constant, there is no statistical difference between LG, HG, and ripple in terms of pathological bain tissue localization. Examining different frequencies for phase in varied behavioral/sleep states using 12 minute awake segments were used in all 10 patients, localization is best via AUROC when delta is the frequency band used to calculate PAC in Slow Wave Sleep / NREM3.Interictal Epileptiform spiking is seen with much higher regularity, although correlated and seen in the SOZ. A great deal of the associated elevations in wide-spectrum (0.5-30 modulating 65-175Hz) PAC (here defined as >2 SD across the entire 2 hour period and assessed across all channels) occurred with IEDs. Since 3 second segments were used to calculate PAC values, sometimes 2 IEDs were within a single epoch although in a small minority of observations. PAC elevations are seen with IEDs and with HFO, although to a greater extend with spikes. However, some IEDs are not associated with elevations in PAC. In fact most detected IEDs were not associated with elevated PAC values or with HFOs for that matter. A minority of IEDs are associated with elevated PAC values and HFOs. At the group and individual levels the average spike count per patient, grouping all SOZ and non-SOZ electrodes together, there is not a statistically significant effect, but if the average count is taken per electrode within a single patient, a significant difference was noted at p = .0031. Subjects on average had 15 (SEM 0.15) electrodes and SOZ electrode counts of 3 (SEM 0.71). When analyzing IED + elevated PAC, grouped SOZ electrodes among patients and within individuals show a strongly significant effect (.0004). When taking into account electrode numbers within each group this significance only increases (.0001). At the individual level, significance of $p<0.05$ is seen for all but one patient in this cohort.

Conclusion: Sleep Stage is critical in the analysis of pathological brain from non-pathological brain and electrophysiologic biomarkers behave differently in the different behavioral states. Here we found evidence to support his proposition in the following ways: low frequency delta phase modulates a broad high frequency amplitude in N3 and has relevance for brain pathology. Phase-Amplitude Coupling is increased in pathologic tissue. Peaks in PAC occur sporadically and infrequently in these patients. PAC is correlated with Interictal Epileptiform Discharges and High Frequency Oscillations however most Interictal Epileptiform Discharges are unrelated to peaks in Phase-Amplitude Coupling. Low Frequency Activity in Beta-band modulates broad high frequency power across low gamma, high gamma, and ripple bands.

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0148

ASSOCIATIONS BETWEEN SUBJECTIVE AND OBJECTIVE SLEEP OUTCOMES AND NIGHTLY PAIN CHANGES IN A CHRONIC PAIN SAMPLE

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Introduction: Patients with chronic pain often experience poor sleep. Research demonstrating the bidirectional nature of the pain/sleep association has focused on interindividual patterns based on between person averages, leaving acute, intraindividual patterns based on night-to-night fluctuations understudied. Because both pain and sleep fluctuate considerably, better understanding of those fluctuations may provide important insight into the pain/sleep relationship. Here we examine inter- and intraindividual associations between subjective and objective sleep outcomes and nightly pain changes in individuals with chronic pain and sleep complaints.

Methods: Adults with chronic pain and sleep complaints (n=169, Mage=52, SD= 12, 95% female) completed 14 days of actigraphy and sleep diaries each morning and evening. Evening diaries recorded pain and sleep medication use (yes/no), and evening pain intensity (0-none, 100-most intense). Actigraphy and morning diaries recorded sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST), and morning pain intensity (0-none, 100-most intense). A nightly pain difference score (morning – evening) was calculated, where positive values indicate worse morning pain relative to previous evening pain. Multilevel models examined the inter- and intraindividual associations between sleep (subjective and objective SOL, WASO, TST) and nightly pain difference scores. Analyses controlled for age, sleep, and pain medication use.

Results: Greater interindividual subjective WASO was associated (B=0.06, SE=0.03, p=0.02) with greater interindividual pain difference scores. Other interindividual sleep outcomes (subjective and objective) were not significantly associated with interindividual pain difference scores. There were no associations between sleep outcomes (subjective and objective) and pain difference scores at the intraindividual level.

Conclusion: Findings show sleep and pain were not linked at the daily, intraindividual level. However, on average, greater WASO was linked with worse morning relative to evening pain. Thus, although a single night of poor sleep may not impact pain, the buildup of fragmented sleep over time may interfere with restorative properties of sleep and exacerbate morning pain. Future work should investigate mechanisms underlying sleep fragmentation (e.g., sleep architecture, physiological arousal) and how such factors relate to nightly pain changes.

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0149

RELIABILITY OF HEART RATE VARIABILITY DURING STABLE AND DISRUPTED POLYSOMNOGRAPHIC SLEEP

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Introduction: Heart rate variability (HRV) is a common metric to estimate autonomic activity during sleep. Frequency-domain HRV is quantified as low (LF) and high (HF) frequency, whereas HRV time-domain indices include root mean square of successive R-R interval differences (RMSSD), and percentage of successive R-R intervals differing by more than 50 ms (pNN50). Despite high HRV use during sleep, it is unknown whether sleep disturbance changes overall reliability of frequency- and time-domain HRV. The purpose of this present study was to determine whether HRV was reliable across arousal-free and arousal-containing periods of sleep.

Methods: Twenty-seven participants (11 male, 16 female, 26±1 years, 27±1 kg/m2) were given an 8-hour sleep opportunity, equipped with continuous two-lead electrocardiography (ECG) and overnight polysomnography (PSG). The ECG recordings were analyzed via fast-Fourier transformation for frequency-domain HRV in a custom software as LF (0.04-0.15 Hz) and HF (0.15-0.4 Hz) HRV. Time-domain HRV was

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.

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0150

AFTERNOON NAPPING DOES NOT IMPACT AUTONOMIC FUNCTION IN HEALTHY ADULTS

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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.

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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.

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