quantified as RMSSD and pNN50. Two separate stable sleep periods (range, 5-10min) absent of arousals were recorded, along with two separate disrupted periods of sleep with at least one arousal were selected in stage II sleep (N2), slow wave sleep (SWS), and rapid eye movement (REM) sleep. LF and HF HRV was log10 transformed due to non-normal distribution. Statistical analysis included intraclass correlations (ICC) of HRV across the four stable and disrupted periods of sleep, with separate ICC analyses across sleep stages ($\alpha = 0.05$).

Results: Time-domain measures (RRI, RMSSD, pNN50) were reliable across arousal-free and arousal-containing sleep cycles, for all three stages (ICC>0.9, p<0.05). HF HRV exhibited similar reliability patterns across N2 sleep (ICC=0.960, p<0.001), SWS (ICC=0.955, p<0.001), and REM sleep (ICC=0.924, p<0.001). LF HRV was reliable in two stages of stable and disrupted sleep in N2 (ICC=0.903, p<0.001), REM (ICC=0.907, p<0.001) sleep, and trending in SWS (ICC=0.616, p=0.089) sleep.

Conclusion: Time- and frequency-domain HRV were reliable between stable sleep with and without cortical arousals, with the exception of LF HRV during SWS. Taken together, HRV may provide a reliable, indirect index of autonomic activity across stable and disrupted sleep.

Support (If Any): Support: Support is provided by the National Institutes of Health (AA-024892; U54GM115371; P20GM103474).

0150

AFTERNOON NAPPING DOES NOT IMPACT AUTONOMIC FUNCTION IN HEALTHY ADULTS

Jennifer Nicevski[1], Gianna Migliaccio[1], Jeremy Bigalke[1], Emma Kerkering[1], Ian Greenlund[1], Anne Tikkanen[1], Jason Carter[1]
Montana State University [1]

Introduction: Proper overnight sleep is important for autonomic nervous system function. However, less is known about the effects of daytime napping on wake autonomic regulation. In the present study, we assessed autonomic function following a daytime nap. We hypothesized that a 90-minute afternoon nap would significantly improve wake heart rate variability (HRV) and blood pressure (BP).

Methods: Fourteen participants (7 female, 24±1 years, 24 ±1 kg/m2) took part in the study. Subjects completed an autonomic function test after no nap (control condition) or a 90-minute nap opportunity (nap condition) on separate days using a randomized, crossover design. During the autonomic test, participants were fitted with three-lead electrocardiography (ECG), continuous beat-to-beat blood pressure (Finapres NOVA, Netherlands), and respiratory monitoring (pneumobelt). The autonomic function test consisted of 5-minutes of spontaneous breathing, 5-minutes of controlled breathing (15 breaths/min), and a 2-minute cold pressor test (CPT). Frequency-domain HRV in the low (LF, 0.04-0.15 Hz) and high frequency (HF, 0.15-0.4 Hz) components were determined via Fast Fourier transformation. Time-domain HRV was quantified using RMSSD and pNN50. Paired sample t-tests were completed between the control and nap sessions.

Results: Mean total sleep time (TST) for the nap session was 74±5 minutes. Contrary to our hypothesis, an afternoon nap did not change wake heart rate (HR, Control: 70±3 vs. Nap: 68±3 bpm, p = .31) or mean arterial pressure (MAP, Control: 77±3 vs. Nap: 83±3 mmHg, p = 0.70). Similarly, no differences were observed in HF (Control: 2632±628 vs. Nap: 2150±494 ms2, p = .33), LF (Control: 1702±373 vs. Nap: 1345±257 ms2, p = .20), or LF/HF (Control: 92±16 vs. Nap: 92±17%, p = .97) between conditions. RMSSD (Control: 82±12 vs. Nap: 79±11 ms, p = .723) and pNN50 (Control: 43±6 vs. Nap: 47±6%, p = .30) were not impacted by a daytime nap. Lastly, changes in HR (Control: Δ14±3 vs Nap: Δ18±3 bpm, p = .114) and MAP (Control: Δ23±4 vs. ΔNap: 27±4 mmHg, p = .28) during CPT were not different between conditions.

Conclusion: An afternoon nap does not appear to significantly influence autonomic function at rest or during CPT in young healthy adults.

Support (If Any):

0151

SUBJECTIVE SLEEP ONSET LATENCY IS INFLUENCED BY THE SLEEP STRUCTURE AND BODY HEAT LOSS IN HUMAN SUBJECTS.

Ryusei Iijima[1], Akari Kadooka[1], Kairi Sugawara[1], Momo Fushimi[1], Mizuki Hosoe[1], Sayaka Aritake-Okada[1]
Saitama Prefectural University [1]

Introduction: Humans can estimate the time that has elapsed during sleep (time estimation ability; TEA). Although research on the TEA during sleep has advanced in the field of sleep research, few studies have focused on the relationship between the subjective sleep onset latency (SOL), which is an indicator of TEA, and objective sleep structures, body heat loss, and body temperature. This paper investigates the association of the subjective SOL with sleep structures such as the objective SOL, duration of each sleep stage, subjective sleep parameters, and body heat loss in healthy young participants.

Methods: Twenty six participants (7 men and 19 women, mean age of 21.5 ± 0.5 years) having no sleep problems participated in a 1-hour polysomnographic recording that obtained objective sleep parameters during the daytime while temperatures of the skin (i.e., dorsum of the hand and foot, forehead, and subclavian) and eardrum were recorded at intervals of 1 min. The distal–proximal skin temperature gradient (DPG), which is a good predictor of body heat loss and sleepiness, was calculated. Subjective parameters, such as the subjective SOL, sleep time, sleep depth, sleepiness, and mood, were evaluated before and after sleep. We examined the association of the subjective SOL with objective sleep parameters, DPG, and other subjective parameters.

Results: Most participants estimated their sleep latency to be longer than their actual SOL (mean objective SOL of 7.6 min vs. subjective SOL of 13.7 min). The objective SOL was significantly correlated with each sleep stage parameter whereas the subjective SOL was negatively correlated with the stage N2 sleep duration (Rho = −0.454, p = 0.020) and correlated with the stage N2 sleep latency (Rho = 0.402, p = 0.051). Participants who estimated a shorter subjective SOL had a higher DPG before sleep periods than that after sleep onset (Rho = −0.692, p < 0.001). Additionally, the subjective SOL was correlated with the subjective sleep depth, subjective wake after sleep onset, and restorative sleep.

Conclusion: The subjective sleep onset latency in the healthy young participants was affected by the degree of body heat loss before sleep onset and stable shallow nonrapid-eye-movement sleep.

Support (If Any):