inferred, and a percentage of slow wave coherence across each of the sleep stages was then calculated for each subject.
Results: In our sample, 65 participants (67.7%) endorsed experiencing chronic pain lasting 3 months or longer, and 54 had a history of TBI (56.3%). Participants endorsing chronic pain had a significantly lowered percent of EEG slow wave coherence during NREM sleep than subjects without chronic pain ($p = 0.01$). NREM EEG slow wave coherence did not correlate with SIQR scores in subjects without TBI ($r = -0.03$, $p = 0.90$), but was significantly negatively correlated in subjects with TBI ($r = -0.32$, $p = 0.02$).
Conclusion: EEG slow wave coherence during NREM sleep is correlated with chronic pain complaints in Veterans with a history of TBI, and could be indicative of neuronal dysfunction during sleep. Further research on slow wave coherence is warranted to understand the underlying mechanisms for the association between chronic pain and poor sleep following TBI.
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070

RESPIRATORY, CARDIAC, EEG, BOLD SIGNALS AND FUNCTIONAL CONNECTIVITY OVER MULTIPLE MICROSLEEP EPISODES

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Introduction: Brief intrusions of unintended sleep can occur in various contexts, for example during resting-state fMRI scans. In addition to changes in neural activity, such microsleep episodes are also associated with shifts in respiration and heartrate. Here we investigated how these concurrent changes alter the dynamics of the BOLD signal in the brain and estimates of functional connectivity.
Methods: Ten participants underwent 6 runs of 20 minute resting-state fMRI scans with concurrent respiration, PPG and EEG recording. Realtime eye-closure monitoring combined with post eye-opening self-reports were used to identify microsleep episodes of different durations.
Results: During microsleep, sustained reductions were observed in arousal as assessed by EEG (ratio of alpha to delta and theta bands), as expected. In comparison, cortical BOLD signal exhibited more complex, temporally multiphasic changes which were consistent across different microsleep durations from 4 to 44s: (i) an initial sleep-onset dip reaching a nadir after ~6s, followed by (ii) an increase above wake baseline that plateaued till awakening. On awakening, (iii) a transient positive bump occurred up to 6s, followed by (iv) an undershoot below baseline lasting ~30s. While seen across the whole brain, these changes showed regional variations, e.g., the signal plateau in the thalamus remained below wake baseline. Sleep onset and awakening were also associated with respective reductions and increases in respiration and heart rate, which affect blood oxygen levels. Brain functional connectivity estimates were altered by the frequency of falling asleep, and this was not resolved by global signal regression.
Conclusion: Falling asleep and awakening are shown here to be associated with large, widespread BOLD signal changes consistent across varied durations of microsleep. These signal changes are intimately intertwined with shifts in respiration and heart rate, which are influenced by common brainstem nuclei controlling sleep. These autonomic contributions to 'brain signal' changes at microsleep onset and awakening are integral to sleep, and urge the integration of autonomic and central nervous system contributions to BOLD signal into frameworks for understanding brain function using fMRI. In addition, the correlation between frequency of microsleep and extent of altered functional connectivity highlight the need to minimize sleep during resting state scans.
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ACTIGRAPHY-DERIVED SLEEP METRICS ARE NOT RELATED TO CENTRAL HEMODYNAMICS OR ARTERIAL STIFFNESS IN HEALTHY ADULTS

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Introduction: Insufficient sleep is an emerging risk factor for cardiovascular disease. To evaluate the hypothesis that decrements in vascular function, due to poor sleep, may serve as a mechanistic link between sleep and cardiovascular disease, we explored relationships of actigraphy-derived sleep metrics with central hemodynamics and arterial stiffness in healthy young adults.
Methods: A total of 23 women and 27 men (23±5 yrs), free of known cardiovascular, metabolic, and renal disease, and not using sleep medication, participated in this study. ActiGraph GT9X wrist-worn accelerometers were used to measure sleep efficiency, total sleep time, wake after sleep onset, and number of awakenings over a seven-day period. Vascular health measures including central pressures and augmentation index at a heart rate of 75 beats per minute (AIx@75) were quantified via pulse wave analysis, and carotid femoral pulse wave velocity (cf-PWV) was assessed using applanation tonometry. Gender-specific z-scores for each of the sleep metrics were summed to assign each participant a "sleep score" (higher score = better sleep), and relationships between sleep scores and vascular health measures were explored using Pearson correlation coefficients.
Results: In men, sleep score (range: -4.92 to 9.10) was not related ($P>0.05$) to central systolic (114±15 mmHg, $r=-0.26$) or diastolic (72±7 mmHg, $r=-0.21$) pressures. Similarly, in women, sleep score (range: -5.02 to 5.34) was not related ($P>0.05$) to central systolic (103±11 mmHg, $r=-0.09$) or diastolic (72±10 mmHg, $r=-0.21$) pressures. Sleep score also failed to predict ($P>0.05$) indices of arterial stiffness, AIx@75 (men = 3.1±12.3, $r=0.04$; women = 5.2±9.5, $r=-0.25$) and cf-PWV (men = 6.2±0.8 m/s, $r=-0.12$; women = 5.7±0.5 m/s, $r=-0.10$).
Conclusion: In young healthy individuals, actigraphy-derived sleep characteristics were not related to central hemodynamics or non-invasive indices of arterial stiffness. Previously documented relationships between sleep and vascular function may be limited to less healthy populations, poorer sleepers, or only for certain sleep metrics.
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SLEEP SPINDLE HARMONICS IN INSOMNIA

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Introduction: Prior research has reported NREM spectral EEG differences between individuals with insomnia and good-sleeper controls, including elevated high-frequency EEG power (beta/gamma bands, ~16-50Hz) and, to a lesser extent, elevations in sleep spindle parameters. However, the mechanisms driving these differences remain unclear. Harmonics have been observed in EEG data as spectral peaks at multiples of a fundamental frequency associated with an event (e.g., for a 14Hz spindle, the 2nd harmonic is expected to be a peak at 28Hz).