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HEART RATE VARIABILITY DURING DAYTIME NAPS IN HEALTHY ADULTS: AUTONOMIC PROFILE AND SHORT-TERM RELIABILITY

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Abstract

In healthy individuals, a reduction in cardiovascular output and a shift to parasympathetic/vagal dominant activity is observed across nocturnal sleep. This cardiac autonomic profile, often measured by heart rate variability (HRV), has been associated with significant benefits for the cardiovascular system. However, little is known about the autonomic profile during daytime sleep. Here we investigated the autonomic profile and the short-term reliability of HRV during daytime naps in 66 healthy young adults. Participants took an 80–120 min polysomnographically-recorded nap at 1:30 PM. Beat-by-beat RR interval values (RR), high (HF) and low frequency (LF) power, total power (TP), HF normalized units and the LF/HF ratio were obtained for 5 min during pre-sleep wakefulness and during nap sleep stages (N2, N3, REM). A subsample of 37 participants took 2 additional naps with two weeks between recordings. We observed lengthening of the RR, higher HF and HF_{nu} and lower LF/HF during NREM, compared with REM and wake, and a marked reduction of LF and TP during N3. Short-term stability of RR and HF ranged across sleep stages between 0.52–0.76 and 0.52–0.80 respectively. Our results suggest that daytime napping in healthy young adults is associated with dynamic changes in the autonomic profile, similar to those seen during nocturnal sleep. Moreover, a reliable intra-individual measure of autonomic cardiac activity can be obtained by just a single daytime nap depending on specific parameters and recording purposes. Nap methodology may be a new and promising tool to explore sleep-dependent, autonomic fluctuations in healthy and at-risk populations.

Keywords

autonomic activity; daytime nap; HRV; intraclass correlations; reliability; REM sleep

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Author contributions: N. Cellini, L. N. Whitehurst and S. C. Mednick developed the study concept and the study design. Testing and data collection were performed by L. N. Whitehurst and E. A. McDevitt. N. Cellini performed the data analysis. All authors contributed to the data and interpretation, drafted the manuscript, provided critical revisions, and approved the final version for submission.

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Introduction

Sleep and the autonomic nervous system (ANS) influence each other in a bidirectional fashion. Changes in the ANS modulate sleep onset as well as the transition between the different stages, and each sleep stage is associated with a distinct autonomic profile (for a review see Trinder, Waloszek, Woods, & Jordan, 2012). A well-established method to non-invasively assess cardiac autonomic activity is heart rate variability (HRV) analysis. The spectral analysis of inter-beat (RR) intervals (i.e., the intervals between individual heart beats) yields information about two main oscillatory components: the Low Frequency (LF: 0.04–0.15 Hz) and the High Frequency (HF: 0.15–0.4 Hz) component (for a comprehensive review about HRV see Berntson et al., 1997). These frequency ranges reflect specific changes in cardiac autonomic regulation. There is wide consensus regarding the significance of the HF component, which reflects the cardiac parasympathetic modulation of respiration. However, the meaning of the LF component is still debated (see Berntson, et al., 1997; Billman, 2013; Montano et al., 2009; Reyes del Paso, Langewitz, Mulder, van Roon, & Duschek, 2013).

Given its minimal intrusiveness, HRV analysis has been widely used to assess cardiac autonomic modulation during sleep (Tobaldini et al., 2013; Trinder, et al., 2012). Young, healthy adults show a specific cardiac pattern at sleep onset, including reduced heart rate (a lengthening of the RR interval) coupled with increased HF activity (Burgess, Trinder, & Kim, 1999; de Zambotti, Covassin, De Min Tona, Sarlo, & Stegagno, 2011; Trinder et al., 2001). These changes suggest a shift of the ANS from sympathetic to parasympathetic regulation in the transition from wakefulness to sleep. Throughout the progression of non-rapid eye movement (NREM) sleep, which is comprised of stages 1, 2 and slow wave sleep (N1-N3), RR intervals continue to lengthen (Bušek, Vaňková, Opavský, Salinger, & Nevšímalová, 2005; de Zambotti et al., 2014; Trinder, et al., 2001), and HF activity remains elevated, with higher vagal modulation during NREM sleep than rapid eye movement (REM) sleep (Bušek, et al., 2005; de Zambotti et al., 2013; Trinder, et al., 2001). These findings indicate an overall reduction in cardiovascular output and dominance of parasympathetic/vagal activity during NREM sleep (Busek et al., 2005), a pattern that has been associated with significant benefits for the cardiovascular system (Trinder, et al., 2012). These fluctuations in the cardiac autonomic profile across sleep and wake are likely explained by a combination of sleep and circadian influences on heart rate (Boudreau, Yeh, Dumont, & Boivin, 2013; Burgess, Trinder, Kim, & Luke, 1997; Trinder, et al., 2001) and may in part be responsible for the homeostatic regulatory balance between sympathetic and parasympathetic activity. This suggested balance has been correlated with reduced risk for cardiovascular disease, diabetes, and all-cause mortality (Thayer, Yamamoto, & Brosschot, 2010), leading some researchers to describe normal sleep as a “cardiovascular holiday” (Trinder, et al., 2012).

While autonomic activity during nocturnal sleep has received attention (for a review, see Tobaldini, et al., 2013), the relationship between daytime sleep (nap) and cardiac autonomic output in healthy populations has not been adequately investigated. This lack of research is surprising considering that, according to the National Sleep Foundation (2014), 53% of adults in America nap at least once in a 7-day period for at least 15 minutes. In other

healthy, adult samples, 60–75% of adults nap at least one time in a 7-day week with average nap durations of about 70 minutes (Dinges, 1992; Pilcher, Michalowski, & Carrigan, 2001). Moreover, the relationship between napping and cardiovascular risk is quite controversial, with epidemiological studies reporting that frequent napping is associated with both increased (Jung, Song, Ancoli-Israel, & Barrett-Connor, 2013; Leng et al., 2014) and decreased (Campos & Siles, 2000) risk for coronary heart disease.

To our knowledge, a study by Boudreau and colleagues (2013) is the only published work investigating the HRV profile during naps in healthy, young adults. The authors examined the influence of circadian rhythm on HRV in a forced 72-hour ultradian sleep/wake protocol that consisted of a 1-hr nap opportunity every other hour for 3 days. They found that heart rate and the HF component have less pronounced sleep-related changes during the day compared to the night. While this paradigm provides information about circadian-related shifts in HRV, the ultradian sleep/wake protocol used by the authors resulted in fragmented sleep periods, limited duration of N3 and REM, and partial sleep deprivation, making it difficult to generalize these results to ecologically-valid nap conditions.

In addition to the lack of studies investigating the HRV pattern across daytime sleep, little is known about the short-term stability of the cardiac autonomic parameters during sleep. In other words, it is still unclear how much autonomic information obtained in a single sleep recording is reliable. Recently, Israel and colleagues (2012) showed high short-term temporal stability (i.e., high intra-class correlation coefficients (ICC)) for HF (computed as normalized units, HF_{nu}) and LF/HF ratio during both nocturnal NREM and REM sleep periods across three consecutive nights. These results suggest that one night of sleep may be sufficient to obtain reliable measures of HRV, which may have practical implications for both research and clinical purpose, i.e., reducing the cost and the burden of several physiological recordings. However, to our knowledge, no other study has investigated the reliability of cardiac autonomic parameters during sleep, and no study has assessed this reliability during daytime naps.

In this context, the aim of the current study is two-fold. Our first goal was to determine the HRV profile across sleep stages during an 80–120 min daytime nap in healthy, young adults. We chose a nap period of this duration and strategically positioned in the mid-afternoon (1:30 PM) to promote a nap with adequate proportions of N2, N3, and REM sleep (see Achermann & Borbély, 2003; Mednick, Nakayama, & Stickgold, 2003; Mednick et al., 2013). We hypothesized that HRV during daytime sleep would resemble the autonomic profile observed during nighttime sleep. Our second goal was to investigate the short-term reliability of HRV parameters during daytime sleep. Due to the different autonomic fluctuations observed in NREM and REM sleep (Amici et al., 2014), we hypothesized that HRV parameters will exhibit higher stability during NREM stages and more instability during REM sleep.

Method

Participants

The investigation took place as part of a larger study about sleep and memory in the Sleep and Cognition Lab at the University of California, Riverside. Sixty-six healthy, non-smoking participants (*M*age = 21.27 ± 2.8 years, 30 Females) with no personal history of neurological, psychological, or other chronic illness provided informed consent, which was approved by the University of California, Riverside Human Research Review Board. Participants included in the study were college students, had a regular sleep-wake schedule (reporting a habitual time in bed of about 7–9 hrs per night), and no presence or history of sleep, psychiatric, cardiovascular or neurological disorders determined during an in-person or phone interview). Participants received monetary compensation and/or course credit for participating in the study.

Procedure

Participants were instructed to spend at least 7 hours of time in bed each night during the week prior to the study. Compliance was monitored with sleep diaries (for the whole week before the experimental session) and actigraphy (for the night before the experimental session). Additionally, participants were instructed to abstain from caffeine and alcohol starting at noon the day prior to the study. On the study day, participants reported to the Sleep and Cognition Lab at at 9 am. At 12:30 pm, electrodes were attached for polysomnography (PSG) recording of sleep. Each participant took a PSG-recorded nap between 1:30 pm and 3:30 pm with five minutes of pre-nap, waking, supine electrocardiogram (ECG) recording. Participants were allotted about 2 hours of time in bed and a trained sleep technician monitored all naps in real-time. Nap sleep variables summarized are reported in Table 1. Thirty-seven participants (*M*age = 21.65 ± 3.04 years, 23 Females) took 2 additional PSG-recorded naps with 2 weeks between recordings, for a total of 3 PSG-recorded naps per participant in this subsample.

Sleep Recordings

PSG recordings were collected using Astro-Med Grass Heritage Model 15 amplifiers with Grass Gamma software. PSG included eight electroencephalogram leads (EEG: F3-A2, F4-A1, C3-A2, C4-A1, P3-A2, P4-A1, O1-A2, O2-A1), bilateral electrooculography (EOG: ROC-A1, LOC-A2), and submental bipolar electromyography (EMG). Each signal was amplified, band-pass filtered (EEG and EOG: 0.3–35 Hz; EMG: 10–100 Hz), and digitized at 256 Hz. Raw data were visually scored in 30-sec epochs following the American Academy of Sleep Medicine (AASM) rules for sleep staging (Iber, Ancoli-Israel, Chesson, & Quan, 2007). In order to reduce the possible level of variability, scoring was performed by a single well-trained sleep technician. However, to ensure reliability of sleep scoring, another independent sleep technician also scored 10% of the sleep records and an intraclass correlation of .86 was reached.

Assessment of cardiac autonomic activity

Electrocardiogram (ECG) data were collected at a 256 Hz sampling rate using a modified Lead II Einthoven configuration. R-wave peaks were detected automatically by Kubios HRV Analysis Software 2.2 (Matlab, Kuopio, Finland), visually examined, and edited for artifacts. The same software was employed to perform the HRV analysis of the R-waves series through the whole sleep time period according to the Task Force of the European Society of Cardiology and North American Society of Pacing and Electrophysiology guidelines (Camm et al., 1996). Missing and ectopic beats were corrected via cubic spline interpolation. Inter-beat intervals were computed and a third order polynomial filter was applied on the time series in order to remove trend components. A Fast Fourier transformation was employed to quantify the absolute spectral power (ms^2) in the LF and HF frequency bands and the total power (TP), which reflects total HRV. From these variables we derived the HF normalized units (HF_{nu}= (HF [ms^2]/ HF [ms^2] + LF [ms^2])*100) and the LF/HF ratio, an index often considered to reflect the sympatho-vagal balance (i.e., the balance between the two branches of the ANS), but whose meaning has been recently put into question (Billman, 2013; Reyes del Paso, et al., 2013). Since the LF normalized units are mathematically reciprocal to HF_{nu} (i.e. $LF_{nu} = 1 - HF_{nu}$), we computed only the HF_{nu} index to avoid redundancy. Since HF, LF and TP measures showed skewed distributions, these variables were transformed via natural logarithm (ln). In addition, we included the RR interval in the analysis as a major index of cardiac autonomic control (Pinna et al., 2007). To control for the potential confounding role of respiration in the outcome of HRV measures without using more intrusive measures, we used the ECG-derived respiration (EDR) provided by Kubios 2.2, an index considered a reliable estimate of the respiratory frequency (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014).

Data Reduction and Statistical Analyses

For the analysis of RR and frequency-domain HRV measures during different sleep stages, consecutive artifact-free 5-min windows of undisturbed sleep were selected across the whole nap using the following rules: 1) the 2-mins preceding and 2) the 5-min epoch selected must be free from stage transitions and arousal. Windows were identified and averaged within for N2, N3 and REM sleep. We also analyzed 5-min of pre-nap wakefulness (Wake). Epochs of N1 and wake after sleep onset (WASO) were not analyzed (Trinder, et al., 2001).

Our main interest in this study was to examine differences in HRV across sleep stages. Therefore, we employed a Repeated Measures ANOVA on each HRV variables (TP_{ln}, HF_{ln}, LF_{ln}, LF/HF ratio, HF_{nu}, RR and EDR), with Wake, N2, N3, and REM, as within-subjects factors. The Huynh-Feldt correction was applied when sphericity assumptions were violated. Tukey's HSD test was used for post-hoc comparisons and partial eta squared (η^2_p) is reported for effect size. Data reported in the ANOVA only reflects subjects who entered all three sleep stages (N2, N3 and REM) during the nap ($N = 33$). Statistical significance was set at $\alpha = 0.05$.

For the subsample of 37 participants with 3 naps recorded, we measured intra-individual stability of the HRV parameters across different naps. A repeated measures ANOVA was

employed for each HRV parameter in each sleep stage (W, N2, N3 and REM sleep) with the three naps as within-subjects factors. The Huynh-Feldt correction was applied where appropriate. Also, from the square root of the mean square error term of the repeated-measures ANOVA we estimated the standard error of measurement (SEM), considered an index of absolute reliability (Atkinson & Nevill, 1998; Stratford & Goldsmith, 1997). Intra-class correlation coefficients (ICCs), an index of relative reliability (Atkinson & Nevill, 1998), were calculated in order to examine the within-participant short-term stability of the cardiac autonomic parameters in each sleep stage across the three recordings. ICCs were computed using a two-way random method with single measure and absolute agreement (ICC(2,1)). The 95% confidence intervals for ICCs were also calculated. Although the significance of the ICC values is still debated (see Atkinson & Nevill, 1998), a prior study considered ICCs ≥ 0.60 as values of reliable short-term stability of HRV parameters during sleep (Israel, et al., 2012). Here we considered ICCs of 0–0.2 as slight agreement, 0.2–0.4 as fair agreement, 0.4–0.6 as moderate agreement, 0.6–0.8 as substantial agreement, and 0.8–1.0 as almost perfect agreement, according to the Landis and Koch scale (Landis & Koch, 1977). Note that for the reliability analyses the number of participants analyzed for each stage changed depending on whether data were present for all the three naps: W: N =36; N2: N =37, N3: N =21; REM: N =20.

Results

Demographic and prior sleep data for the whole sample are shown in Table 1.

The sleep architecture of the experimental nap is reported in Table 2.

HRV profile across a daytime nap

ANOVAs revealed a significant effect of Stage on HF_{ln} ($F_{(3,96)}$ = 4.69, p =.008, ε = .79, η^2_p=.13), LF_{ln} ($F_{(3,96)}$ = 7.23, p < .001, ε = .84, η^2_p =.18), TP_{ln} ($F_{(3,96)}$ = 14.39, p < .001, ε = .92, η^2_p = .31), HF_{nu} ($F_{(3,96)}$ = 24.28, p < .001, ε = .88, η^2_p = .43), LF/HF ratio ($F_{(3,96)}$ = 9.86, p < .001, ε = .77, η^2_p = .24), and RR intervals ($F_{(3,96)}$ = 15.13, p < .001, ε = .66, η^2_p = .32). Post-hoc comparisons revealed a lengthening of RR intervals during each sleep stage compare to wake (Wake v N2 p < .001; Wake v N3 p < .001; Wake v REM p = .038), and between NREM stages and REM sleep (N2 v REM p = .015; N3 v REM p = .021; Figure 1a). TP_{ln} was reduced during N3 compared to pre-sleep wakefulness (p = .002), N2 (p < .001) and REM (p < .001) (Figure 1b). REM also showed a higher TP_{ln} compared to wakefulness (p = .039) (Figure 1b). HF_{ln} power was highest during N2 and significantly greater than wake (p = .003) and N3 (p = .038) (Figure 1c), however, due to significant fluctuations in TP_{ln}, the normalized unit (HF_{nu}) was used to interpret patterns across sleep stages. We found that HF_{nu} was significantly increased during N3 compared to Wake (p < .001), N2 (p = .001), and REM (p < .001) (Figure 1e), and during N2 compared to REM (p < .001). LF power showed a significant reduction in N3 compared to N2 (p = .003) and REM (p < .001) (Figure 1d). LF/HF ratio also showed a significant decrease in N3 compared to pre-sleep wakefulness (p < .001), N2 (p = .003) and REM (p < .001) (Figure 1f). Lastly, EDR did not differ across the stages ($F_{(3,96)}$ = 1.42, p =.242, ε = .56, η^2_p=.04), indicating

that HRV changes in the different sleep stages were not merely due to change in the respiratory rate.

It is worth noting that the Repeated Measures ANOVA with sleep stages as within-subjects factor limited the analysis to the participants who entered all three sleep stages (N = 33). Therefore, in order to maximize the number of comparisons, we also ran a series of exploratory t-tests comparing, for all HRV parameters, each sleep stage. The results seems to be consistent with the ANOVAs output (see Supplemental Table 1). In addition, the same autonomic pattern across sleep stages was observed in all three naps recorded in the subsample (see Supplemental Table 2).

Overall, these results show that HRV profiles during a daytime nap follow a similar pattern as nighttime sleep (Bušek, et al., 2005; de Zambotti, et al., 2014; Trinder, et al., 2001).

Reliability of HRV parameters

We then analyzed the inter-individual reliability of HRV parameters across three daytime naps. As reported in Table 3, sleep parameters were similar across the three naps.

At a descriptive level, the three HRV recordings showed a similar pattern, including increased cardiac vagal activity (i.e., lengthening of RR intervals, increased HF_{ln} and HF_{nu} activity) during sleep compared to pre-sleep wakefulness and a marked reduction of LF_{ln}, with a consequent drop of TP during N3 (Table 4). Repeated measures ANOVAs confirmed the absence of any significant differences across the three recordings for each HRV index (Table 4).

The ICCs and the SEM showed different levels of short-term stability among the different parameters and across sleep stages (Table 4). RR intervals and HF_{ln} showed good reliability across different recordings in each stage (20–34% of variance was due to random error) with the exception of REM, which showed moderate ICC values and higher SEMs. TP_{ln} showed a moderate reliability for all the stages (but not for REM), whereas LF showed fair ICC values (with a 71% of random error in REM) and higher SEMs for each stage except N2. High random error of LF explains the low short-term reliability of the LF/HF ratio, including elevated SEM and low ICC in each sleep stage except N3, where LF_{ln} power drops and its contribution to the ratio is reduced. Lastly, HF_{nu} showed a moderate reliability for all sleep stages, whereas EDR showed very low stability in each sleep stage.

Discussion

In the current study, we assessed sleep-stage-specific changes in HRV during an afternoon nap in healthy, young adults. Our results indicate that dynamic changes occur in the HRV profile during a daytime nap. Specifically, we observed a distinct increase in cardiac vagal activity across all sleep stages relative to pre-sleep wakefulness, as indexed by higher HF_{ln} and heart rate deceleration (i.e., a lengthening of the RR interval). We also witnessed higher HF_{ln} and HF_{nu} and lower LF/HF during NREM sleep, and the reverse pattern during REM, as a consequence of the increased LF_{ln} component in this paradoxical stage. We also observed a strong decrease in TP_{ln} during N3 mainly due to the significant decrease in LF_{ln}

power during this sleep stage (note that HF_{ln} was not significantly reduced in N3). These data suggest discrete fluctuations in LF_{ln} power and an overall dominance of vagal activity during N3 sleep in a daytime nap. Indeed, this autonomic shift was also reflected in the LF/HF ratio, which was also at is lowest during N3 sleep. Note that since the mechanism underlying LF_{ln} is still debated (Billman, 2013; but also Montano, et al., 2009; Reyes del Paso, et al., 2013), strong inferences about the physiological meaning of changes in LF, LF/HF ratio and HF_{nu} are difficult to make for these parameters, but we report them for descriptive purposes. Notwithstanding, our descriptive data are consistent with previous literature showing that LF power drops after sleep onset, steadily decreases across NREM sleep, and rises again during REM sleep (Bušek, et al., 2005; Trinder, et al., 2001). Likewise, the LF/HF ratio is reduced during N2 and N3 compared to wakefulness, and markedly increased during REM sleep (Burgess, Penev, Schneider, & Van Cauter, 2004), with a similar or higher ratio during REM relative to wakefulness (Bušek, et al., 2005; de Zambotti, et al., 2014; de Zambotti, et al., 2013).

Taken together, these findings are consistent with previous reports on the cardiac autonomic profile during nocturnal sleep in younger and older adults (Ako et al., 2003; Bonnet & Arand, 1998; Bušek, et al., 2005; de Zambotti, et al., 2014; de Zambotti et al., 2012; de Zambotti, et al., 2013; Elsenbruch, Harnish, & Orr, 1999) as well as short episodes of daytime sleep (Boudreau, et al., 2013). Collectively, these studies demonstrate an overall reduction in cardiovascular output and dominance of parasympathetic/vagal activity during NREM sleep (Busek et al., 2005), which has been associated with significant benefits for the cardiovascular system (Trinder, et al., 2012).

Short-term reliability of HRV parameters

We also assessed the absolute (i.e., SEM) and relative (i.e., ICCs) short-term reliability of HRV-derived cardiac autonomic parameters during three daytime naps in a subsample of 37 participants. Overall, during daytime naps, HRV parameters showed good to moderate short-term reliability, with some variability across sleep stages and autonomic parameters. RR intervals and HF_{ln} resulted in good reliable short-term stability, whereas we observed moderate stability for TP_{ln} and HF_{nu} and fair reliability for $LF_{ln,}$ and LF/HF, with some specific exceptions depending on sleep stages. Indeed, N3 was the most stable stage for HRV parameters (ICC ranging from 0.29 for LF_{ln} to 0.76 for RR and reduced SEMs). This higher stability may be due to the general metabolic down-regulation and the relative cardiorespiratory stability that occurs in this sleep stage (Brandenberger, Buchheit, Ehrhart, Simon, & Piquard, 2005; Somers, Dyken, Mark, & Abboud, 1993). Moreover, it has been shown that HRV parameters and delta EEG activity (0.5–4 Hz), the dominant EEG band during N3 sleep, are temporally associated. Specifically, during nocturnal sleep, changes in cardiac vagal activity precede changes in delta activity by a few minutes (Brandenberger, Ehrhart, Piquard, & Simon, 2001; Dumont et al., 2004; Jurysta et al., 2003). It has been suggested that this temporal relationship may result from interaction between the brain centers involved in both autonomic and sleep control (e.g., brain stem, hypothalamus; Dumont, et al., 2004). Also, since this relationship is stronger during the first NREM period, it has been proposed that it may also be connected to homeostatic sleep regulation factors (Rothenberger et al., 2015).

On the other hand, REM sleep showed the highest variability between parameters mainly due to LF_{ln} fluctuations, which likely compromised TP_{ln} and LF/HF reliability. The high degree of REM variability may be due to the consequence of both the profound autonomic and respiratory intra-stage instability (Guilleminault, Pool, Motta, & Gillis, 1984; Richard et al., 2007; Somers, et al., 1993). Although the typical phasic changes in sympathetic and parasympathetic discharge characterizing REM sleep have been associated with transient events such as rapid eye movements and myoclonic twitches, the reason for these autonomic instabilities are still unknown (Amici, et al., 2014).

Compared to the only other study investigating short-term stability of HRV parameters during nocturnal sleep (Israel, et al., 2012), we observed lower ICC values for the variables in common, i.e. HF_{nu} and LF/HF. This could be a consequence of the lower number of sleep bins analyzed in our data when compared to Israel and colleagues (2012), a limitation of the current study, which may have resulted in increased variability in our output. Another factor may be the overall lighter and more disrupted sleep (e.g. higher N1 proportion, lower sleep efficiency) that is typical of daytime naps (Cellini, Buman, McDevitt, Ricker, & Mednick, 2013; Kanady, Drummond, & Mednick, 2011). In addition, we analyzed 3 naps with 2 weeks between recordings, whereas Israel and colleague analyzed three consecutive nights. Lastly, Israel and collaborators (2012) analyzed all NREM stages together, but here, we treated N2 and N3 as distinct stages. This is an important contrast between this work and Israel and colleagues considering N2 and N3 have distinct neural and physiological profiles, which were highlighted in the current study by the prescribed autonomic patterns parsed by sleep stage.

Daytime naps and health outcomes

Indeed, there is on-going discourse surrounding the benefits and drawbacks of napping for health outcomes, with the literature split between napping as beneficial or a sign of health decline, (Dhand & Sohal, 2006). Specifically, some research has shown that napping is correlated with increased nighttime sleep disruption (Owens et al., 2010), excessive daytime sleepiness and depression (Foley et al., 2007), as well as all-cause (Jung, et al., 2013) and cardiovascular mortality (Leng, et al., 2014). Other studies have associated napping with normal nighttime sleep, distinct cognitive and health benefits like increased cognitive alertness, decreased daytime sleepiness, and decreased risk for myocardial infarction, and coronary death (Dhand & Sohal, 2006; Gillberg, Kecklund, Axelsson, & Akerstedt, 1996; Jung, et al., 2013; Leng, et al., 2014; Naska, Oikonomou, Trichopoulou, Psaltopoulou, & Trichopoulos, 2007). In this framework, our data showed that daytime sleep is associated with lengthened of RR intervals and increase vagal activity indicating that during naps there is a “quiescence” of the cardiovascular system (a “cardiovascular holiday”) similar to what is observed during nocturnal sleep (Trinder, et al., 2012). These results, combined with studies showing that midday naps restore proinflammatory cytokines (i.e., interleukin 6) and catecholamine levels altered by a night of sleep loss (Faraut et al., 2015; Vgontzas et al., 2007), suggest a potential beneficial long-term effect of daytime sleep on cardiovascular health. However, the current sample was comprised of young, healthy adults with no history of physical or psychiatric illness. Thus, in order to gain a better understanding of the role of

napping in cardiovascular health, further studies with clinical sample and different age ranges are warranted.

Limitations

The results of the current study should be interpreted in light of some limitations, such as the lack of a direct comparison of individual's daytime and nighttime HRV profile. As indicated by Boudreau and colleagues (2013), who investigated the HRV changes in a forced 72-hour ultradian sleep/wake protocol that consists of a brief nap opportunity (1 hr) every other hour for 3 days, sleep stage-specific effects on the ANS is dependent on specific times of the day. Specifically, Boudreau and colleagues (2013) showed that when compared to daytime sleep, sleep-specific fluctuations in the HRV profile were more noticeable when N2 and N3 occurred in the first part of the night, the time of day when propensity for these stages is greatest (~2:00 AM), while for REM, these changes were more prominent in early morning hours (~5:30 AM), also when the circadian drive for REM is higher. Although the forced ultradian sleep/wake protocol resulted in fragmented sleep periods (an average total sleep time of 27 min per nap episode), limited the duration of N3 and REM and lead to partial sleep deprivation, it also provided critical information about circadian-related shifts in HRV. Thus, combining our results with Boudreau and colleagues (2013) findings, it is possible that the HRV profile during a daytime nap resembles the profile observed during nighttime sleep, but with less pronounced circadian-related changes. Future studies should address this issue by directly investigating the inter-individual's HRV changes during both nocturnal and diurnal sleep.

Another limitation regards the absence of a direct assessment of respiration rate. Indeed changes in respiratory rate can markedly affect HRV (Song & Lehrer, 2003). However, from the ECG trace we computed the electrocardiogram derived respiration (EDR), which is purported to be a reliable estimate of respiration rate (Tarvainen, et al., 2014). The analysis of EDR across sleep stages showed that no significant differences in respiration rate, suggesting that respiration alone cannot account for the change in HRV parameters observed during sleep. Future studies should confirm these results using more precise measurement techniques, such as respiratory bands.

Other limitations are intrinsic of daytime naps. Compared to nocturnal sleep, daytime naps are characterized by an overall lighter and more disrupted sleep (e.g. higher N1 proportion, lower sleep efficiency; Cellini, et al., 2013; Kanady, et al., 2011). This, combined with the intrinsically short sleep period of naps, resulted in a limited number of HRV epochs analyzed, which may have resulted in an overall increase of variability in our data. This also limited the number of participants who reached all sleep stages. However, it should be noted that the same autonomic profile across stages was observed when analyzing the two additional naps of the subsample.

Future direction: HRV during nap as a new tool for assessing cardiovascular health?

The assessment of autonomic function during sleep may be very useful for populations at risk for sleep and cardiovascular disorders (Legramante & Galante, 2005). Several sleep disorders show alterations of the ANS during wake (Cellini, de Zambotti, Covassin, Sarlo, &

Stegagno, 2014; Grimaldi et al., 2010; Hilton et al., 2001) as well as during sleep (Tobaldini, et al., 2013). Moreover, HRV during sleep has been used as an index of sleep fragmentation (Sforza, Pichot, Cervena, Barthelemy, & Roche, 2007), as a screening tool for sleep breathing disorders (Hayano et al., 2011; Heneghan et al., 2008), for assessing the effectiveness of treatments in sleep-apnea patients (Karasulu, Epöztürk, Sökücü, Dalar, & Altin, 2010; Thomas et al., 2007) and to predict mortality in elderly people (see Stein & Pu, 2012). Moreover, it has been suggested that N3 sleep, given the absence of external influences such as movements, environmental noise and voluntary breathing, may be an optimal window to assess "self-controlled" autonomic activity (Brandenberger, et al., 2005). In this context, the current results suggest that daytime nap studies may be an efficient, alternative way to measure cardiac autonomic activity during sleep. Importantly, participants in the current study were not sleep-deprived, which was verified through both sleep diaries and actigraphy monitoring prior to the in-lab nap, and all participants were able to initiate sleep during their nap session strategically positioned in the circadian cycle at 1:30 PM.

It can be argued that a daytime nap can influence nighttime sleep, for instance reducing the power of slow oscillations, which may results in in circadian misalignment and possible detriments to cardio-metabolic health. However, studies showing a negative effect of naps on health used naps that were positioned in the late afternoon/early evening (Werth, Dijk, Achermann, & Borbely, 1996) whereas previous studies using naps positioned in the early afternoon showed no detrimental effect on nocturnal sleep, at least on older adults (Campbell, Murphy, & Stauble, 2005; McDevitt, Alaynick, & Mednick, 2012; Pilcher, et al., 2001). Thus, our findings suggest that daytime sleep recordings may be a cost-effective alternative to full-night assessments in determining an individual's risk for autonomic, cardiovascular and other related diseases as well as for research purposes. Moreover, the possibility of daytime-hospital recording sessions may increase compliance. Future studies should investigate the feasibility of using HRV during nap as a new tool for assessing cardiovascular health.

Conclusion

In conclusion, our results suggest that daytime napping in healthy, young adults is associated with dynamic changes in the autonomic profile, similar to those seen during nocturnal sleep. Moreover, HRV parameters during a daytime nap revealed fair to good short-term stability depending on parameters and sleep stages. These results suggest that a reliable intra-individual measure of autonomic cardiac health can be obtained by just a single daytime nap depending on specific parameters and recording purposes. Thus, nap methodology may be a new and promising tool to explore important sleep-dependent, autonomic fluctuations in healthy and at-risk populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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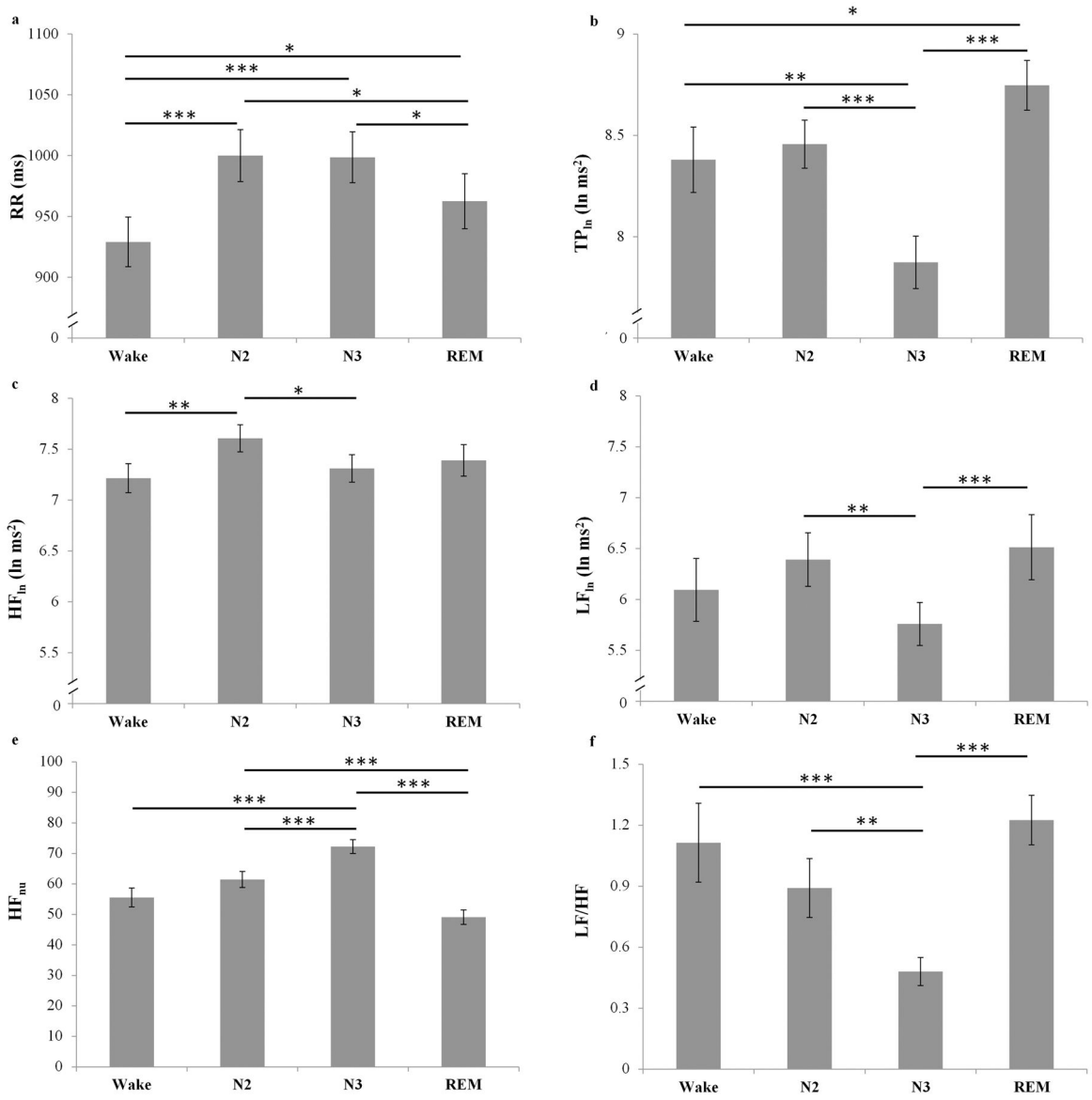

Figure 1.

Table 1

Demographics and prior sleep data for the whole sample.

	Mean (SD)	N° (%)
Sociodemographic		
Age (years)	21.27 (2.8)	
Female/Male		30/36 (45.5/54.5)
Ethnicity		
Asian		5 (7.6)
Hispanic		25 (37.9)
Caucasian		13 (19.7)
African-American		20 (30.3)
Other		3 (4.5)
Education (years)	15 (2.33)	
Health behavior/status		
Caffeine/day	0.77 (0.74)	
Alcohol/week	1.13 (2.76)	
Nap/week	1.50 (1.40)	
BMI (kg/m^2)	22.84 (4.11)	
Night prior the experiment (actigraphy)		
Bedtime	00:15:53 (01:05:57)	
Wake time	07:32:23 (00:40:40)	
TBT (min)	436.86 (52.45)	
TST (min)	373.19 (53.50)	
SOL (min)	13.91 (17.14)	
WASO (min)	55.23 (22.61)	
SE (%)	83.17 (7.68)	
Week prior the experiment (sleep diary)		
Bedtime	00:06:48 (01:14:32)	
Wake time	08:1:14 (01:19:01)	
TBT (min)	491.07 (108.62)	
TST (min)	464.73 (61.28)	
SOL (min)	15.00 (16.53)	
WASO (min)	5.76 (12.53)	
SE (%)	94.52 (16.56)	

Sleep onset latency (SOL), sleep efficiency (SE), wake after sleep onset (WASO), total bed time (TBT), total sleep time (TST).

Table 2

Sleep architecture descriptive.

PSG parameters	Mean (SD)
TST (min)	80.05 (20.73)
N1 (min)	8.14 (5.11)
N2 (min)	35.74 (13.89)
N3 (min)	21.82 (14.58)
REM (min)	12.18 (10.39)
WASO (min)	11.86 (14.84)
SOL (min)	6.30 (5.99)
SE (%)	80.77 (18.30)

Sleep onset latency (SOL), sleep efficiency (SE), wake after sleep onset (WASO), total sleep time (TST), polysomnography (PSG), Non-rem Stage 1, 2, 3 (N1-3), rapid eye movement (REM) sleep.

Table 3

Descriptive results for sleep parameters during the three recordings.

	Nap 1	Nap 2	Nap 3	$F_{2,72}$	*p*
TIB (min)	99.24 (11.91)	99.52 (11.53)	94.66 (7.65)	0.09	0.91
TST (min)	81.34 (21.67)	81.66 (22.91)	84.93 (19.18)	1.32	0.27
N1 (min)	8.14 (5.08)	9.11 (4.19)	10.19 (6.10)	1.25	0.29
N2 (min)	37.61 (13.26)	35.77 (13.42)	37.72 (16.10)	0.50	0.96
N3 (min)	21.92 (14.95)	23.55 (18.26)	23.03 (15.28)	0.20	0.98
REM (min)	13.68 (10.26)	13.23 (9.92)	13.98 (8.70)	0.29	0.75
WASO (min)	12.04 (15.89)	11.91 (17.90)	9.69 (13.66)	0.62	0.54
SOL (min)	5.86 (6.77)	4.45 (3.51)	4.91 (7.01)	0.89	0.42
SE (%)	81.54 (20.97)	83.34 (19.33)	86.42 (16.93)	1.62	0.20

Sleep onset latency (SOL), sleep efficiency (SE), wake after sleep onset (WASO), time in bed (TIB), total sleep time (TST), NREM Stage 1, 2, 3 (N1-3), REM sleep. Values are expressed as means (standard deviation). *F* and *p* values are shown for the repeated-measure ANOVA between the three recordings.

Table 4

HRV parameters during the three recordings.

Parameter	Stage	Nap1	Nap2	Nap3	*F*	*p*	SEM	ICC (95% CI)
RR (ms)	W	931.38 (107.19)	930.41 (123.41)	938.24 (124.89)	0.14	0.87	69.68	0.66 (0.49–0.79)
RR (ms)	N2	999.36 (123.91)	992.76 (127.57)	997.81 (127.94)	0.09	0.91	70.27	0.70 (0.53–0.82)
RR (ms)	N3	981.87 (109.7)	974.56 (110.18)	989.78 (126.35)	0.35	0.70	59.05	0.76 (0.56–0.88)
RR (ms)	REM	947.90 (112.13)	982.46 (143.25)	971.33 (122.00)	0.81	0.45	87.64	0.52 (0.26–0.75)
TP_{ln}	W	8.30 (0.81)	8.41 (0.72)	8.26 (0.85)	0.78	0.46	0.53	0.56 (0.28–0.73)
TP_{ln}	N2	8.32 (0.77)	8.37 (0.79)	8.33 (0.93)	0.06	0.94	0.62	0.46 (0.26–0.65)
TP_{ln}	N3	7.78 (0.83)	7.98 (1.09)	7.94 (0.82)	0.46	0.63	0.65	0.51 (0.23–0.75)
TP_{ln}	REM	8.71 (0.46)	8.81 (0.90)	8.81 (0.83)	0.14	0.86	0.64	0.29 (0.00–0.60)
HF_{ln}	W	7.16 (0.79)	7.16 (0.83)	7.15 (0.89)	0.01	0.99	0.38	0.80 (0.68–0.88)
HF_{ln}	N2	7.41 (0.79)	7.48 (0.87)	7.42 (0.85)	0.18	0.83	0.50	0.61 (0.41–0.78)
HF_{ln}	N3	7.17 (0.85)	7.41 (1.12)	7.34 (0.98)	0.86	0.43	0.61	0.63 (0.39–0.81)
HF_{ln}	REM	7.42 (0.51)	7.38 (0.87)	7.35 (0.87)	0.08	0.92	0.58	0.52 (0.25–0.75)
LF_{ln}	W	7.05 (0.95)	7.19 (0.80)	6.95 (0.95)	0.89	0.42	0.77	0.28 (0.08–0.50)
LF_{ln}	N2	7.09 (0.86)	7.17 (0.84)	7.01 (0.77)	0.69	0.50	0.58	0.52 (0.33–0.69)
LF_{ln}	N3	6.05 (0.86)	6.41 (1.19)	6.37 (0.76)	0.25	0.30	0.81	0.29 (0.03–0.58)
LF_{ln}	REM	7.38 (0.51)	7.57 (0.81)	7.35 (0.72)	0.72	0.49	0.62	0.20 (0.00–0.51)
HF_{nu}	W	52.69 (19.93)	49.85 (16.99)	54.12 (15.55)	0.86	0.43	14.08	0.36 (0.16–0.57)
HF_{nu}	N2	56.61 (17.60)	57.63 (13.81)	57.53 (15.84)	0.11	0.89	10.22	0.59 (0.41–0.74)
HF_{nu}	N3	73.42 (14.01)	71.19 (14.33)	68.31 (15.68)	1.48	0.24	9.64	0.56 (0.31–0.77)
HF_{nu}	REM	51.12 (12.59)	45.13 (16.43)	49.94 (15.37)	1.79	0.18	10.57	0.49 (0.22–0.73)
LF/HF	W	1.46 (1.86)	1.26 (0.96)	1.02 (0.67)	1.47	0.24	1.10	0.25 (0.05–0.47)
LF/HF	N2	1.14 (1.14)	0.88 (0.46)	0.90 (0.56)	1.98	0.14	0.62	0.37 (0.15–0.55)
LF/HF	N3	0.48 (0.48)	0.49 (0.36)	0.59 (0.53)	0.78	0.47	0.33	0.46 (0.19–0.70)
LF/HF	REM	1.17 (1.17)	1.65 (0.96)	1.26 (0.84)	2.65	0.08	0.69	0.32 (0.06–0.61)
EDR (Hz)	W	0.23 (0.03)	0.22 (0.05)	0.22 (0.05)	0.57	0.57	0.04	0.07 (0.00–0.29)
EDR (Hz)	N2	0.23 (0.03)	0.23 (0.03)	0.22 (0.03)	1.57	0.22	0.02	0.25 (0.05–0.47)
EDR (Hz)	N3	0.24 (0.03)	0.23 (0.03)	0.23 (0.05)	0.09	0.92	0.04	0.20 (0.00–0.50)

Parameter	Stage	Nap1	Nap2	Nap3	*F*	*p*	SEM	ICC (95% CI)
EDR (Hz)	REM	0.24 (0.03)	0.23 (0.03)	0.22 (0.03)	2.07	.13	0.03	0.26 (0.01–0.55)

Descriptive values are reported as the means (standard deviation) of the 37 participants who underwent three naps. *F* and *p* values for the repeated measure ANOVAs between the three recordings. Note that number of participants analyzed for each stage changes depending on whether data are present for all the three naps. W: *n*=36; N2: *n*=37, N3: *n*=21; REM: *n*=20. ECG-derived respiration (EDR); High Frequency (HF); Low Frequency (LF), Natural Logarithm (ln); Normalized Units (nu); Total Power (TP).