Estimation of cardiopulmonary coupling by ECG-derived respiration signals during sleep

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Both the cardiac and the pulmonary systems are controlled by the autonomic nervous system and their activities are coupled with each other. The strength of coupling is affected by the activation and dominance of sympathetic or parasympathetic tones. As autonomic tones vary parallel to the progress of sleep, accompanying variation of cardiopulmonary coupling (CPC) can be expected. In this study, we derived respiration signals from ECG and evaluated the accuracy of derived respiration signals as a measure of CPC strength. If the coupling is strong, the accuracy of ECG-derived respiration (ECGdR) signals will increase. Otherwise, the estimation error will increase.

A whole night polysomnogram (PSG) was recorded for four healthy subjects (4 males, 32 ± 3.74 years) and sleep stages were scored by the experts. For each sleep stage, three successive motion-free epochs (i.e. total of 90 s) were selected for analysis. Respiration was derived from ECG by R-peak detection, calculation of the RR intervals, interpolation with cubic-spline function and filtering with 0.2–0.8 Hz of passband (the normal frequency range of the respiratory signals). For each sleep stage, the peaks of ECGdR spectra were compared with peak frequencies of reference respiration signals measured from the nose with thermocouple sensors during PSG recording. And, respiration intervals from the ECGdR and reference respiration signals were compared and the root mean square error (RMSE) was calculated as a measure representing the CPC strength.

During the non-REM sleep, RMSE decreased as sleep progressed from light into deep sleep. Also, in some cases, RMSE during the REM sleep was much larger than during the non-REM sleep. We suggest that it is caused by the relative parasympathetic dominance during non-REM sleep vs. REM sleep. In conclusion, ECGdR could be utilized for the evaluation of CPC as well as for the estimation of the depth of non-REM sleep.

doi:10.1016/j.ijpsycho.2010.06.160

Sleep deprivation selectively impacts different prefrontal cortex regions

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Research findings demonstrating short term sleep deprivation effects on prefrontal cortex (PFC)-related cognitive performance (such as temporal memory, decision making and language skills) have led some authors to suggest that sleep deprivation impacts primarily PFC-associated performances (Harrison and Horne, 2000). However, little is known about the selective effects of sleep deprivation on specific regions within the PFC and its relation to the possible mechanism by which sleep reduction affects PFC activity. The present study examined the impact of mild (24 h) sleep deprivation on cognitive performance related to different parts of the PFC: the Dorsolateral PFC (DLPFC, Brodmann’s areas 8, 9, 45) which is associated with attention, cognitive flexibility and verbal fluency (Zakzanis et al., 2005), the Orbitofrontal Cortex (Brodmann’s areas 10, 11, 47), a PFC region associated with the application of reward rules and risk taking behavior (Hartstra et al., 2010), and the Ventrolateral PFC (Brodmann’s areas 44, 45, 47) which is associated with speech comprehension (Saur et al., 2008). A total of 14 healthy young subjects (age range 21–23) who were screened for any sleep disorders were tested over two days. One test day followed normal nocturnal sleep and the other test day followed 24 h of sleep deprivation. Thirteen subjects completed all the cognitive tasks. For one subject, data is available only for the letter cancellation task. Testing took place approximately at 10 a.m. and included three DLPFC activity-related tasks (Number Cancellation Task, Trail Making Test and the Wisconsin Card Sorting Test) and one orbitofrontal activity-related task (the Iowa Gambling Test). Subjects also performed a speech comprehension task related to Ventrolateral PFC activity, during which they were asked to repeat a one syllable word at each of the following conditions—1. Quiet. 2. Speech noise background 3. White noise background and 4. 60% compression.

Results indicated a significant effect of one night sleep deprivation as manifested by longer completion times at the Number Cancellation Task (t(13) = –4.149, p < 0.01) and Trail Making Test (numbers only, t(12) = –3.154, p < 0.01). At the Speech Comprehension task, a significantly smaller number of words were identified after one night of sleep deprivation during the speech noise background (t(12) = –3.88, p < 0.01) and the 60% compression (t(12) = –3.481, p < 0.01) conditions. No significant results have been found with regard to the Iowa Gambling Test performance. Conclusions: In the current study, Speech Comprehension, A Ventrolateral PFC-related cognitive performance, was significantly impaired after 24 h of sleep deprivation. Sleep deprivation predominantly affected attention-related DLPFC focused tasks (Number Cancellation and Trail Making A). No sleep deprivation effects have been found at the Iowa Gambling Test,
an Orbitofrontal PFC-related cognitive performance task. These findings suggest that sleep deprivation selectively affects different PFC regions.

References

doi:10.1016/j.jippsycho.2010.06.162

Neuropsychological characteristics of nightmare sufferers: Preliminary evidence for impaired frontal inhibitory functions

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Introduction: Different lines of research suggest that REM sleep and dreaming involve the intense functioning of an emotional frontolimbic network facilitating off-line emotional information processing (Walker, 2009). Moreover, this emotional processing may facilitate the regulation of affective states by restructuring emotional memories into broader cortical networks. Dysphoric dreaming and frequent nightmares may reflect the impairment of such emotional integration and reorganization. Recently, Levin and Nielsen (2007) traced a neurocognitive model of dreaming that integrates the advances of the neuroscience of sleep, clinical sleep research, and the neuroscientific literature on affective regulation. One of the model's main assumptions is that nightmare sufferers may be characterized by impaired prefrontal inhibitory functions causing amygdalar over-reactivity, and thus, malfunctioning affective regulation during the intense emotional state of dreaming. In order to test this hypothesis, we aimed to characterize the frontally localized inhibitory functions of nightmare sufferers.

Methods: Thirty two nightmare sufferers and matched controls were selected from a large pool of university students after completing different sleep and dream-related questionnaires. A neuropsychological test battery (emotional go/no-go, fluency, and Porteus maze tasks) and the STAI questionnaire were used to assess the participants’ frontal inhibitory functions and levels of anxiety.

Results: Nightmare sufferers were characterized by significantly higher rates of perseveration errors in the Fluency tasks and dream disturbances were associated with impaired frontal inhibitory functions. These results remained significant after controlling for sleep quality and levels of anxiety.

Conclusion: Our results provide partial support for the neurocognitive model of dreaming (Levin and Nielsen, 2007) and suggest that the neuropsychological and psychophysiological characterization of nightmare sufferers would shed more light on the cortical mechanism of affective regulation in sleep. We consider that the neuropsychological and psychophysiological investigations of different parasomnias are an intriguing way to integrate clinical and experimental sleep research.

References

Retina inversion and visual fiber crossings are the principal neuroanatomical mechanisms, providing the mirror transformations in the visual system

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doi:10.1016/j.jippsycho.2010.06.164

The impact of a sporadic poor night’s sleep: Contingent negative variation as an event-related parameter of cognitive preparation processes

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Introduction: Subjective sleep quality is known to fluctuate from night to night, especially in insomnia patients. The relevance of these subtle changes for subsequent daytime cognitive functioning is unclear. In this study, we investigated cognitive performance following a subjectively good versus subjectively poor night’s sleep in healthy sleepers. In addition to reaction times, event-related potentials were analyzed.

Methods: Participants were healthy sleepers (N = 23) between 18 and 28 years old. About half of the subjects reported an acceptable sleep quality with regard to the night before in comparison with their regular sleep quality. Present results suggested that a sporadic poor night’s sleep did not differ between the Poor and the Good Night groups (t(11) = 1.041; n.s.). A mixed 2 (poor vs. good night group) × 4 (location) repeated measures ANOVA indicated a significant interaction effect for N1 as well as for P2 (F(3.33) = 3.197; p < .05 and F(3.33) = 3.018; p < .05 respectively). Posthoc tests showed that N1 at Fpz was marginally significantly larger in the Good Night than in the Poor Night group (p < .10). On the contrary, the amplitude of P2 at Fpz was significantly larger in the Poor Night than in the Good Night condition (p < .05). No other significant main or interaction effects were found, except for a main effect of location for all three ERP components.

Conclusion: Present results suggested that a sporadic poor night’s sleep did not result in a deterioration of cognitive performance on a stop-signal task in terms of reaction times. However, changes in the ERP components indicated that the underlying cognitive processes were affected in that a lower level of attention orientation towards imperative stimuli (smaller N1) was compensated for by improved suppression of irrelevant information (larger P2) after a poor night as compared to a good night sleep. Devoto et al. (2003) have previously found indications of such mechanisms in insomnia patients. Present findings might in part question the specificity of these cognitive compensation strategies for insomnia.

doi:10.1016/j.jippsycho.2010.06.164