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ORIGINAL RESEARCH

Changes in Heart Rate Variability and Baroreflex Sensitivity During Daytime Naps

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Background and Objectives: Changes in autonomic cardiac activity during night sleep are well documented. However, there is limited information regarding changes in the autonomic cardiac profile during daytime naps. Heart rate variability (HRV) and baroreflex sensitivity (BRS) are reliable measures of autonomic cardiac activity. The purpose of this study was to determine the changes in HRV and BRS during daytime naps in healthy men.

Methods: This was a cross-sectional study of 25 healthy men. Polysomnographic recording with electrocardiogram monitoring was conducted for all volunteers during a 50–80 min nap between 3.30 pm and 5.30 pm. Five-minute segments during pre-nap wakefulness, non-rapid eye movement (NREM) sleep stages (N1, N2, and N3), rapid eye movement (REM) sleep stage, and post-nap wakefulness were used to measure changes in the variation in HRV parameters, including inter-beat interval (RR-interval), total spectral power (TP), high-frequency power (HF), low-frequency power (LF), and low frequency/high-frequency ratio (LF/HF). BRS was also measured for 10 min during pre- and post-nap wakefulness using finger arterial pressure measurement (Finometer Pro ®).

Results: HRV increased significantly during NREM sleep compared with that during pre-nap wakefulness ($p < 0.05$), as reflected by RR-interval prolongation, higher HF, and increased HF_{nu} (normalized units). Furthermore, there was a parallel reduction in TP, LF, and LF/HF ratio during NREM sleep, indicating parasympathetic predominance over cardiac autonomic activity. HF and HF_{nu} were significantly reduced during REM sleep compared with that during NREM sleep ($p < 0.05$). BRS did not show significant differences between pre- and post-nap wakefulness.

Conclusion: We observed a progressive increase in parasympathetic activity during daytime sleep as NREM sleep deepened compared with that during wakefulness and REM sleep. Daytime nap may have a favorable cardiovascular impact.

Keywords: daytime naps, HRV, baroreflex sensitivity, polysomnography, autonomic activity

Introduction

The autonomic nervous system (ANS) mediates several physical and psychological changes such as adaptation to anxiety, stress, activity of daily living, and sleep. The initiation of sleep and the transition between sleep stages are partially controlled by ANS.[1] In addition, wakefulness as well as each sleep stage have its own distinguishable pattern of autonomic activity, resulting in corresponding changes in cardiovascular and respiratory functions.[2,3] Therefore, the transition between NREM sleep, REM sleep, and arousal state are associated with fluctuations in arterial blood pressure (BP), heart rate, and respiratory pattern.[4] A reduction in BP during NREM sleep is associated with an increase in the heart period; however,

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these changes are restored during REM sleep to the levels at wakefulness. Brief periods of wakefulness after sleep onset (WASO) during NREM sleep are also characterized by distinct stereotypic cardiovascular changes, starting with a decrease in the heart period, followed by a burst elevation in BP, following which the heart period and BP return to baseline levels. A similar sequence of events is reported to occur during BP elevation during wakefulness and REM sleep.[5] Respiration exhibits regular slow rhythm during NREM sleep and is predominantly under chemical control; in contrast, it becomes irregular during REM sleep and depends on behavioral factors.[4]

These changes in the autonomic function during wakefulness and different sleep stages are controlled by a complex mechanism involving interaction between several neuronal centers located in the hypothalamus and brainstem. Various excitatory neurotransmitters (e.g., glutamate) and inhibitory neurotransmitters (e.g., γ-aminobutyric acid) are released by these centers during arousal and different sleep stages, producing alterations in the autonomic function in a state-dependent manner. Furthermore, inputs from orexin-producing hypothalamic neurons, cholinergic neurons, and aminergic neurons exert a modulating effect on these neuronal circuits. The overall interaction between these neuronal groups mediates the central sympathetic and parasympathetic outputs to the cardiovascular and respiratory system.[3,5] The physiological decrease of BP during NREM sleep is caused by generalized cardiovascular suppression and baroreflex resetting as a consequence of central autonomic effect with parasympathetic dominance. In contrast, BP increases during REM sleep because of sympathetic outflow changes, thereby causing a net effect of increased peripheral vascular resistance.[5]

Heart rate variability (HRV) is a sensitive, noninvasive measurement of ANS activity, and it is used to assess cardiac autonomic activity as it reflects heartbeat regularity.[6] HRV is a result of the dynamic balance between the two main components of ANS: the sympathetic and parasympathetic nervous systems. The parasympathetic nervous system can act quickly to lower the heart rate (HR), whereas sympathetic activity acts relatively slower to increase HR. Therefore, an elevation in HRV may represent an increase in parasympathetic activity, whereas a low-value HRV may reflect elevated sympathetic action.[7,8]

The frequency domain of HRV analysis has two major components: high frequency (HF, 0.15–0.4 Hz) and low frequency (LF, 0.04–0.15 Hz) bands.[9] The HF component mirrors the parasympathetic modulation of the heart and is associated with respiration. The LF component is considered to reflect sympathetic activity, but this idea remains highly disputable.[9,10] It is well documented that a specific autonomic cardiac pattern, reduced HR and increased HF activity, is associated with the initiation of sleep in healthy young adults.[11,12] Therefore, sympathetic control during wakefulness shifts to parasympathetic control during sleep, subsequently altering HRV during sleep in different stages.[8]

Reduced HRV is an independent risk factor for cardiovascular morbidity and mortality.[7] The relationship between nighttime sleep, autonomic activity, and cardiovascular risk has been well studied; short nighttime sleep (<5 h) or long nighttime sleep (>9 h) is correlated with increased cardiovascular risk.[13] Previous studies demonstrated that HF activity remains elevated and RR intervals continue to lengthen during NREM sleep, whereas LF power decreases throughout the NREM sleep stages.[12,14] Conversely, during REM sleep, there is an increase in the LF component and a reduction in HF power.[15] These findings suggest the predominance of vagal/parasympathetic activity during NREM sleep, leading to more cardiovascular stability compared with that during REM sleep stage, which is predominated by sympathetic activity and cardiovascular instability.[8]

Few studies have investigated the autonomic cardiac output during daytime naps compared with that during nocturnal sleep.[2,16] Furthermore, the relationship between daytime nap and cardiovascular risk is still disputable. A large prospective study of 3462 participants showed that 1–2 naps per week reduce the risk for cardiovascular events.[17] Another study showed that daytime nap in apparently healthy individuals is inversely associated with the risk of coronary diseases.[18] In contrast, frequent napping was found to be associated with an increased risk for cardiovascular mortality.[19–21]

Nighttime sleep has been extensively researched, and its beneficial cardioprotective effect is well established. However, the current literature suggests that daytime nap is associated with less restorative and protective benefits; these discrepancies in the cardioprotective effect between nighttime and daytime sleep could be attributed to the circadian physiological influence on both sleep and autonomic functions. According to the two-process model of sleep regulation, the circadian process (C) makes daytime nap with less sleep propensity.[22] In addition, many cardiovascular activities, such as electrocardiographic records, cardiac conductive physiology, HRV, and BRS, exhibited

diurnal variations under the influence of circadian autonomic and hormonal changes as well as external stimuli.[23]

Baroreflex sensitivity (BRS) is a concept of HR reflex resulting from stimulation or de-stimulation of baroreceptors in the walls of blood vessels to regulate BP.[24] Baroreflex control is an indicator of autonomic effects on the heart and thus can reflect many clinical aspects. The effect of sleep on BRS remains unclear and is still under investigation.[25] For instance, Conway et al reported a significant increase in BRS during sleep stages,[26] whereas Nakazato et al reported inconclusive results.[27] Thus, the impact of daytime nap on BRS remains unknown.

Therefore, the aim of this study was to investigate changes in HRV and BRS during daytime naps in healthy men. We hypothesized that the daytime nap would be characterized by a pattern of autonomic activity dominated by parasympathetic activity, similar to that observed during nighttime sleep.

Methods

Participants

This was a cross-sectional study conducted in the sleep laboratory at Imam Abdulrahman Bin Faisal University (IAU) between October 22, 2019, and March 9, 2020. Twenty-five healthy men aged 18–25 years were enrolled in the study. Individuals were excluded if they suffered from sleep disorders (e.g., obstructive sleep apnea, insomnia, and narcolepsy), were using any sleep aid medications (e.g., benzodiazepines and melatonin), or had known cardiovascular, respiratory, neurological, or psychological illnesses. Other exclusion criteria included shiftwork, caffeinated beverage consumption within the past 24 h, use of recreational drugs, intense regular physical exercise, and any major life stress (such as examination periods, divorce, emotional and social problems).

Sleep deprivation was assessed in the participants using a sleep diary for 2 weeks before presentation to the lab. Subsequently, any subject with average sleep duration of <6 h was excluded. A validated Pittsburgh Sleep Quality Index (PSQI) questionnaire was used to evaluate sleep efficiency in each participant before enrollment in the study; those with low sleep quality were excluded (global score ≥ 17). All participants provided written informed consent, and this research was performed in accordance with the Helsinki Declaration of Ethics in Human Studies.[28] The research was approved by the IAU Research Review Board (IRB-2020-069-Med).

Procedure

Participants were advised to sleep at least 7 hours the night prior to the experiment and to avoid caffeinated drinks and vigorous activity during the day before the study. On the day of the experiment, each participant arrived at the lab at 2:30 pm. At 3:30 pm, all electrodes were placed on the participants for PSG recording to determine different sleep stages, with parallel ECG monitoring for HRV calculation. Each participant took a 50–80-min nap between 3.30 pm and 5.30 pm. In addition, a continuous finger arterial BP measurement instrument (Finometer) was placed for 10 min before and after the nap to measure BRS.

Polysomnographic Recording

The study focused on daytime naps. Sleep stages were recorded using a Philips Respironics ALICE 6 Polysomnography (PSG) machine with computerized software (Sleepware G3; version 3.7.1). The montage included bilateral electrooculography (EOG: L-EOG-M2 and R-EOG-M2), six electroencephalograms (EEG: F3M2, F4M1, C3M2, C4M1, O1M2, O2M1), and submental bipolar electromyography (EMG). Data were scored in 30-s epochs based on the American Academy of Sleep Medicine guidelines for the staging of sleep;[29] Stage N1 was scored if the alpha rhythm was attenuated and replaced by low-amplitude mixed frequency activity for more than 50% of the epoch, stage N2 was scored by the presence of K-complex or sleep spindle, stage N3 was scored if there were waves of frequency of 0.5–2 Hz and peak-to-peak amplitude of >75 μV, and REM stage was scored if there were rapid eye movements in the EOG accompanied by low chin EMG tone.

A single trained sleep expert conducted the scoring to reduce potential variation. Score variables were total sleep time (TST), sleep efficiency (SE), wakefulness after sleep onset (WASO), sleep latency (from light off to N1), NREM sleep stages (N1, N2, and N3), and REM sleep stage. Data were recorded in a softcopy datasheet and stored on a Microsoft Excel sheet on a secured computer.

Measurement of Heart Rate Variability

ECG was recorded using a Philips Respironics ALICE 6 PSG machine and software (Sleepware G3; version 3.7.1) using the updated Einthoven Lead I configuration, with a 256-Hz sampling rate. The HRV module of LabChart Pro software (version 8.1.13) was used to analyze the HRV across different stages of sleep. A 5-min segment of each sleep stage was selected and visually examined to remove

artifacts or ectopic beats and to insert missing beats. The following HRV parameters were analyzed: RR-interval as a major time-domain index for overall HRV, LF to reflect sympathetic activity, HF and HF in normalized units (HF_{nu}), which reflects the parasympathetic activity, and total power (TP), a representation of total HRV in the frequency domain.[2,30] Additionally, the LF/HF ratio was used to indicate sympathetic–parasympathetic balance.[30] Respiratory rate is a confounding variable that may affect HRV;[31] thus, a respiratory belt was used to simultaneously calculate the respiratory rate with HRV measurement.

Measurements of Baroreflex Sensitivity

BRS was measured by analyzing instantaneous changes in HR in response to changes in arterial BP.[32]

HR and arterial BP were measured by recording finger arterial BP using Finometer Pro® (FMS, Amsterdam, Netherlands) adjusted against an oscillometric BP cuff. The Finometer ® was applied 10 min before and after a nap period while participants were in the supine position. BRS was automatically calculated using a dedicated software (PRVBRS; FMS, Amsterdam, Netherlands) using cross-correlation method, and data were analyzed using BeatScope software (version 1.1.0.6). For each series of RR-interval/systolic BP (SBP), a linear regression model was applied, and the mean of each slope was used to calculate the average of all slopes to determine the BRS.[33]

Data Analysis

The frequency domain of HRV and RR-interval were analyzed for each wake–nap stage, ensuring that each epoch was free from any artifact, stage transitions, or arousal. Windows were identified and averaged for N1, N2, N3, and REM sleep stages. An additional 5 min of pre-nap and 5 min of post-nap wakefulness were also analyzed.

A repeated-measures analysis of variance was applied for each HRV variable (RR, TP, HF, HF_{nu}, LF, and LF/HF ratio) as well as respiratory rate, with pre-nap wakefulness, N1, N2, N3, REM sleep, and post-nap wakefulness as the within-subjects' factors. The Greenhouse–Geisser correction was applied for violated sphericity assumptions. Tukey's honest significant difference test with Bonferroni correction was used for post hoc comparisons. Student's *t*-test was used to compare BRS between pre- and post-nap wakefulness. Data are expressed as the mean ± standard error of the mean. For skewed data, natural log transformation (ln) was applied to assume a normal distribution. Data were considered statistically significant when $p < 0.05$. All statistical analysis was performed using SPSS statistics version 24.0 (IBM corporation, Armonk, NY, USA).

Results

Twenty-five healthy men were recruited in this study. The demographic and experimental daytime nap characteristics are shown in Table 1.

Changes in Heart Rate Variability Across Daytime Naps

There were statistically significant effects of different wake–nap stages in the following HRV measures: lnRR, lnTP, lnHF, HF_{nu}, lnLF, and LF/HF ratio ($p < 0.05$). Post hoc comparisons revealed significant lengthening of lnRR during each sleep stage compared with that during pre-nap (pre-nap vs. N1, $p = 0.004$; pre-nap vs. N2, $p = 0.016$; pre-nap vs. N3, $p = 0.001$). There was a significant reduction in lnRR during post-nap wakefulness compared with that during the N1, N2 and N3 stages (post-nap vs. N1, $p = 0.007$; post-nap vs N2, $p = 0.007$; post-nap vs. N3, $p < 0.001$). lnRR was higher during N3 than during N2 ($p = 0.018$) (Figure 1A). lnTP was the highest during post-nap than during other stages and was significantly greater than that during pre-nap ($p = 0.024$), N1 ($p = 0.024$), N2 ($p = 0.042$), and N3 ($p = 0.04$) (Figure 1B). lnHF power was significantly increased during N3 compared with that during pre-nap wakefulness ($p = 0.024$) (Figure 1C). lnLF power was increased significantly during post-nap compared with that during pre-nap ($p = 0.002$) and N3 ($p = 0.005$) (Figure 1D). We found that HF_{nu} was significantly increased during N3 compared with

Table 1 Demographic and Daytime Nap Characteristics

Demographics	Mean ± SEM
Age (years)	23.36 ± 0.3
BMI (kg/m^2)	26.63 ± 1.4
Nap architecture characteristics	
TST (min)	58.84 ± 5.1
N1 (min)	8.04 ± 1.4
N2 (min)	24.72 ± 4.1
N3 (min)	20.54 ± 3.7
REM (min)	4.27 ± 1.4
WASO (min)	26.09 ± 4.1
SL (min)	10.99 ± 2.4
SE (%)	57.87 ± 5.6

Note: Data are expressed as means ± SEM unless stated.
Abbreviations: TST, total sleep time; N1, stage 1 non-rapid eye movement sleep; N2, stage 2 non-rapid eye movement sleep; N3, stage 3 non-rapid eye movement sleep; REM, rapid eye movement sleep; WASO, wakefulness after sleep onset; SL, sleep latency; SE, sleep efficiency.

that during pre-nap wakefulness ($p = 0.009$), N1 ($p = 0.004$), and post-nap wakefulness ($p < 0.001$). In addition, HF_{nu} was significantly reduced during post-nap wakefulness compared with that during other stages (post-nap vs. pre-nap, $p = 0.012$; post-nap vs. N1, $p = 0.042$; and post-nap vs. N2, $p < 0.001$) (Figure 1E). The LF/HF ratio was significantly reduced during N3 compared with that during pre-nap ($p = 0.040$), N1 ($p = 0.011$), and post-nap ($p = 0.005$). Another significant reduction of the LF/HF ratio was observed during N2 compared with that during N1 ($p = 0.024$). The highest LF/HF ratio was observed during post-nap and was significantly greater than pre-nap ($p = 0.047$) and N2 ($p = 0.008$) (Figure 1F).

All analyses were conducted on 19 participants who entered five wake–nap stages (pre-nap wakefulness, N1, N2, N3, and post-nap wakefulness). Interestingly, nine participants entered REM sleep stage during the 50–80-min nap. Therefore, we conducted additional analysis for the nine participants who had REM sleep activity (Figure 2) to investigate changes in HRV parameters during REM sleep.

Two parameters of HRV (lnHF and HF_{nu}) exhibited significant changes during the REM sleep stage. lnHF was significantly reduced during the REM sleep stage compared with that during N3 ($p = 0.046$, Figure 2A), and HF_{nu} was also significantly decreased in the REM sleep stage compared with that during both N2 ($p = 0.048$) and N3 ($p = 0.011$, Figure 2B). The respiratory rate did not differ across the stages ($p = 0.053$), suggesting that respiratory rate did not affect changes in HRV in the different wake–nap stages.

Changes in Baroreflex Sensitivity Between Pre- and Post-Nap Wakefulness

There were no significant changes in SBP, diastolic BP (DBP), and BRS between pre- and post-nap wakefulness (Figure 3A–C).

Discussion

In this study, we aimed to characterize the changes in HRV and BRS in different wakefulness–nap stages in healthy adults. Our findings showed that the HRV profile changed dynamically during daytime nap. Compared with that during pre-nap, discrete changes in cardiac autonomic activity were observed across various sleep stages; this is consistent with the findings of several previous studies. One study found that compared to quiet wake, the R-R intervals and the

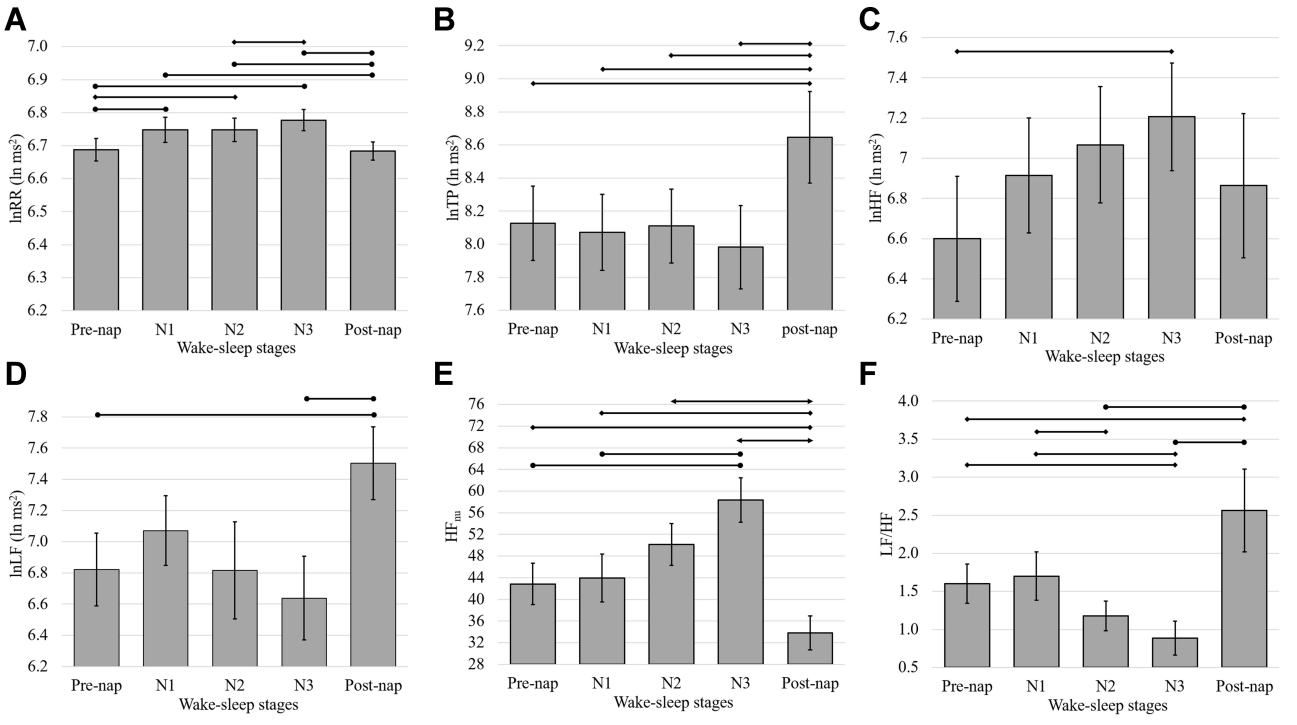

Figure 1 Changes in HRV measures across different wake-sleep stages. (**A**) Changes in RR across different wake-sleep stages. (**B**) Changes in TP across different wake-sleep stages. (**C**) Changes in HF across different wake-sleep satges. (**D**) Changes in LF across different wake-sleep stages. (E) Changes in HF across different sleep-wake stages. (**F**) Changes in LF/HF ratio across different wake-sleep stages. Data expressed as mean ± SEM. (◆——◆) p <0.05; (●——●) P <0.01; (◀——▶) P <0.001.

Abbreviations: ln, natural logarithm; RR, R-R interval; HF, high frequency; TP, total power; LF, low frequency; nu, normalized unit; Pre-nap, wakefulness before nap period; N1, stage 1 NREM; N2, stage 2 NREM; N3, stage 3 NREM; Post-nap, wakefulness after nap period.

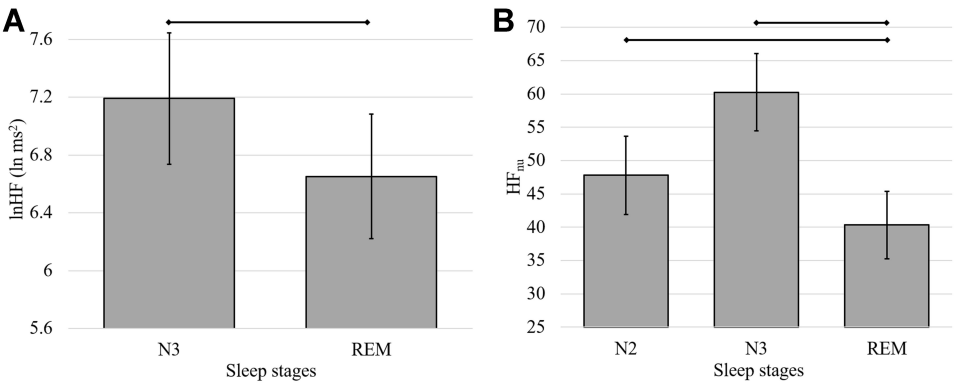

Figure 2 Comparison of HRV measures between the REM and NREM stages. (**A**) Comparison of HF between REM and N3.(**B**) Comparison of HFnu between the REM, N2 and N3. Data expressed mean± SEM; (◆——◆) p <0.05.
Abbreviations: ln, natural logarithm; HF, high frequency; nu, normalized unit; N2, stage 2 non-rapid eye movements sleep; N3, stage 3 non-rapid eye movement sleep; REM, rapid eye movement sleep.

normalized high-frequency power were significantly increased, suggesting a higher parasympathetic control of the heart.[34] In addition, Cellini et al reported the dominance of parasympathetic activity during NREM sleep, mainly by lengthening of RR-interval and increased HF power and HF_{nu} compared with that observed during pre- and post-nap wakefulness.[35] The lowest LF level reported in the present study was during N3, which is consistent with the results of previous studies showing a decrease in the LF level after sleep onset and steady decrease during NREM sleep.[1,2,14] Interestingly, we noticed an abrupt parallel increase of TP and LF during post-nap wakefulness compared with that during pre-nap wakefulness and N3, which may indicate relative sympathetic dominance and may indicate an association between TP and LF; however, the exact mechanism behind this association remains debatable.[8,10,36] In addition, we observed that increased LF was accompanied by shortening of the RR-interval; this further supports the association between LF and sympathetic activity.

The progressive reduction of the LF/HF ratio during sleep stages N2 and N3 compared to that during pre-nap observed in the present study and then an increase during post-nap also support the notion of a shift in the sympathovagal balance during the sleep stages, with a restoration of sympathetic dominance during the post-nap stage. Our data is consistent with those reported in earlier studies that also demonstrate reduced LF/HF ratio during N3 compared with that during pre-nap wakefulness.[11]

Notably, the HF band is widely accepted to reflect cardiac parasympathetic nerve activity.[6–8] In contrast, the LF band was assumed to reflect a dominant sympathetic effect[6,9] but this is debatable.[10] Thus, the LF/HF ratio was proposed as an index describing the balance between the two branches of the ANS (sympathetic and

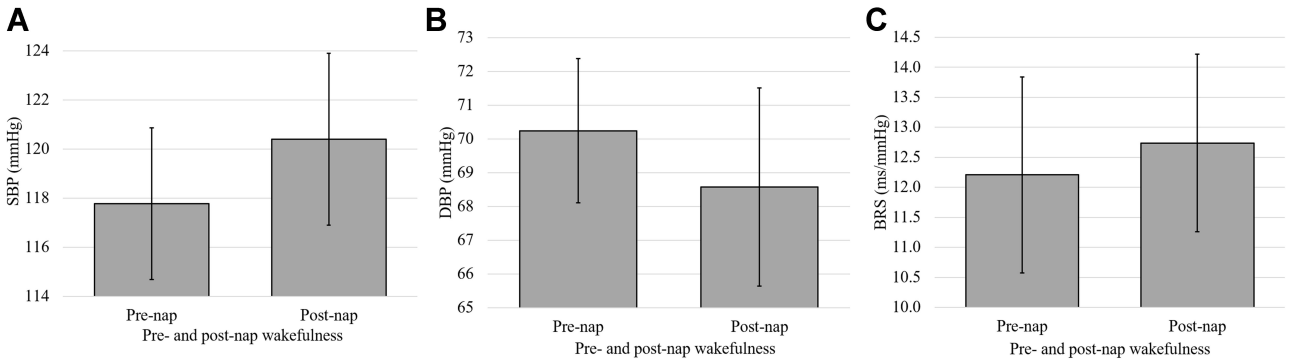

Figure 3 Comparison of BP and BRS measures during pre- and post-nap wakefulness. (**A**) Comarison of SBP during pre-and post-nap wakefulness. (**B**) Comparison of DBP during pre-and post-nap wakefulness. (**C**) Comparison of BRS during pre-and post-nap wakefulness. Data expressed as mean ± SEM.
Abbreviations: BP, blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; BRS, baroreflex sensitivity; Pre-nap, wakefulness period before nap period; Post-nap, wakefulness period after nap period.

parasympathetic), in which an increased LF/HF ratio reflects sympathetic dominance, whereas reduction in this ratio indicates parasympathetic dominance.[37] However, despite wide usage of LF/HF ratio as an index to assess the sympathovagal balance, this index should be interpreted with strict caution because many limitations have been described. The LF component of HRV has been found to not reflect the sympathetic effect alone but rather reflect a complex interaction of sympathetic, parasympathetic, and other less known factors. As a consequence, the LF/HF ratio should be interpreted with caution, taking into consideration all these limitations and contributing factors.[10]

The pattern of parasympathetic dominance in autonomic activity during daytime nap reported herein was similar to that observed during night sleep. Accordingly, some studies conducted during nighttime sleep showed enhanced vagal cardiac activity during NREM sleep with a parallel increase in the RR-interval and HF and decrease in HR.[1,16] Furthermore, another study reported an overall reduction in cardiovascular output accompanied by vagal cardiac activity dominance during NREM sleep.[14] This pattern of vagal activity seems to be a "cardiovascular holiday" in which relative "quiescence" of the cardiovascular system occurs during daytime nap, which suggest possible long-term benefits of daytime nap on cardiovascular health given that it appears to provide autonomic stability during NREM sleep.[2] Although the impact of daytime naps on cardiovascular system remains controversial, our results support the idea that the vagal pattern of NREM sleep exerts a protective effect on the cardiovascular system.[1,34,35] However, other studies have shown that napping was associated with worsening prognosis and an increase in all-cause mortality in patients with cardiovascular disease,[38] thereby warranting further investigations in this area of research. Furthermore, the markedly increased LF and TP during post-nap wakefulness observed herein needs further investigations as they could imply that napping leads to a post-nap increase in sympathetic activity, which could be potentially harmful, or that napping provides an advantage by preparing the body for a much-required sympathetic comeback following peaceful rest.

Furthermore, the correlation between autonomic dysfunctions during sleep with cognitive impairment and cerebrovascular events has been also investigated. A recent study by Buratti et al suggests that autonomic dysfunction during sleep is associated with a high risk of developing lacunar stroke, and abnormal HRV might be implicated in the recurrence of cerebrovascular events in these patients.[39] In addition, short daytime naps are effective in improving vigilance and cognition in healthy individuals as well as those with sleep disorders.[40] Another study reported that short napping periods of <60 min had a significant protective effect against the development of Alzheimer's disease, whereas longer napping is associated with an increased risk of this disease.[41] Daytime naps of approximately 60–90 min have been found to improve memory consolidation of new events that occurred prior to the nap.[40]

BRS measurements revealed no significant changes in BRS, SBP, and DBP before and after the nap. To the best of our knowledge, there is no literature comparing BRS before and after naps, and some studies reported only changes in BRS during sleep. Conway et al reported a significant increase in BRS during sleep stages,[26] whereas Nakazato et al reported inconclusive results.[27]

One limitation of the present study was that only men had been recruited. As such, our results may not be generalized to women considering sex-related hormonal fluctuations that could impact nocturnal sleep and daytime nap.[42] Another limitation was that BRS data during sleep stages could not be obtained because of electrical interference between the Finometer® and polysomnography devices, thereby limiting BRS data analyses to wakefulness stages.

Conclusion

Our results suggest that NREM sleep stages during daytime napping were predominated by parasympathetic nervous activity with a reduction in sympathetic activity, whereas REM sleep promoted a reduction in parasympathetic activity and/or an enhancement in sympathetic activity. Our findings support the notion that a daytime napping is beneficial for cardiovascular health in healthy young adults.

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Disclosure

The authors report no conflicts of interest for this work.

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