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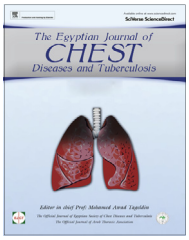

ORIGINAL ARTICLE

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;
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Time-domain;
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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.

Statistical analysis

Statistical data analysis was carried out using the Statistical Package for Social Sciences software (SPSS for Windows, version 17.0; SPSS Inc, Chicago, IL). Descriptive statistics were presented as mean ± standard deviation (SD). Analyses of subgroups according to the severity of OSAS were performed with independent *t*-test. Comparisons between control and different stages of OSAS severity were done using analysis of variance (ANOVA) test or the Kruskal–Wallis test. Correlation analysis was done using Spearman's correlation analysis test. Statistical significance was set at $p < 0.05$.

Results

Of the 80 OSAS study patients, 74 (92.5%) patients were males whereas 6 patients (7.5%) were females. The mean (SD) of age was 47.01 (10) years, with a range of 29 to 73 years. The 25 subjects in the control group were 17 (68%) males whereas the remaining 8 (32%) subjects were females. The differences of the mean values of age (47.01 ± 10 vs. 38.45 ± 16.95, respectively), TIB (463.38 ± 39.79 vs. 464.8 ± 38.21, respectively), and TST (369.82 ± 88.87 vs. 349.32 ± 89.02) were statistically non-significant between patients with OSAS and control. The duration of light sleep (stages 1 & 2 NREM sleep) significantly increased in OSAS in comparison to control ($p = 0.000$), whereas the duration of both REM stage and deep sleep (stages 3 & 4 NREM sleep) significantly decreased in OSAS in comparison to control ($p = 0.001$ and $p = 0.003$, respectively). REM stage latency as well as ArI significantly increased in OSAS in comparison to control ($p = 0.000$ and $p = 0.000$, respectively). Both time and frequency-domain variables of HRV analysis among the patients with OSAS and control are listed in Table 1 with comparison between the two groups. In time-domain analysis, only SDNN and SDNN index were significantly increased in OSAS in comparison to control ($p = 0.039$, and $p = 0.02$, respectively) {Table 1}. Frequency-domain variables namely total power, VLF power, and LF power were significantly increased in OSAS in comparison to control ($p = 0.01$, $p = 0.024$, and $p = 0.018$, respectively) Table 1.

AHI, apnea–hypopnea index; ArI, arousal index; BMI, body mass index; HF, high frequency; HR, heart rate; HRV TI, heart rate variability triangular index; LF, low frequency; NN50, the number of interval differences of successive NN intervals greater than 50 ms; OSAS, obstructive sleep apnea syndrome; REM, rapid eye movement sleep stage; RL, REM latency; RMSSD, square root of the mean squared differences of successive NN intervals; S1, stage 1 sleep; S2, stage 2 sleep; S3, stage 3 sleep; S4, stage 4 sleep; SDANN, standard deviation of average NN interval; SDNN, standard deviation of NN interval; SL, sleep latency; VLF, very low frequency.

Regarding the severity of OSAS; 27 patients (33.75%) had mild-to-moderate OSAS, while the remaining 53 patients (66.25%) had severe OSAS. The mean AHI for mild-to-moderate OSAS subgroup was 15.46 ± 8.2, and 70.18 ± 24.05 for severe OSAS subgroup. The differences of the mean values of age (47.46 ± 9.57 vs. 46.79 ± 10.29, respectively), body mass index {BMI} (35.26 ± 7.49 vs. 37.63 ± 8.83, respectively), TIB (471.28 ± 55.88 vs. 459.58 ± 29.07, respectively), and TST (376.14 ± 96.57 vs. 366.85 ± 85.86) were statistically non-significant between mild-to-moderate, and severe OSAS subgroups. The duration of light sleep (stages 1 & 2 NREM sleep) increased with the increase in the severity of OSAS ($p = 0.001$), whereas the duration of REM stage decreased with the increase in the severity of OSAS ($p = 0.002$). REM stage latency was significantly different among different stages

Table 1 Demography, Sleep profiles, Respiratory events & HRV variables in patients with OSAS & Control.

Variable	OSAS ($n = 80$) Mean ± SD	Control ($n = 25$) Mean ± SD	*P*
Age (yrs)	47.01 ± 10	38.45 ± 16.95	0.264
BMI (kg/m^2)	36.85 ± 8.44	31.87 ± 6.17	0.006
TIB (min)	463.38 ± 39.79	464.8 ± 38.21	0.47
TST (min)	369.82 ± 88.87	349.32 ± 89.02	0.477
S1 & S2 (%)	69.59 ± 15.87	58.35 ± 11.32	0.000
S3 & S4 (%)	14.87 ± 16.48	20.01 ± 14.08	0.003
REM (%)	15.93 ± 9.05	21.58 ± 11.64	0.001
AHI (event/h of TST)	51.72 ± 32.88	2.07 ± 1.41	0.000
ArI (number/h of TST)	47.68 ± 24.04	22.79 ± 17.36	0.000
SL (min)	15.89 ± 15.61	30.84 ± 53.38	0.681
RL (min)	154.3 ± 87.94	117.11 ± 74.52	0.000
RR Interval (ms)	818.93 ± 107.12	837.08 ± 90.5	0.293
SDNN (ms)	101.56 ± 43.45	84.44 ± 30.65	0.039
SDNN index (ms)	78.46 ± 44.01	58.28 ± 20.73	0.02
RMSSD (ms)	66.08 ± 63.17	46.4 ± 36.16	0.165
NN50 count	4627.13 ± 4659.27	5225.72 ± 6614.14	0.43
NN50 of total HR (%)	15.18 ± 16.19	16.18 ± 17.59	0.562
SDANN (ms)	90.14 ± 123.5	79.16 ± 79.31	0.382
HRV TI	18.35 ± 7.18	17.44 ± 6.54	0.099
Average Total Power (ms^2)	14576.89 ± 8131.54	11124.5 ± 2704.69	0.01
Average VLF power (ms^2)	8874.31 ± 6078.2	6460.44 ± 2370.23	0.024
Average LF power (ms^2)	4104.54 ± 3259.66	3055.92 ± 1053.41	0.018
Average HF power (ms^2)	1379.28 ± 894.62	1437.16 ± 743.13	0.755
LF/HF ratio	4.2 ± 4.8	2.87	0.104

Table 2 Demography, Sleep profiles, Respiratory events & HRV variables in subgroups of patients with OSAS.

Variable	Mild-to-Moderate OSAS (n = 27) Mean ± SD	Severe OSAS (n = 53) Mean ± SD	P
Age (yrs)	47.46 ± 9.57	46.79 ± 10.29	0.782
BMI (kg/m^2)	35.26 ± 7.49	37.63 ± 8.83	0.244
TIB (min)	471.28 ± 55.88	459.58 ± 29.07	0.229
TST (min)	376.14 ± 96.57	366.85 ± 85.86	0.676
S1 & S2 (%)	60.94 ± 10.62	73.66 ± 16.37	0.001
S3 & S4 (%)	18.39 ± 13.72	13.21 ± 17.51	0.216
REM (%)	20.67 ± 8.48	13.66 ± 8.49	0.002
AHI (event/h of TST)	15.46 ± 8.2	70.18 ± 24.05	0.000
ArI (number/h of TST)	28.4 ± 18.8	56.37 ± 20.98	0.000
SL (min)	16.5 ± 15.16	15.6 ± 15.97	0.818
RL (min)	101.73 ± 62.01	181.83 ± 87.49	0.000
RR Interval (ms)	841.91 ± 91.27	808.14 ± 113.06	0.215
SDNN (ms)	88.65 ± 34.58	107.61 ± 46.12	0.084
SDNN index (ms)	63.44 ± 32.46	85.51 ± 47.15	0.046
RMSSD (ms)	59.57 ± 54.8	69.14 ± 67.04	0.552
NN50 count	3887.87 ± 4502.38	4974.12 ± 4736.57	0.36
NN50 of total HR (%)	13.49 ± 15.76	15.97 ± 16.48	0.549
SDANN (ms)	131.04 ± 209.94	70.94 ± 34.36	0.054
HRV TI	15.57 ± 4.88	19.65 ± 7.75	0.023
Average Total Power (ms^2)	10895.46 ± 3792.61	16229.78 ± 9007.45	0.01
Average VLF power (ms^2)	6417.91 ± 3232.71	9977.18 ± 6730.38	0.02
Average LF power (ms^2)	2924.18 ± 1026.62	4634.49 ± 3756.19	0.04
Average HF power (ms^2)	1387.27 ± 922.08	1375.69 ± 891.71	0.96
LF/HF ratio	2.82 ± 1.86	4.82 ± 5.55	0.105

of severity of OSAS (p = 0.000). ArI significantly increased with the increase in the severity of OSAS (p = 0.000). Both time-domain and frequency-domain variables of HRV analysis among the different subgroups of OSAS severity are listed in Table 2 with comparison between the different subgroups. In time-domain analysis, SDNN index, and HRV triangular index variables were significantly different among different stages of severity of OSAS (p = 0.046, and p = 0.023, respectively) being greater in severe OSAS subgroup for both SDNN index and HRV triangular index variables (Table 2). Frequency-domain variables namely average total power, average VLF power, and average LF power were significantly different among the subgroups (p = 0.01, p = 0.02, and p = 0.04, respectively) {Table 2}.

AHI, apnea–hypopnea index; ArI, arousal index; BMI, body mass index; HF, high frequency; HR, heart rate; HRV TI, heart rate variability triangular index; LF, low frequency; NN50, the number of interval differences of successive NN intervals greater than 50 ms; OSAS, obstructive sleep apnea syndrome; REM, rapid eye movement sleep stage; RL, REM latency; RMSSD, square root of the mean squared differences of successive NN intervals; S1, stage 1 sleep; S2, stage 2 sleep; S3, stage 3 sleep; S4, stage 4 sleep; SDANN, standard deviation of average NN interval; SDNN, standard deviation of NN interval; SL, sleep latency; VLF, very low frequency.

AHI was correlated with different HRV indices; AHI correlated positively with SDNN (r = 0.247, p = 0.036), SDNN index (r = 0.306, p = 0.009), average total power (r = 0.323, p = 0.006), average VLF power (r = 0.248, p = 0.037), average 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) {Table 3 & Figs. 1–7}.

Table 3 Correlation between AHI & HRV indices.

Variable	AHI (n = 80)	
	r	P
RR inerval (ms)	−0.247	0.036
SDNN (ms)	0.247	0.036
SDNN index (ms)	0.306	0.009
RMSSD (ms)	0.194	0.102
NN50 count	0.185	0.119
NN50 of total HR (%)	0.161	0.177
SDANN (ms)	−0.194	0.102
HRV TI	0.172	0.149
Average Total Power (ms^2)	0.323	0.006
Average VLF power (ms^2)	0.248	0.037
Average LF power (ms^2)	0.384	0.001
Average HF power (ms^2)	−0.087	0.469
LF/HF ratio	0.342	0.004

AHI, apnea–hypopnea index; HF, high frequency; HR, heart rate; HRV TI, heart rate variability triangular index; LF, low frequency; NN50, the number of interval differences of successive NN intervals greater than 50 ms; RMSSD, square root of the mean squared differences of successive NN intervals; SDANN, standard deviation of average NN interval; SDNN, standard deviation of NN interval; VLF, very low frequency.

Discussion

It is well known that in patients with OSAS, the heart rate (HR) usually slows during the apnea and then increases markedly as the subject takes a breath [8]. These respiratory

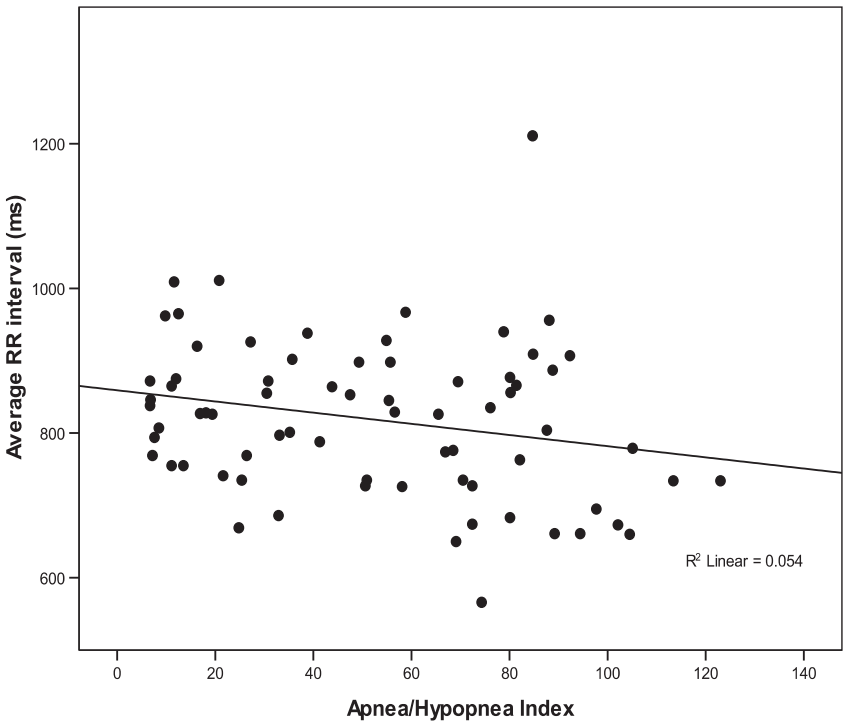

Figure 1 Correlation between AHI and RR interval in OSAS.

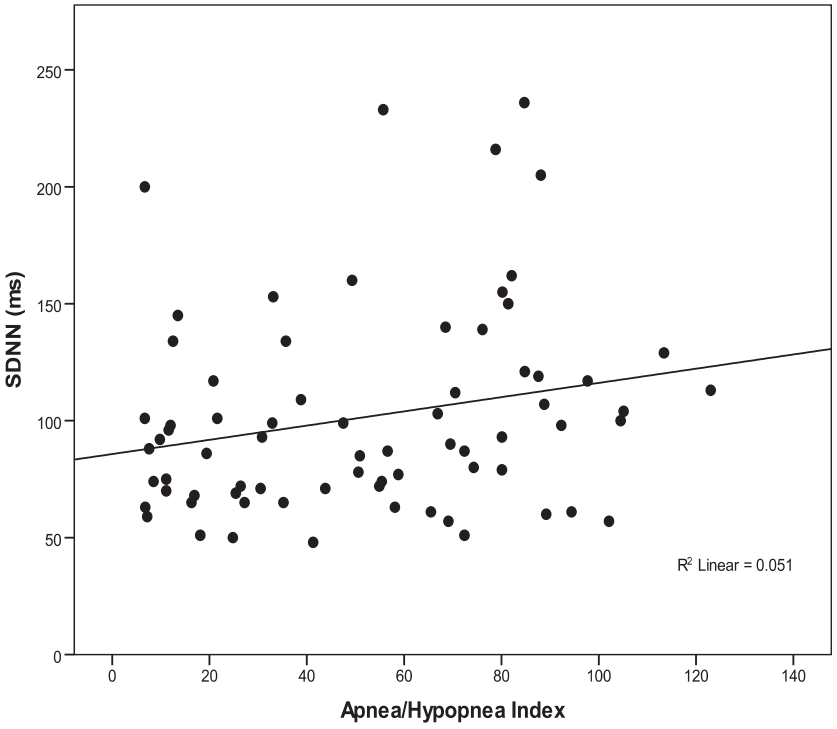

Figure 2 Correlation between AHI and SDNN variable in OSAS.

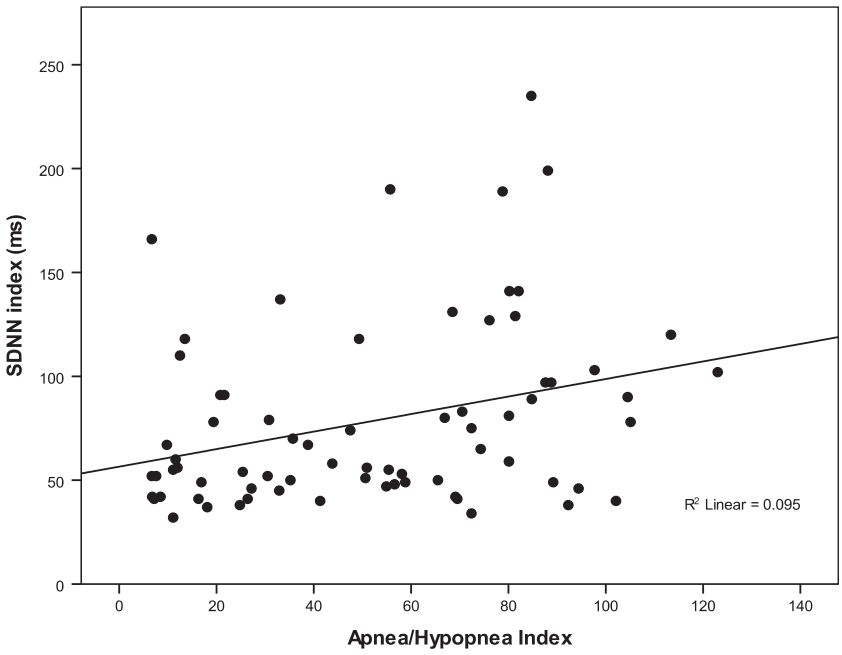

Figure 3 Correlation between AHI and SDNN index in OSAS.

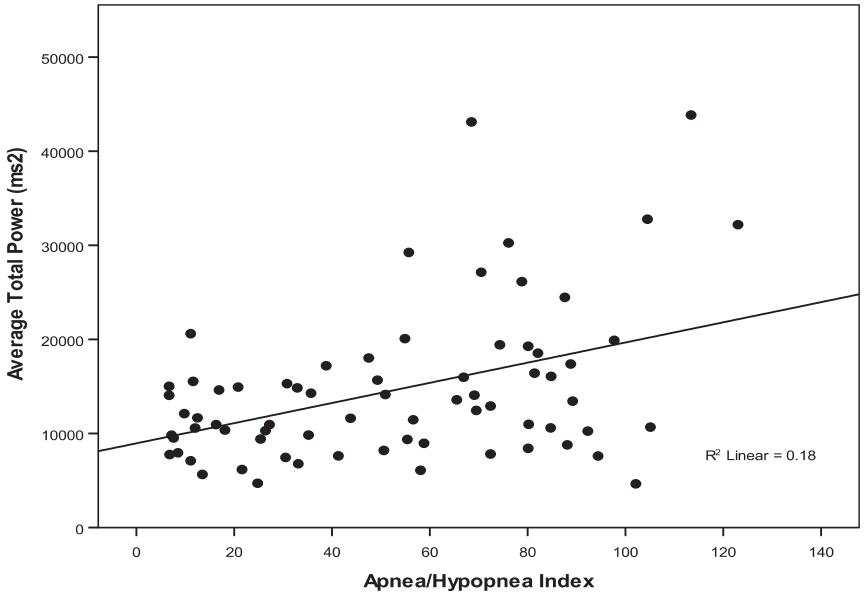

Figure 4 Correlation between AHI and total power in OSAS.

event-related hypoxemia and arousal results in autonomic nervous system (ANS) dysregulation in the form of enhanced sympathetic nervous system activity that is thought to be implicated in the occurrence of HRV [15].

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

Previous studies have documented that the repetitive upper airway obstructions in OSAS during sleep causes an initial stimulation of the parasympathetic ANS, this is followed by an abrupt activation of the sympathetic ANS as a result of occurrence of hypoxemia and hypercapnia (acting through the chemoreflexes). Thus, at night, the alternate strong successive parasympathetic and sympathetic drives dramatically enhance RR variability [16,17].

In the past, time-domain HRV was used frequently. In 1981, Akselrod et al. [18] introduced power spectral analysis of heart rate fluctuations to quantitatively evaluate beat-to-beat cardiovascular control. These frequency domain analyses contributed to the understanding of autonomic background of RR interval fluctuations in the heart rate record [19,20]. Spectral analysis gives selective information on parasympathetic and sympathetic function [12].

The principal findings of this study indicated that most of the frequency-domain variables (or spectral analysis) of

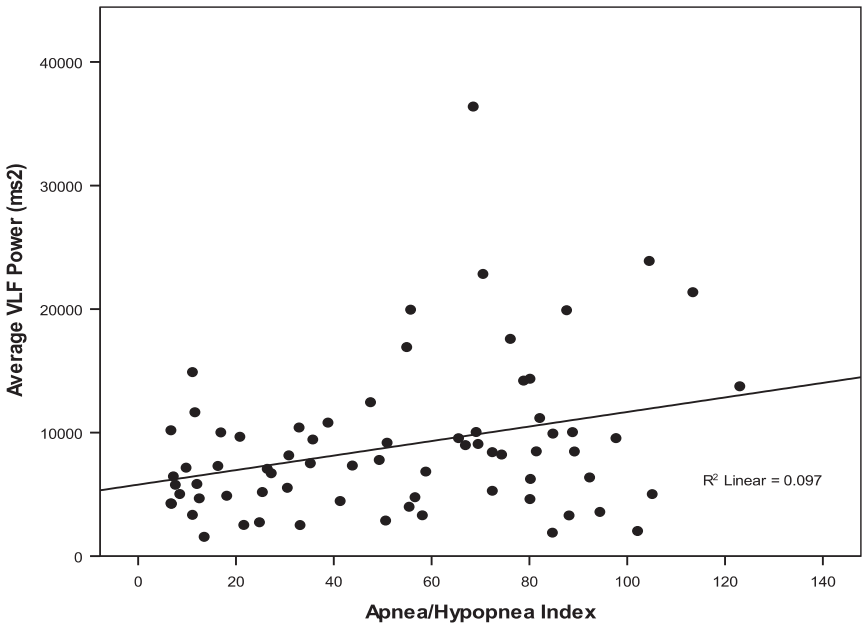

Figure 5 Correlation between AHI and VLF power in OSAS.

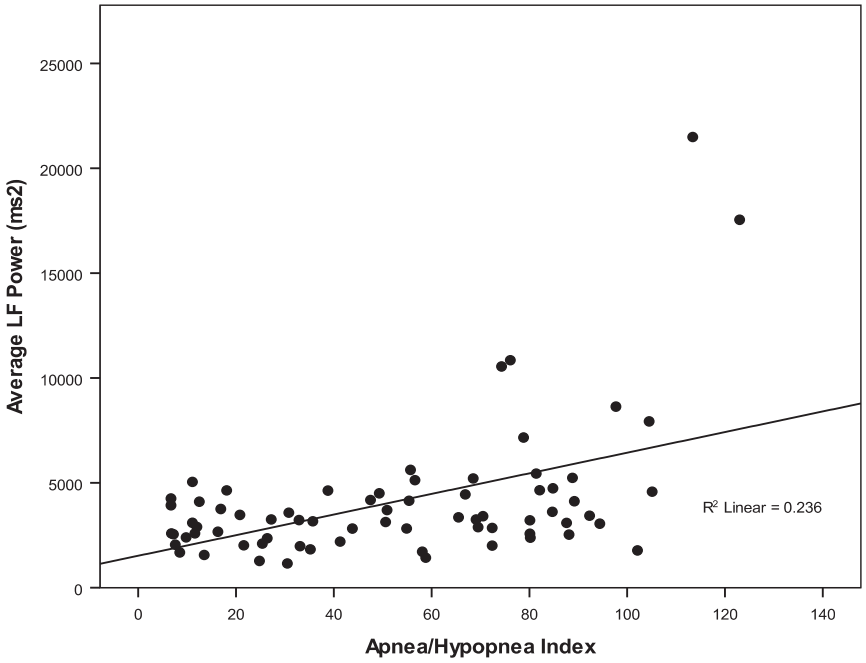

Figure 6 Correlation between AHI and LF power in OSAS.

HRV especially; average total power, average VLF power, and average LF power were the more sensitive measures differentiating patients with OSAS from control being higher in the OSAS group of patients in comparison to control. Moreover, the same variables were also sensitive in discrimination OSAS patients with mild-to-moderate stage of severity from those with severe OSAS being higher in the severe subgroup in comparison to the mild-to-moderate subgroup. On the other hand, time-domain variables failed to discriminate significantly between the two subgroups. Only SDNN and SDNN index were significantly different between patients with OSAS and control. Similarly, only SDNN index and HRV triangular index time-domain variables were able to discriminate significantly between patients with mild-to-moderate OSAS from those with severe OSAS. One of the possible explanations for the reason why most time-domain variables were not better than frequency domain variables in this study is that frequency domain techniques may be better than time-domain analysis in the precise evaluation of changes of sympatho-vagal balance [21]. The LF component has been regarded as a parameter reflecting sympathetic activity. Changes in the LF band spectral power reflect a combination of sympathetic and parasympathetic ANS outflow variations, while changes in the HF band spectral power reflect vagal modulation of cardiac

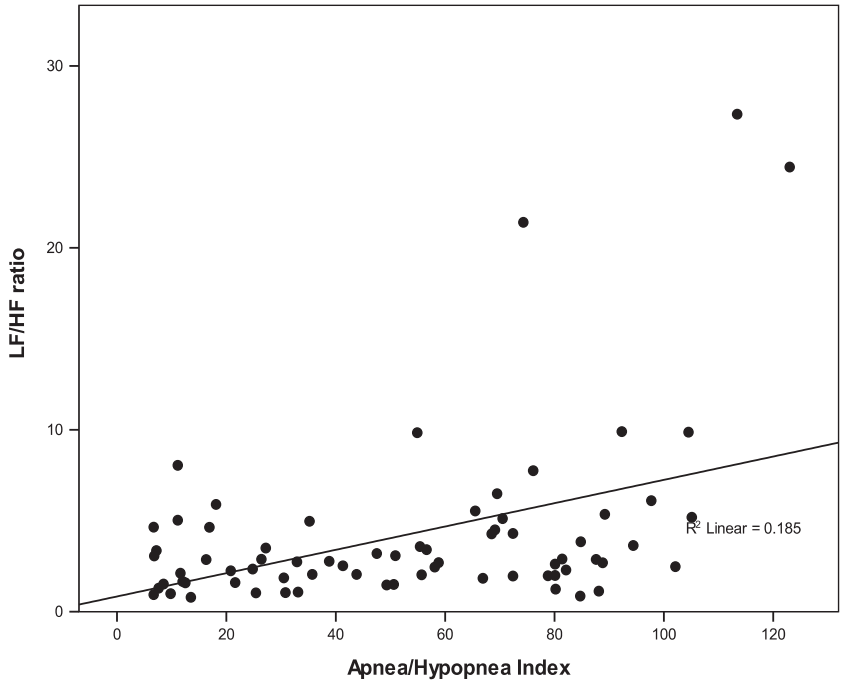

Figure 7 Correlation between AHI and LF/HF variable in OSAS.

activity. The LF/HF power ratio is used as an index for assessing sympatho-vagal balance. Thus, the changes in HRV in this study might suggest that in patients with OSAS, the impairment of the ANS is due to sympathetic stimulation and lack or withdrawal of parasympathetic activity. This is not surprising on basis of the assumption that parasympathetic control for heart rate has a very short latency enabling a beat-to-beat basis change, but synaptic nor-epinephrine mediating sympathetic influence is metabolized relatively slowly [21]. The HRV triangular index tends to correlate with total power in frequency domain analysis. It also reflects the overall amount of variability and has known to be affected by both sympathetic and parasympathetic activity but more influenced by lower bands than higher bands [21]. Although SDNN and SDNN index seemed to carry a degree of significance in OSAS in this study besides being long considered to reflect estimate of overall HRV[12] yet, it is noteworthy that in many previous studies it is indicated that time-domain features are calculated over 24 hours (long term) recordings, while frequency-domain features are calculated over 2–5 min (short term) recordings [22,23] .Thus, SDNN being a time-domain variable, is better to be calculated over a 24-hour period especially that SDNN reflects all the cyclic components responsible for variability in the period of recording and as the period of monitoring decreases, SDNN estimates shorter and shorter cycle lengths and the total variance of HRV subsequently decreases [24] making the changes in variables less valuable. Accordingly, the results of frequency-domain variables in this study should be considered more seriously than those of time-domain variables because the duration of recording was only nocturnal.

In this study, the correlation between the severity of OSAS, represented by AHI, was tested against the different HRV indices; AHI correlated positively with all frequency-domain variables except for average HF power, whereas SDNN and SDNN index of time-domain variables correlated positively with AHI but RR interval correlated negatively. The strongest positive correlation was found in average LF power. Thus, HRV, especially frequency-domain analysis, in OSAS was not just helpful in the identification of patients with OSAS but can also be regarded as a guide for the severity of the disease.

Park and colleagues [25] in a recent study investigated the correlation between the severity of OSAS and HRV indices among 59 patients with moderate and severe OSAS. Their results were closely identical to the results in this study; most of the frequency-domain variables especially total power, VLF power, LF power as well as HRV triangular index time-domain variable, were significantly increased in severe OSAS compared with mild-to-moderate OSAS. Moreover, AHI correlated positively with total power, VLF power, LF power, and LF/HF ratio, but correlated negatively with RR interval. Yet, this study lacked a healthy control group for comparison. In another study, similar results were obtained by Aydin and colleagues [26] who investigated cardiac autonomic activity in 36 patients with OSAS as well as in 34 control, they reported that total power, VLF, LF, and LF/HF ratio were higher in OSAS patients than those of controls, and that LF and LF/HF ratio were increased in severe OSAS group compared with mild OSAS group. Moreover, Guilleminault et al. [8] and Le Heuzey et al. [27] in previous studies suggested using HRV as a tool for preliminary identification of OSAS independently of the assistance of a sleep laboratory. However, some other studies showed contradicting results to those in this study; Yang and colleagues [28] studied the influence of obstructive sleep apnea on heart rate turbulence in 65 patients with OSAS and reported no difference of either time or frequency-domain variables between mild-to-moderate and moderate-to-severe OSAS patients. Gula and colleagues [29] reported that the LF/HF ratio was higher among patients with moderate OSAS compared to those with severe OSAS. These contradicting findings are not surprising because the criteria of the population under study in these

studies are different, the duration of recording for HRV are variable with different methods for data analysis and variable resolution of the devices used.

It is worth mentioning that one of the points of strength in this study was excluding OSAS patients having clinical disorders causing blunted autonomic activity and subsequent affection of HRV. Moreover, the inclusion of normal control healthy group with negative sleep studies in this study acts as a reference that helps to obtain statistically significant results and represents an additional point of strength in this study. Yet, the need for assessment of diurnal HRV as well as testing the impact of CPAP therapy on HRV in patients with OSAS needs to be further investigated. Nevertheless, the unmatching of BMI between OSAS patients and control might represent a limitation in this study.

Conclusion

This study indicates that OSAS predisposes to clinically significant nocturnal impairment of the cardiac autonomic function as evidenced by nocturnal HRV analysis mainly through spectral analysis and to lesser extent through time-domain analysis and this impairment was correlated to the severity of the disease. Accordingly, HRV represents one of the most simple, promising tools for the assessment of ANS in patients with OSAS and can serve as a simple, powerful screening tool for cases with suspected OSAS.

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