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EEG beta power and heart rate variability describe the association between cortical and autonomic arousals across sleep

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ABSTRACT

Cortical and autonomic arousals have been found to be closely associated. As arousal events are not evenly dispersed across sleep, we hypothesized the relationship between high frequency electroencephalogram (EEG) power and autonomic arousal indices differ between non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. One night of polysomnographic recording was performed on a group of 18 subjects using a portable recorder. The EEG was collected from C3/Fz. Sleep stages and cortical arousals were visually scored. Cardiac autonomic modulation was assessed from heart rate variability, where the high frequency power (HF) indicates parasympathetic modulation, and the low frequency to high frequency power ratio (LF/HF) represents sympathetic modulation. During NREM sleep, EEG beta power was significantly correlated with LF/HF ($r = 0.40 \pm 0.06$), and the relationships were more positive than during REM sleep (LF/HF: $r = 0.20 \pm 0.08$; EOG power: $r = -0.13 \pm 0.05$). The relationship of beta power with LF/HF was associated with the incidence of cortical arousal, particularly during NREM sleep. With respect to alpha power, it was only marginally related to HF or LF/HF. In addition, the coefficients of determination were lower for alpha power than for beta power in terms of the relationships to HF, LF/HF and EOG power. This study shows a higher relationship between cortical and autonomic activation during NREM sleep, and the association is better described by beta power. This finding suggests NREM sleep may be of greater therapeutic potential in view of reducing cardiovascular disease associated with sleep fragmentation, and beta power may provide a better index to evaluate the effect.

1. Introduction

Sleep fragmented by frequent arousals, as in sleep apnea, has been associated with sympathetic overactivity and cardiovascular disease (Smith et al., 1998; Chouchou et al., 2013; Pepin et al., 2014). These arousals can be categorized as either cortical arousal or autonomic arousal, based on the presence of specific physiological components, namely EEG desynchronization and sympathetic activation, respectively (Halasz et al., 2004). Several investigations have revealed a close relationship between cortical and autonomic arousals that higher sympathetic activity is associated with the occurrence of cortical arousal (Chen et al., 2013; Bonnet and Arand, 1997; Sforza et al., 2002; Abbott et al., 2013). The majority of research focuses on analysis of arousal events (Bonnet and Arand, 1997; Sforza et al., 1999; Sforza et al., 2000; Sforza et al., 2002; Togo et al., 2006). However, cortical arousals were not homogenously distributed across sleep (Bonnet and Arand, 1997; Terzano et al., 2000). Thus, it is not known if the relationship between cortical and autonomic arousals persists across the whole sleep.

For the scoring of cortical arousal, the alpha rhythm appears to be the frequently identified EEG morphology (Halasz et al., 2004; Bonnet and Arand, 2007b). The presence of alpha waves during sleep states without arousal episodes, nevertheless, suggests functional correlates of alpha beyond arousal (Pivik and Harman, 1995; Ehrhart et al., 2000). On the contrary, the functional role of EEG beta activity appears to be more unitary. Beta activity has been suggested to be involved in behavioral arousal and attentional processes (Tzischinsky and Lavie, 1994; Lamarche and Ogilvie, 1997), and is increased when a cortical arousal occurs (Black et al., 2000; Poyares et al., 2002; Thomas, 2003). Observations made on the EEG response accompanying autonomic activation have shown increases in both alpha and beta power (Togo et al., 2006), but it is not known if alpha and beta rhythms differ in their relationships with autonomic arousal when examined across the whole sleep.

Abbreviations: HF, the high frequency power of heart rate variability; LF/HF, the low frequency power to HF ratio of heart rate variability; NREM, non-rapid eye movement sleep; REM, rapid eye movement sleep.

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In order to measure autonomic arousal, spectral analysis of heart rate variability (HRV) provides a non-invasive assessment (Bootsma et al., 1994; Yang et al., 2002; Kuo et al., 2004; von Borell et al., 2007), in which the power in high frequency (HF) component is taken as parasympathetic activity and the low frequency to high frequency power ratio (LF/HF) is taken as sympathetic activity (Bootsma et al., 1994; Rajendra Acharya et al., 2006). Based on previous findings on the relationship between cortical and autonomic arousals, we hypothesize the relationship between cortical and autonomic arousals differs between different sleep stages, and EEG alpha and beta power differ in their relationships with autonomic arousal.

2. Materials and methods

2.1. Subjects

In the current study, a group of subjects, 9 young women and 9 young men, were investigated. All were in good health (BMI = 20.41 ± 0.55) with regular night sleep habits and no hypnotic drug abuse or above-average alcohol, caffeine or nicotine consumption. None had a history of psychopathology or any medical condition known to influence sleep (including noticeable snoring) or the autonomic nervous system. All subjects gave written informed consent, and the procedures used in this study were approved by the Institutional Review Board of National Yang-Ming University.

2.2. Data recording

Electrophysiological signals were recorded using a miniature (5.2 × 3.1 × 1.2 cm, 11 g) physiological signal recorder (TD1, Taiwan Telemedicine Device Company, Taiwan) fixed onto the chest wall with tape. Subjects were allowed to carry out all normal daily activities except for vigorous physical exercise, and they slept at their own homes. Four electrophysiological signals, EEG, electrooculogram (EOG), electromyogram (EMG), and electrocardiogram (ECG) were recorded to give a simplified version of standard sleep monitoring (Rechtschaffen and Kales, 1968; Kuo and Yang, 2009). The EEG was recorded from C3/Fz. The EOG was recorded from a pair of differential electrodes placed 1 cm above right outer canthus and 1 cm below left outer canthus. The EMG was recorded from the submental area. The ECG was recorded from V2 site on the chest. The EEG, EOG, EMG, and ECG were respectively amplified 2000, 1000, 1000, and 250 fold, and then separately filtered at 0.34 Hz to 53 Hz, 0.034 Hz to 53 Hz, 16 Hz to 113 Hz and 1.6 Hz to 113 Hz, respectively. After digitization of the EEG, EOG, EMG, and ECG signals at different sampling rates (125, 125, 250, and 500 Hz, respectively), the acquired dataset was stored on a flash memory for subsequent off-line analysis. Due to the limitation of the ambulatory device, breathing was not recorded.

2.3. Signal analysis

Computer assisted sleep analysis was carried out according to the criteria set by American Academy of Sleep Medicine (AASM) (Iber et al., 2007), and verified by a qualified sleep technician. The consciousness states were classified into wakefulness, NREM sleep and REM sleep. Cortical arousals were scored using the AASM criteria (Iber et al., 2007). Autonomic function was assessed using HRV analysis, from which LF/HF indicates sympathetic activity and HF indicates parasympathetic activity (Kuo et al., 1999; Kuo and Yang, 2009). Only epochs scored as NREM or REM sleep were analyzed.

For the HRV analysis, QRS complexes in the ECG were identified, and the resulting stationary R-R intervals (RR, the inverse of heart rate) were resampled and linearly interpolated at a rate of 64 Hz (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Kuo et al., 1999; Kuo and Yang, 2009). The resampling rates of the EEG and EOG signals were 64 Hz. The EEG, EOG, and RR signals were truncated into successive 64-s epochs and then subjected to Hamming windows with 50% overlapping (Kuo and Chan, 1993). The estimation of the power density was calculated by fast Fourier transform (Kuo et al., 1999). For each time segment, the LF (0.04–0.15 Hz) and HF (0.15–0.4 Hz) power of the RR spectrogram, the alpha (8–12 Hz) and beta power (16–32 Hz) of the EEG spectrogram, and the low frequency power (0.05–0.25 Hz) of the EOG spectrogram were quantified (Kuo and Yang, 2009).

Motion artifacts in EEG were automatically rejected with the use of an additional recording of 3-axis accelerometer signals (the accelerometer was incorporated into the miniature recorder), which has been applied to recognize artifacts induced by movements (Martini et al., 2010; Virtanen et al., 2011). Bodily activity was quantified by the standard deviation of the acceleration measures, and epochs with above-threshold activity were excluded from analysis (Fig. 1B). The threshold was manually adjusted to avoid contamination of the EEG spectrogram by motion artifacts.

2.4. Statistical analysis

HF, LF/HF and PEOG were logarithmically transformed to correct for the skewed distribution (Kuo et al., 1999). The relationships between EEG spectral power and HRV measures were calculated using Pearson's correlation (r), and the correlation coefficients were then made to distribute normally using Fisher's Z-transformation (Zr). The goodness-of-fit of the regression line (the proportion of variance in one variable explained by differences in the other) was assessed by the coefficients of determination (r^2, the squared correlation coefficient). Significance of the Zr was tested using one sample *t*-tests against zero. Comparisons of the Zr or r^2 between alpha power and beta power or between NREM sleep and REM sleep were made using paired *t*-tests. To assess the statistical interaction between EEG spectral power (alpha and beta power) and sleep stages (NREM and REM sleep) with respect to Zr and r^2, two-way ANOVA with repeated measures (two within-subjects factors: frequency and stage) was applied. Statistical significance was taken at $P < 0.05$. Values are expressed as mean ± SEM.

3. Results

3.1. Sleep data for the subjects

The subjects had total sleep time of 412.67 ± 10.75 min (stage 1: 10.46 ± 1.18%, stage 2: 54.23 ± 1.35%, stage 3: 8.39 ± 1.66%, REM: 26.92 ± 0.83%), with sleep latency of 19.97 ± 3.21 min and sleep efficiency of 91.98 ± 1.06%. Raw traces of the EEG, RR, and EOG with corresponding spectrograms from an example subject are presented in Fig. 1. To reject motion artifacts, sleep data accompanied with above-threshold activity were excluded from analysis (Fig. 1B). Compared with the unprocessed EEG spectrogram (Fig. 1A), the artifact-associated power spectral density was removed.

3.2. EEG and autonomic dynamics during NREM sleep

During NREM sleep, beta power was inversely correlated with RR ($Zr = -0.19 \pm 0.06$, $P < 0.05$), and in parallel, had a higher relationship to LF/HF ($Zr = 0.45 \pm 0.04$, $P < 0.05$) but a marginal relationship to HF ($Zr = 0.07 \pm 0.05$, $P = 0.20$, Fig. 2A). EOG activity has been previously suggested to contain information on sympathetic activity (Kuo and Yang, 2009) and therefore it was, as expected, positively correlated with beta power throughout NREM sleep ($Zr = 0.51 \pm 0.06$, $P < 0.05$). With respect to alpha power, however, no correlation with RR, HF, LF/HF or EOG power was noted during NREM sleep, and the correlation coefficients with LF/HF and EOG power were significantly lower than those of beta power ($P < 0.05$, Fig. 2A). In addition, during NREM sleep (Fig. 2B), beta power had higher coefficients of determination than did