Cardiac autonomic control in the obstructive sleep apnea

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Introduction: The sympathetic activation is considered to be the main mechanism involved in the development of cardiovascular diseases in obstructive sleep apnea (OSA). The heart rate variability (HRV) analysis represents a non-invasive tool allowing the study of the autonomic nervous system. The impairment of HRV parameters in OSA has been documented. However, only a few studies tackled the dynamics of the autonomic nervous system during sleep in patients having OSA.

Aims: To analyze the HRV over sleep stages and across sleep periods in order to clarify the impact of OSA on cardiac autonomic modulation. The second objective is to examine the nocturnal HRV of OSA patients to find out which HRV parameter is the best to reflect the symptoms severity.

Methods: The study was retrospective. We have included 30 patients undergoing overnight polysomnography. Subjects were categorized into two groups according to apnea–hypopnea index (AHI): mild-to-moderate OSAS group (AHI: 5–30) and severe OSAS group (AHI > 30). The HRV measures for participants with low apnea–hypopnea indices were compared to those of patients with high rates of apnea–hypopnea across the sleep period and sleep stages.

Results: HRV measures during sleep stages for the group with low rates of apnea–hypopnea have indicated a parasympathetic activation during non-rapid eye movement (NREM) sleep. However, no significant difference has been observed in the high AHI group except for the mean of RR intervals (mean RR). The parasympathetic activity tended to increase across the night but without a statistical difference. After control of age and body mass index, the most significant correlation found was for the mean RR (p = 0.0001, r = −0.248).

Conclusion: OSA affects sympathovagal modulation during sleep, and this impact has been correlated to the severity of the disease. The mean RR seemed to be a better index allowing the sympathovagal balance appreciation during the night in OSA.

Keywords: autonomic nervous system; sleep apnea; heart rate; sleep; circadian

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Obstructive sleep apnea (OSA) is a common disorder affecting at least 2–4% of the adult population (1, 2). It is characterized by the repetitive complete or partial collapses of the pharyngeal airway during sleep.

The diagnosis of OSA requires the presence of nocturnal and diurnal symptoms associated with an apnea–hypopnea index (AHI) greater than 5 events per hour on polysomnography (PSG). The presence of 15 or more obstructive respiratory events per hour of sleep in the absence of symptoms is also sufficient for the diagnostic (1).

It is known that the risk of cardiovascular diseases is increased in OSA (2, 3). The sympathetic activation is considered to be the main mechanism involved in the development of cardiovascular diseases (4, 5).

The heart rate variability (HRV) analysis represents a non-invasive tool allowing the study of the autonomic nervous system (6). HRV can be measured by different methods. Time-domain, frequency (spectral)-domain, and geometrical methods are the most common ones. In the time-domain method, heart rate taken at any time or distance between consequent normal complexes is determined. In the frequency-domain analysis, recordings are evaluated either in 2–5 min or in 24-h time intervals. Frequency-domain method is analyzed by studying the power spectral density. It provides information about the power...
distribution across frequencies (6). Three main spectral
components are calculated.

The main two components are the low-frequency band
(LF) and the high-frequency band (HF). Some investiga-
tors have accepted the LF to be an indicator for sympa-
thetic activity. In contradiction, HF is determined by vaga-
nal activity. The LF/HF ratio has been derived from these values to show sympat-
vagal balance (6). The HRV can be used as a marker of
autonomic modulation of the heart (7). A significant rela-
tionship between the HRV decrease and the cardiovas-
cular risk has been demonstrated and reported in several
diseases (8).

Several studies report a circadian rhythm of HRV. In
fact, compared to wakefulness, the heart rate of a
normal adult slows down during sleep (9, 10). However,
during rapid-eye movement (REM) sleep, the heart rate
does not differ significantly during the wake state, but
during non-REM (NREM) sleep, the heart rate decreases
progressively across the sleep periods. This pattern has
been associated with progressive sympathetic inhibition
during sleep, parasympathetic activation during NREM
sleep, and parasympathetic inhibition during REM sleep
(9). In patients presenting OSA, an imbalanced cardiac
autonomic function has been documented (11–14).

Several studies have shown that the HRV analysis has
the potential of distinguishing the OSA severity. The HRV
has also been proposed as an alternative diagnostic tool
(15, 16). However, few studies tackled the dynamics of the
autonomic nervous system during sleep in patients having
OSA (9).

The aim of this study is to analyze the HRV over sleep
stages and across sleep periods in order to clarify the OSA
impact on cardiac autonomic modulation. The second
objective is to examine the nocturnal HRV of OSA patients
to find out which parameter in time- or frequency-domain
analysis is the best to reflect the severity of symptoms.

Methods

Study design

The study has been planned as a retrospective cohort
study.

Study population

Data of 141 male patients admitted in our sleep labora-
tory for snoring and PSG performed between the period
of January 2006 and March 2013 were analyzed.

The subjects met the following inclusion criteria: male,
aged between 18 and 60 years old, the apnea–hypopnea
index greater than 5 events per hour with clinical symp-
toms or only greater than 15 events per hour (1). The
choice of including men only was an aim to guarantee the
group homogeneity and due to the absence of information
concerning the female hormonal status (17, 18). Patients

known to have cardiovascular, renal, hepatic diseases,
thyroid dysfunction, diabetes, a history of endocrinopa-
thy or drug use that interact with the autonomic nervous
system, a history of operations or CPAP treatment for
OSA, periodic limb movements during sleep (PLMS),
were excluded from the protocol.

Basing on these criteria, we have analyzed the records
of 66 patients included in the protocol. At this stage, HRV
analysis has been performed in 30 patients with their
consent, excluding 36 cases having artifacts and ectopic
heart beats in their ECG or having less than three sleep
NREM–REM cycles. The subjects were categorized into
two groups: mild-to-moderate OSA group (n = 16) and
severe OSA group (n = 14). The first has been defined as
subjects with an AHI ≥ 5 and ≤ 30 and the latter as
subjects with an AHI > 30 (1).

The following clinical data were recorded: the Epworth
Sleepiness Scale and the body mass index (BMI).

Polysomnography

Overnight PSG was performed using DeltaMed (France,
Coherence 4 NT) and Nihon Kohden (Japan, 2011) for
PSG performed after 2012. Sleep states were assessed by
recording biopotentials (electroencephalogram, electro-
myogram, electrooculogram), qualitative recordings of
respiratory effort (piezo sensors), airflow (thermal sen-
sors), and oxygen saturation (pulse oxymetry). The sampl-
ing frequency for the equipment DeltaMed was 256 Hz
and 500 Hz for Nihon Kohden.

Respiratory events were scored as follows: apnea has
been defined as a cessation of the airflow for more than
10 sec. Hypopneas have been defined as a reduction of
more than 50% of the oro-nasal airflow amplitude during
10 sec, accompanied by > 3% desaturation and/or arousal.
Hypopneas are classified as obstructive if there is evi-
dence of upper airway resistance such as snoring, para-
doxical motion in the respiratory bands, or inspiratory
flow limitation on nasal pressure signal (1).

Polysomnographic scoring and staging was based on
Rechtschaffen and Kales study, and episodes of arousals
were assessed according to the guidelines in the previous
studies (19).

HRV analysis

To study the HRV in different cycles, periods of 5 min
have been selected from REM sleep and NREM sleep
stages (represented by stage sleep 2 and slow wave sleep)
in each third of sleep period. Transitions between sleep
stages accompanied by sympathetic activation have been
avoided (20).

For each selected segment, the occurrence of obstruc-
tive respiratory events has been noted. The arousals have
been avoided (9). The selected segments of the ECG have
been extracted and converted into text files.

Electrocardiographic signals acquired by the polysom-
ographic machine were digitalized by the sampling rate

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of 256 Hz for DeltaMed equipment and 500 Hz for Nihon Kohden equipment. The analysis of HRV was performed by the Kubios HRV (version 2.1, Finland) software after research and correction of artifacts, in accordance with the guidelines issued by the European Society of Cardiology and The North American Society of Pacing and Electrophysiology in 1996 (6).

Analyzed time-domain variables were mean RR and RMSSD. Mean RR is the mean of RR intervals. RMSSD is the square root of the mean of the sum of the squares of differences between adjacent RR intervals. 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, 0.15–0.40 Hz). The LF/HF ratio was also included in the statistics. Normalized values of HF (nuHF) and LF (nuLF) bands have been re-calculated using the formulas of nuLF = LF/HF + LF and nuHF = HF/HF + LF.

**Statistical analysis**

Results were expressed as means (SD). When variables showed a normal distribution, a one-way ANOVA was performed, followed by a post hoc test. When variables were not normally distributed, a non-parametric test for independent samples was performed, followed by a Mann-Whitney U test. Bivariate correlations were estimated with Pearson or Spearman coefficients, as appropriate. A value of $p < 0.05$ two-tailed has been considered significant with 95% confidence intervals.

**Results**

**Clinical and PSG characteristics**

The differences of mean age (38 ± 9.4 vs. 42 ± 7 years) and BMI (29 ± 5 vs. 30 ± 5 kg/m) were not significant between the mild/moderate and the severe OSA groups. As indicated in Table 1, the two groups were comparable as far as total sleep time, sleep efficiency, and the percentage of sleep stage 2 are concerned. Compared with the mild/moderate group, the severe OSA group had a higher stage 1 sleep, REM sleep percentage, a higher arousal index, and a lower mean oxyhaemoglobin saturation as determined by pulse oxymetry (SpO2).

**HRV analysis during sleep stages**

In order to determine whether the heart rate autonomic control differs significantly from wake base line for each sleep stage, HRV indices for each sleep stage have been compared to their corresponding wake and other sleep stage measures.

Table 2 shows that with all participants pooled together, during S2, the mean RR increased in relation to the wake stage accompanied with a significant increase of HF and HFnu and a decrease of LFnu and LF/HF ratio compared to REM sleep, the fact that reflects a para-

| Variable     | Mild/Moderate OSA (n = 16) Mean ± SD | Severe OSA (n = 14) Mean ± SD | p  |
|--------------|-------------------------------------|-------------------------------|----|
| SPT (min)    | 417 ± 52.4                          | 426.65 ± 76.27               | > 0.05 |
| SE (%)       | 83.69 ± 6.62                        | 78.1 ± 9.27                  | > 0.05 |
| SL (min)     | 13.28 ± 6.44                        | 11.10 ± 7                    | > 0.05 |
| Arl (total)  | 8.67 ± 6                            | 15.7 ± 8.22                  | 0.015 |
| S1 (%)       | 11.26 ± 3.8                         | 16.5 ± 9.6                   | 0.03  |
| S2 (%)       | 53.3 ± 7.05                         | 58.53 ± 9.3                  | > 0.05 |
| S3 + S4 (%)  | 16.86 ± 5.54                        | 9.4 ± 10.5                   | 0.02  |
| REM (%)      | 18.34 ± 3.6                         | 15.2 ± 8                     | 0.02  |
| Average SpO2 | 95.4 ± 2.09                         | 89.3 ± 3.05                  | 0.02  |
| ODI          | 20.6 ± 7.2                          | 56.63 ± 22.2                 | 0.02  |

OSA, obstructive sleep apnea; SPT, sleep period time; SL, sleep latency; SE, sleep efficiency; S1, stage 1 sleep; S2, stage 2 sleep; S3, stage 3 sleep; S4, stage 4 sleep; REM, REM sleep stage; AHI, apnea–hypopnea index; ODI, oxygen desaturation event index; Arl, arousal index.

sympathetic activation and sympathetic inhibition during NREM sleep.

No significant difference has been observed in the high AHI group except for the mean RR which increased significantly during NREM sleep compared to the wake stage.

Compared to mild/moderate group, the high AHI group showed a significantly higher LFnu and HF/HF during S2 ($p = 0.013$, $p = 0.008$). The HFnu and HF were significantly lower in the severe OSA group during S2 ($p = 0.013$). During SP, the mean RR was higher in mild/moderate group ($p = 0.026$). These results indicated a significant cardiac deceleration during sleep stages in the mild/moderate OSA group compared to high AHI group.

Figures 1 and 2 illustrate the profile of mean RR and the LF/HF ratio along sleep stages.

**The circadian effect**

To study the variation of HRV indices along the night, the sleep period has been divided into three parts. During NREM sleep, the parasympathetic activity tends to increase during the night (Fig. 3a and b). But it was not significant. The same result has been observed during REM sleep (Fig. 3c and d).

**Apnea effect**

By comparing the periods of 5 min, which are marked by the occurrence of respiratory events, with those without any obstructive respiratory event, we have found that the RMSSD was significantly lower in the absence of events (Table 3).

**Correlation between the OSA severity and the HRV indices during sleep**

Bivariate correlation has been estimated first. In the overall study group (n = 30), there was a negative correlation
Table 2. Comparison of HRV indices during sleep stages and wake

| Stage | All participants | Low AHI | High AHI |
|-------|-----------------|---------|----------|
|       | Value           | Multiple comparison | Value | Multiple comparison | Value | Multiple comparison |
|       | (Mean ± SD) | p |       | (Mean ± SD) | p |       | (Mean ± SD) | p |
| Mean RR | W 22 | 935.6 ± 15 | 0.03 | W > S2 | 13 | 903 ± 148.5 | 0.03 | S2 > W | 9 | 797.9 ± 11 | 0.03 | S2 > W |
|       | S2 73 | 975.6 ± 15 | REM > W | 38 | 998.67 ± 16 | 35 | 950.58 ± 133.7 | REM > W |
|       | SWS 30 | 942.08 ± 14 | 19 | 956.41 ± 17 | 35 | 950.58 ± 133.7 | REM > W |
|       | REM 103 | 860 ± 14 | 57 | 969.6 ± 162 | 11 | 899.8 ± 100.6 |
| RMSSD | W 22 | 63.53 ± 43 | > 0.05 | W > S2 | 13 | 52.5 ± 31.1 | > 0.05 | S2 > W | 9 | 48.95 ± 43 | > 0.05 |
|       | S2 73 | 60.39 ± 43 | S2 > REM | 38 | 54.45 ± 39 | 35 | 73.27 ± 45.63 | REM > W |
|       | SWS 30 | 65.16 ± 45 | 19 | 60.64 ± 513 | 11 | 59.97 ± 26.29 |
|       | REM 103 | 51.02 ± 35 | 57 | 59.72 ± 41.9 | 46 | 71.9 ± 48.21 |
| LF | W 22 | 45.57 ± 17 | > 0.05 | W > S2 | 13 | 47.24 ± 17.7 | > 0.05 | S2 > W | 9 | 42.31 ± 24 | > 0.05 |
|       | S2 73 | 44.99 ± 17 | S2 > REM | 38 | 41.88 ± 17 | 35 | 49.58 ± 16.38 | REM > W |
|       | SWS 30 | 50.47 ± 15.7 | 19 | 45.5 ± 19.68 | 11 | 44.01 ± 12.65 |
|       | REM 103 | 45.22 ± 20.2 | 57 | 50.21 ± 15.4 | 46 | 50.79 ± 16.3 |
| HF | W 22 | 42.64 ± 20 | 0.02 | S2 > REM | 13 | 40.7 ± 17.16 | 0.01 | S2 > REM | 9 | 49.89 ± 29 | > 0.05 |
|       | S2 73 | 44.31 ± 21.2 | S2 > REM | 38 | 43.87 ± 20 | 35 | 36.41 ± 20.22 | REM > W |
|       | SWS 30 | 34.50 ± 19.1 | 19 | 47.17 ± 22.8 | 11 | 39.36 ± 17.83 |
|       | REM 103 | 44.46 ± 22.7 | 57 | 35.79 ± 18.7 | 46 | 32.9 ± 19.8 |
| LFnu | W 22 | 52.71 ± 20 | 0.018 | REM > S2 | 13 | 53.19 ± 19.2 | 0.02 | S2 < REM | 9 | 47.36 ± 28 | > 0.05 |
|       | S2 73 | 51.71 ± 20.3 | S2 > REM | 38 | 47.01 ± 20 | 35 | 58.91 ± 19.98 |
|       | SWS 30 | 60.69 ± 19.1 | 19 | 49.84 ± 22.6 | 11 | 54.95 ± 16.19 |
|       | REM 103 | 50.81 ± 23.2 | 57 | 59.43 ± 19.1 | 46 | 62.25 ± 19.2 |
| HFnu | W 22 | 47.18 ± 20 | 0.018 | S2 > REM | 13 | 46.27 ± 20.4 | 0.02 | S2 > REM | 9 | 47.36 ± 28 | > 0.05 |
|       | S2 73 | 48.22 ± 2.3 | S2 > REM | 38 | 52.9 ± 20.0 | 35 | 40.97 ± 19.97 |
|       | SWS 30 | 39.21 ± 19 | 19 | 50.09 ± 22.5 | 11 | 44.98 ± 16.18 |
|       | REM 103 | 49.06 ± 23.2 | 57 | 40.48 ± 19.0 | 46 | 37.63 ± 19.2 |
| LF/HF | W 22 | 2.07 ± 0.4 | 0.018 | REM > S2 | 13 | 1.62 ± 0.8 | 0.02 | REM > S2 | 9 | 1.87 ± 0.6 | > 0.05 |
|       | S2 73 | 1.64 ± 0.3 | REM > SWS | 38 | 1.62 ± 0.5 | 35 | 2.56 ± 0.5 |
|       | SWS 30 | 2.84 ± 0.5 | 19 | 1.67 ± 0.38 | 11 | 2.87 ± 0.4 |
|       | REM 103 | 1.72 ± 0.4 | 57 | 2.71 ± 0.6 | 46 | 3.01 ± 0.5 |

*HRV indices for each sleep stage were compared to their corresponding wake and other sleep stage measures by ANOVA test followed by post hoc test, when variables were normally distributed. When variables were not normally distributed, a non-parametric test for independent samples was performed, followed by a Mann-Whitney U test.*

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between AHI and RR intervals ($p = 0.042, r = 0.224$), HF ($p = 0.001, r = -0.218$) and HFnu ($p = 0.001, r = -0.226$).

A positive correlation has been found for LFnu ($p = 0.001, r = 0.225$), LF ($p = 0.017, r = 0.16$) and LF/HF ratio ($p = 0.001, r = 0.226$).

After adjustment of age and BMI, the most significant correlation relationship found was for the mean RR (Table 4).

**Discussion**

The main aim of this study was to test the hypothesis that OSA was associated with an impaired cardiac autonomic modulation over the night. The second hypothesis was that HRV indices were affected by OSA severity.

Our results supported the first hypothesis. First, the mild/moderate OSA group presented a parasympathetic activation from wake to NREM sleep and a reversal to a balance of less parasympathetic modulation during REM sleep. This pattern is consistent with the findings of other studies performed in healthy subjects about the cardiac autonomic modulation along the sleep stages. These studies showed a reduced sympathetic outflow and increased parasympathetic modulation shift from wake to NREM sleep, a pattern reversed by entering in REM sleep (21–25).

The changes in the sympathovagal balance could be driven by oscillations in the metabolic demand during sleep, which markedly decreases during deep sleep and increases during REM sleep (26).

However, this shift of cardiac autonomic modulation is impaired among OSA severe group which showed no statistical difference between the sleep stages except for the mean RR. Our data suggest a more parasympathetic modulation reduction during sleep in the high AHI group. These findings are similar to those reported by other...
studies (27–29). Second, no statistically significant difference was found between the values of HRV indices across the night. However, the parasympathetic activity tended to increase with the progression of sleep during NREM and REM sleep associated to the decrease of sympathetic activity. These results suggest a circadian effect on the autonomic nervous system and a better quality of sleep reached progressively during the night. Several studies reported a circadian rhythm of heart rate with lower levels during the night compared to the day with a peak level at awakening (10, 30). This rhythm is driven by an endogenous central pacemaker located in the suprachiasmatic nucleus of the hypothalamus (10).

Our results are consistent with the findings of some studies performed during normal sleep which could not identify a clear sympathovagal modulation across the night (21, 25).

There are some data concerning the dynamic of HRV of OSA patients during sleep. Our findings are partially similar to those of Da Silva et al. (9) who showed a significant cardiac deceleration during the last quartile of the night, more pronounced in the high AHI group.

Table 3. Comparison of HRV during free-event periods to those with obstructive events

| Variable | Free-event periods (n = 74) | With event periods (n = 123) | p |
|----------|-----------------------------|-------------------------------|---|
| Mean HR  | 66.36 ± 10.82               | 65.21 ± 9.05                 | 0.23 |
| Mean RR  | 935.4 ± 162.28              | 950.88 ± 142.26              | 0.08 |
| RMSSD    | 49.28 ± 32                  | 73.2 ± 48.6                  | 0.04 |
| LF       | 47.24 ± 18.63               | 47.94 ± 15.86                | 0.4 |
| HF       | 41.98 ± 21.04               | 38.2 ± 20.56                 | 0.07 |
| LFnu     | 53.61 ± 21.5                | 57.17 ± 20.06                | 0.08 |
| HFnu     | 46.3 ± 21.49                | 42.72 ± 20.05                | 0.06 |
| LF/HF    | 2.41 ± 0.3                  | 2.25 ± 0.4                   | 0.1 |

Table 4. Partial correlation of AHI and HRV indices controlling age and body mass index of the subjects with obstructive sleep apnea

| Variable | p     | r_p  |
|----------|-------|------|
| Mean RR  | <0.0001 | -0.248 |
| LF       | 0.024  | 0.144 |
| LFnu     | 0.025  | 0.143 |
| HF       | 0.037  | -0.133 |
| HFnu     | 0.024  | -0.144 |
| LF/HF    | 0.1    | 0.12  |

Fig. 3. Profiles of the mean of the RR intervals (mean RR) and the low frequency/high frequency (LF/HF) ratio for the different parts of sleep period during NREM sleep (a, b) and REM sleep (c, d). Error bars represent ± standard error of the mean.
A mechanism of parasympathetic overcompensation was evoked. This mechanism was associated with the increase of hydraulic pressure and the stimulation of carotid–aortic chemoreceptor in response to apnea (9).

The second hypothesis of this study was that HRV indices were correlated to the severity of OSA. A significant but small correlation was found for the frequency HRV parameters. Most of the temporal indices failed to show a significant correlation except for the mean RR. After control of age and BMI, the mean RR was found to be the most affected by AHI. Our results are partially similar to those of Zhu et al. (31) who found a negative correlation with the mean RR, suggesting it to be the best index to assess the sympathetic–parasympathetic balance over the night in OSA. However, no statistical difference was found for the spectral indices in their study. In some other previous studies, LF/HF was found to present the most significant correlation with the AHI (28, 32, 33).

Limitations
This study is one of few that have assessed the HRV by sleep stage and at the different parts of night with the presence of sleep apnea syndrome. However, it presents quite a few limitations. It is a retrospective study with a reduced sample. The interpretation of HRV parameters during the wake period may be biased due to the lack of control over environmental factors. The population study was based only on men, therefore not allowing a study of the differences according to gender. The choice of including men only has been previously explained.

One of the main limits of our study concerns measurement conditions of HRV during sleep.

The spectral study requires the respect of the signal stationary condition, which should vary little through time. Respecting this condition becomes an issue when studying parameters during sleep with the presence of OSA (14). Indeed, apnea induces tachycardia during arousal proceeding by reinforcement of respiratory sinus arrhythmia (14).

Measuring the HRV during this period does not respect the stationary condition. In spite of this, some authors have studied the effect of the respiratory events on HRV like Guilleminault et al. (34) who have found a significant increase of LF/HF and decrease of HF after the event.

Some other authors have chosen to study the HRV during the periods without respiratory events (28). This choice is also problematic because it is very hard for some patients to find free-event periods, without mentioning the risk of not scoring subclinical events (14).

The objective of this work was to study the modulation of the HRV parameters during different sleep stages at night aiming for studying the dynamics of the autonomous nervous system during the sleep cycles. It was difficult to find periods without respiratory events with a number of patients. These periods were included, while noting the onset of the obstructive events. The comparison of obstructive events with those without it shown in the RMSSD was significantly higher in the presence of respiratory event.

The increase of the RMSSD, which habitually testifies to the reinforcement of the parasympathetic nervous activity modulated by respiration, may be attributed to modifications in the cardiac rhythm that accompany these episodes. Its inability to distinguish between increasing HRV due to an erratic rhythm and an increase in parasympathetic activity makes it problematic and not reflective of the parasympathetic nervous system (14).

No other statistical difference was found including the mean RR. This may be attributed to the presence of no scored subclinical events.

Conclusion
This study is one of few investigating the HRV by sleep stage at different parts of night with the presence of OSA. It shows the effects of OSA on sympathovagal modulation during sleep and this impact was correlated to the severity of the disease.

The mean RR seemed to be a better index allowing the sympathovagal balance appreciation during night in the OSA. Our findings could be used further to confirm the contribution of HRV parameters as a screening tool in the OSA.

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The authors declare they have no conflicts of interest concerning this article.

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