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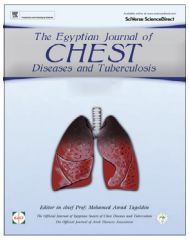

ORIGINAL ARTICLE

Nocturnal heart rate variability analysis as a screening tool for obstructive sleep apnea syndrome

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KEYWORDS

Obstructive sleep apnea syndrome;
Heart rate variability;
Polysomnography;
Time-domain;
Spectral analysis

Abstract *Background:* Recurrent intermittent hypoxia and subsequent increased sympathetic nervous system activity have been adopted as possible mechanisms underlying cardiac rhythm disturbances in obstructive sleep apnea syndrome (OSAS).

Methods: We analyzed nocturnal heart rate variability (HRV) in 80 patients (74 males, 6 females, mean age 47.01 ± 10 yrs) with polysomnographically verified OSAS to assess the changes in nocturnal HRV indices, and to investigate the correlation between these changes to the severity of OSAS. The 80 patients were subdivided into 2 subgroups based upon the severity of OSAS; the first subgroup consisted of 27 patients with mild-to-moderate OSAS, while the second subgroup consisted of 53 patients with severe OSAS. For control group, 25 healthy individuals were included in the study.

Results: In time-domain analysis, the mean of the standard deviation of all RR intervals for all 5-min segments (SDNN index) was significantly different between patients with OSAS and control as well as among different stages of severity of OSAS ($p = 0.02$, and $p = 0.046$, respectively). The standard deviation of all RR intervals (SDNN) was significantly different between patients with OSAS and control ($p = 0.039$). HRV triangular index was significantly different among different stages of severity of OSAS ($p = 0.023$). Frequency-domain variables namely total power, very low frequency (VLF) power, and low frequency (LF) power were significantly increased in patients with OSAS in comparison to control ($p = 0.01$, $p = 0.024$, and $p = 0.018$, respectively), as well as among OSAS subgroups ($p = 0.01$, $p = 0.02$, and $p = 0.04$, respectively). Stepwise multiple logistic regression analysis revealed that AHI correlated positively with SDNN ($r = 0.247$, $p = 0.036$), SDNN index ($r = 0.306$, $p = 0.009$), total power ($r = 0.323$, $p = 0.006$), VLF power ($r = 0.248$, $p = 0.037$), LF power ($r = 0.384$, $p = 0.001$), and LF/HF ratio ($r = 0.342$, $p = 0.004$), but correlated negatively with RR interval ($r = -0.247$, $p = 0.036$).

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Conclusion: OSAS predisposes to clinically significant nocturnal impairment of the cardiac autonomic function as evidenced by nocturnal HRV analysis and this impairment was correlated to the severity of OSAS. Accordingly, HRV can serve as a simple, powerful screening tool for cases with suspected OSAS.

Introduction

Obstructive sleep apnea syndrome (OSAS) is a common disorder affecting at least 2–4% of the adult population. The signs, symptoms and consequences of OSAS are a direct result of the derangements that occur due to repetitive collapse of the upper airway: sleep fragmentation, hypoxemia, hypercapnia, marked swings in intrathoracic pressure, and increased sympathetic activity [1].

Several previous studies have demonstrated relationships between sleep disordered breathing (SDB) and cardiac arrhythmias [2–7]. In OSAS, the recurrence of apneas all through the night elicits a typical and cyclic heart rate pattern consisting of cyclical brady/tachycardia [8]. Proposed mechanisms to explain these changes include the potentially pro-arrhythmic contributions of apnea-induced hypoxia and increased sympathetic nervous system activity [9–12].

The variation in the time period separating consecutive heartbeats has come to be conventionally described as heart rate variability (HRV). Over the last 25 years, HRV analysis has established itself as a non-invasive research and clinical tool for indirectly investigating both cardiac and autonomic system function in both health and disease [13].

Polysomnography (PSG) represents the ''gold standard'' for obtaining a reliable diagnosis of OSAS. Owing to the increased prevalence of OSAS as well as the elevated cost for performing PSG, there is a strong need for the development of reliable low–cost techniques for the diagnosis of this condition.

The main goal of this study was to characterize the changes in nocturnal HRV measurements among patients with polysomnographically verified OSAS and to investigate the correlation between these changes and the severity of OSAS.

Materials & methods

The population under study consisted of randomly selected 80 patients with PSG confirmed OSAS. The 80 patients were subdivided into 2 subgroups; the first subgroup consisted of 27 patients with mild-to-moderate OSAS, while the second subgroup consisted of 53 patients with severe OSAS. Exclusion criteria were hypertension, recent myocardial infarction, heart failure, history of alcohol intake, and history of operations of continuous positive airway pressure (CPAP) treatment for OSAS. For control group, 25 healthy individuals were included in the study. This study was approved by the local ethical committee of the Faculty of Medicine at Ain Shams University.

Polysomnography

Nocturnal full night PSG was performed for OSAS patients and control using a 24 channel computerized system (N4000 Embla, Somnologica, Iceland) including the monitoring of electroencephalogram (EEG), submental and anterior tibial electromyogram (EMG), oxygen saturation, electrocardiogram (ECG), inductance plethysmography of chest wall and abdomen, nasal pressure sensor, and oronasal thermister. The parameters, settings, filters, technical specifications, sleep stage scoring and event scoring were done in accordance with the American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events [14].

Obstructive apnea was defined as the cessation of airflow for at least 10 seconds with persistent respiratory effort. Hypopnea was defined as a 30% or greater decrease in flow lasting at least 10 s and associated with a 4% or greater oxyhemoglobin desaturation. An alternative definition for hypopnea was a 50% or greater reduction in flow lasting at least 10 s and associated with either 3% or greater oxyhemoglobin desaturation or an arousal. Total obstructive apnea–hypopnea index (AHI) was calculated as the number of obstructive apneas and hypopneas per hour of total sleep time (TST). Time in bed (TIB), TST, duration of rapid eye movement (REM) stage, duration of non-rapid eye movement (NREM) stage including both light sleep stages (stage 1 & 2 NREM sleep) and deep sleep stages (stage 3 & 4 NREM sleep), sleep latency as well as REM latency were measured. Arousal index (ArI) was calculated as the number of arousals per hour of TST. The threshold for diagnosis of OSAS was set at an AHI $\geqslant 5$ and the severity of OSAS was arbitrarily defined by cut-off levels of AHI; 5–30 episodes per hour of TST for mild-to-moderate, and more than 30 episodes per hour of TST for severe.

Heart rate variability analysis

Electrocardiographic signals acquired by the PSG were digitalized. The analysis was done only for normal beats. HRV was evaluated by using both time and frequency-domain variables. Time-domain variables include; the average length between each QRS complex (average RR interval), the standard deviation of all RR intervals (SDNN), the mean of the standard deviation of all RR intervals for all 5-min segments (SDNN index), the square root of the mean of the sum of the squares of differences between adjacent RR intervals (RMSSD), the number of pairs of adjacent RR intervals differing by more than 50 ms in the entire analysis interval (NN50 count), the NN50 count divided by the total number of all RR intervals (NN50 of total HR%), standard deviation of average NN interval (SDANN), and the total number of RR intervals divided by maximum height of the histogram excluding boundaries (HRV triangular index). In frequency-domain analysis, the power was calculated for very low frequency (VLF, 0.0033–0.04 Hz), low frequency (LF, 0.04–0.15 Hz), and high frequency bands (HF, 0.15–0.4 Hz) as well as the LF/HF ratio.