simultaneously with full-night PSG assessment at the Misericordia hospital Sleep Disorder Clinic (Winnipeg, Canada). The AHI of subjects were collected after their PSG assessment by sleep technicians.

The breathing sounds during both wakefulness and sleep were collected with a small microphone (Sony ECM-77B) inserted into a small chamber allowing 2 mm cone-shape space with skin, and mounted over suprasternal notch of the trachea. To ensure the microphone would not be misplaced during the night, we used a soft neckband which was sealed softly around patient's neck to sustain microphone and chamber in site. The sounds were amplified by a Biopac (DA100C) amplifier with band-pass filter in the range of 0.5-5000 Hz, and digitized at 10240 Hz sampling rate.

This study was approved by the Biomedical Research Ethics board of the University of Manitoba and all the participants signed an informed consent form prior to data collection. The participants' demographic information is shown in Table 1. Using PSG data, we grouped the participants into mild-OSA (AHI<15) and severe-OSA (AHI>15) groups (15 in each group).

Table 1: Patients' Demographic Information

Severity	Age	AHI	BMI	Neck size	Gender (M:F)
Mild-OSA	40.7	2.16	29.63	40.95	(11:4)
Severe-OSA	48.7	37.45	33.63	45.66	(13:2)

Signal analysis

We examined all the recorded tracheal breathing sounds data manually by listening to and observing the sounds in the time-and-frequency domain to exclude noisy signals or those with snoring sounds. The inspiratory and expiratory phases were identified semi-manually using our technique elaborated in [10]. During sleep, snoring usually occurs in inspiration phase. Therefore to avoid plausible snoring sounds, we selected 4 noise-free expiratory sounds in the supine position. During wakefulness, we also analyzed 4 noise-free expiratory sounds for comparison to those during sleep. We extracted sleep data only from stage 2 of sleep because that was the most common stage in our dataset. To remove the effect of low and high frequency noises, including heart and muscle sounds and ambient noise, each individual expiratory sound segment was passed through a 5^{th}-order Butterworth band-pass filter in the range of [75-2500 Hz].

In this study, we performed the same preprocessing steps as in [9]. Each expiratory sound was first normalized by its variance envelope (moving average filter of signal with 64 sample sequence) to remove its extra fluctuations. Subsequently, they were normalized by their energy (standard deviation) to compensate for plausible different flow rates in each breathing cycle. To ensure that breathing sounds are stationary, we estimated the flow from the sounds using the method in [11], and considered the sounds segments corresponding to the upper 40% of the estimated flow; during that period the breathing sounds can be considered stationary [12]. Next, we estimated the power spectrum density (PSD) and phase response of the stationary portion of each expiratory sound signal. PSD was estimated using Welch's

method in 50% overlapping windows of 205-point (~20 ms). The phase spectrum was calculated as unwrapped phase of the Fourier Transform of each selected expiratory sound. Each feature was then averaged over the 4 expiratory sounds of each subject.

Next, from the PSD of each subject 3 frequency based parameters were calculated: 30%-frequency (the frequency at which the PSD reaches 30% of the total power), mean power frequency (MPF) (the frequency at which the PSD reaches 50% of total power) and 70%-frequency (the frequency at which the PSD reaches 70% of the total power).

The PSD, phase spectra and the three frequency features were compared between the groups and between the wakefulness and sleep. For comparison, we used paired and unpaired t-tests (for within and between group comparisons); a p-value of 0.05 was considered as significant.

RESULTS & DISCUSSION

Power spectral analysis of mouth breathing sounds during wakefulness showed that in low frequencies (160-250 Hz) the average PSD of mild subjects was significantly more than that of severe subjects ($p<0.0006$) (Fig. 1); this pattern was reversed during sleep ($p<0.0002$) (Fig. 2). A similar pattern was observed for nose breathing sounds but with more overlap between the two groups. On the other hand, the average PSD of the severe group at high frequencies (1050-2300Hz) was higher than that of the mild group during both wakefulness and sleep; this difference was significant only during sleep ($p<0.002$).

The MPF and 70%-frequency features of severe group were significantly higher than those of mild group ($p<0.007$ and $p<0.02$, respectively). It implies that the PSD of the severe group, on average, had more power in higher frequencies; that was more significant during sleep. On the other hand, the 30%-frequency of mild group was significantly ($p<0.02$) lower than those of severe group. This finding implies that the PSD of the mild group during wakefulness have more power in lower frequencies. These findings are congruent with the results of our previous studies [9], [13] and imaging studies [4], [6] that during wakefulness the UAs of severe subjects were found to be both thicker and more compliant than that of mild-OSA subjects, and also that during sleep the OSA subjects showed an increased stiffness. Congruent with the mentioned studies, the spectral pattern of low and high frequencies observed in this study suggest a simultaneous regional compliance and stiffness in the UA of severe OSA subjects.

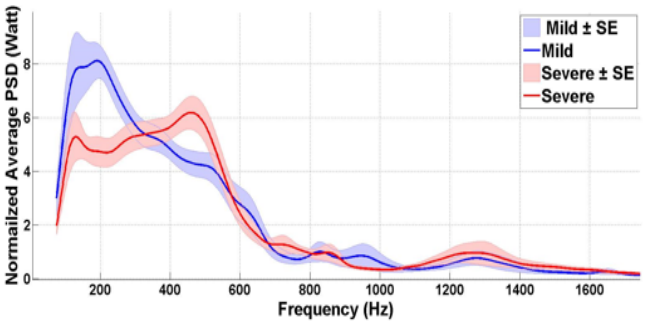

Figure 1: Average power spectra (solid lines) of supine expiratory mouth breathing sounds during wakefulness with their standard error intervals (shadows) in mild (blue) and severe (red) OSA groups

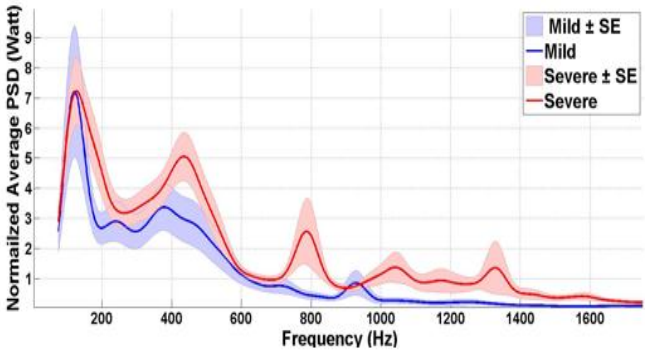

Figure 2: Average power spectra (solid lines) of supine expiratory sounds during stage 2 of sleep with their standard error intervals (shadows) in mild (blue) and severe (red) OSA groups

Phase assessment of the recorded sounds during wakefulness showed that the average phase spectrum of the severe group was approximately equal to that of the mild group. We speculate this is due to the fact that during wakefulness the dilator muscle's activity of people with severe OSA is increased to compensate for their narrower UA [14]. On the other hand, during sleep the absolute value of the average phase spectrum of the mild group was higher than that of the severe group

($p<0.01$) (Fig. 3). This implies a larger delay in transmitting breathing sounds signal through the UA in severe subjects. This might be related to the structural and physiological changes of the UA during sleep, such as more airway narrowing and collapsibility during sleep.

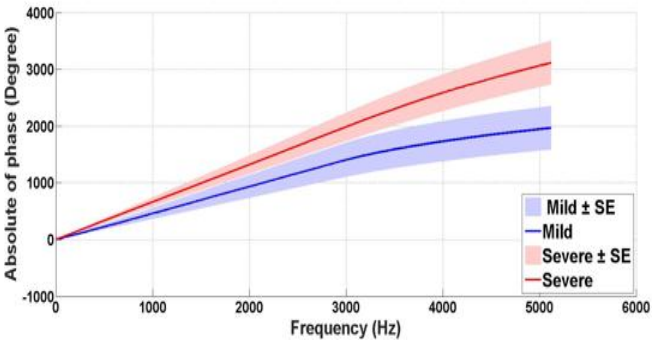

Figure 3: Absolute value of the average phase (solid lines) of expiratory sounds during stage 2 of sleep with their standard error intervals (shadows) in mild (blue) and severe (red) OSA groups

CONCLUSION

In this study, we investigated the changes of spectral and phasic characteristics of expiratory sounds from wakefulness to sleep in mild and severe OSA groups. Our results are indicative that the UA of severe subjects have more thickness and compliance during wakefulness and have an increased stiffness during sleep. The results of phase analysis during wakefulness are also congruent with the increased activity of UA dilator muscles of severe subjects during wakefulness. On the other hand, the results of phase analysis during sleep suggest a change in the UA anatomy of severe OSA subjects during sleep, which leads to a larger delay in transmitting sounds through the UA. Overall, the results of this study are encouraging to use tracheal breathing sounds for inspecting the UA changes due to OSA from wake to sleep. Of course these results have to be confirmed in a larger population.

ACKNOWLEDGMENT

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