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Quantitative Evaluation of Opposing Drives in Sleep-Wake Regulation Using EEG Measures

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Abstract

The opponent model of sleep-wake regulation proposes two opposing drives for sleep and wake. However, accurately measuring these drives in the electroencephalographic (EEG) signal has been challenging. In previous research, we identified that the first and second principal components of variation in the EEG power spectrum can serve as markers for the sleep and wake drives, respectively. This study aimed to validate and expand the measurement methodology by introducing a novel approach to uncover differences in the EEG signatures of these drives. New single EEG measures were calculated from recorded waking and sleep EEG signals of 100 participants, encompassing night sleep, multiple naps, and sleep deprivation. These measures captured differences between distinct sleep-wake sub-states by analyzing the differences between pairs of EEG spectra. Two typical patterns emerged as spectral EEG signatures of the sleep and wake drives. Principal component analysis of the calculated single measures yielded the two largest components representing these opposing drives. The time courses of scores on these components closely resembled the time courses of scores on principal components of variation in the EEG power spectrum. The findings demonstrate that this methodology enables quantitative evaluations and model-based simulations of the regulatory processes underlying normal and abnormal sleep-wake alternations.

Keywords: Sleep-wake Regulation, EEG Power Spectrum, Principal Component Analysis, Opponent Model, EEG Signatures

I. Introduction

Since the introduction of the two-process model, which suggests two fundamental processes in sleep-wake regulation (homeostatic and circadian), slow-wave activity in the electroencephalographic (EEG) signal has been widely considered as the standard for quantifying the homeostatic process or sleep drive [1]. It has been used to describe, predict, and simulate the repayment of sleep debt during Non-Rapid Eye Movement (NREM) sleep following periods of wakefulness. However, the circadian process, which is another basic regulatory process, was not expected to be identified through EEG analysis. An alternative conceptualization known as the opponent model interprets these two processes as competing drives for sleep and wake, yet EEG analysis was not anticipated to reveal an indicator of the opposing wake drive associated with the circadian process [2]. The EEG power spectrum, as a reflection of the combined influences of the opposing sleep-wake regulatory processes, can be separated into orthogonal and uncorrelated principal components to discern the brain signatures of these processes. It has been observed that the score on the 2nd principal component shows consistent changes during the transition from the alertness sub-state of wakefulness to the deepest sub-state of sleep, known as slow wave sleep or N3 stage [3]. In contrast, the score on the 1st principal component remains low during wakefulness and stage 1 sleep, but rapidly increases after entering established NREM sleep stages (stages 2 and 3). These temporal patterns of the principal component scores suggest that the 1st and 2nd components of the EEG spectrum represent the opposing drives for sleep and wake, respectively [4-6]. While slow wave activity in the EEG power spectrum appears to reflect the combined effect of the 1st and 2nd principal components, it cannot be interpreted as a pure

slow wave activity in the waking EEG signal provide no information about these drives, unlike when measured during sleep. Increased propensity to enter NREM sleep is often referred to as "accumulation of sleep pressure," but slow wave activity indexes are insufficient for directly measuring such accumulation until the actual onset of NREM sleep. In contrast, we have previously demonstrated that the time course of the score on the 2nd principal component, obtained from analyzing the waking EEG signal, reliably informs about the processes underlying transitions between wakefulness sub-states. Lastly, data on slow wave activity and the score on the 1st principal component differ in their indication of the involvement of stage 1 (N1) in the process of sleep debt repayment.

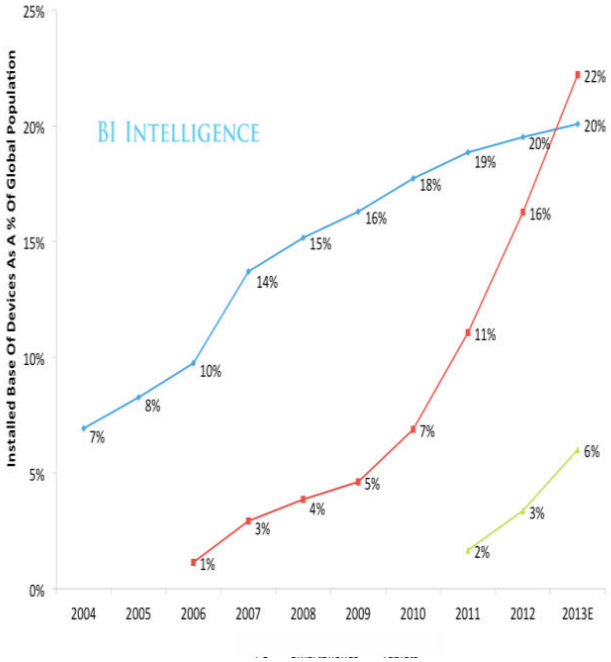

Fig. 9. Growing propensity to enter NREM sleep

VII. Conclusion

Although slow wave activity indexes are widely used as EEG indicators for one of the fundamental regulatory processes proposed by sleep-wake regulation models, they are inadequate for examining the postulated interaction between these processes. Previous research has suggested using two other indexes, namely the scores on the 1st and 2nd principal components of the EEG spectrum, to differentiate the contributions of these basic sleep-wake regulatory processes. The present analysis builds upon and validates this finding, introducing a slightly different approach aimed at directly extracting the EEG signatures of the opposing regulatory processes. Differential spectra, which represent the discrepancies between pairs of spectra at different levels of these processes, were calculated and utilized to derive a comprehensive set of single measures summarizing these differences. Through principal component analysis of this set, significant loadings of each individual measure on either the 1st or 2nd principal component were identified. These measures, akin to the scores on the 1st and 2nd principal components of the EEG spectrum, were interpreted as EEG indicators for the two opposing sleep-wake regulatory processes. Moreover, this approach enabled the visualization of the EEG signatures associated with each process. Consequently, this methodology is recommended to facilitate quantitative evaluations and model-based simulations of the opposing regulatory processes that underlie normal and abnormal alterations between sleep and wake states.

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