ELECTROENCEPHALOGRAPHIC CHANGES AFTER ONE NIGHT OF SLEEP DEPRIVATION

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ABSTRACT - Total or partial sleep deprivation (SD) causes degrading effects on different cognitive and psychomotor functions that might be related to electrophysiological changes frequently observed. In the present study, we investigated the effects of one night of sleep deprivation on waking EEG. Experimental protocol consisted of recording electroencephalographic data from eleven healthy young subjects before (baseline) and after (time 2) one night of sleep deprivation. A natural log transformation was carried out and showed a significant increase in theta T6 (p=0.041), O2 (p=0.018) and OZ (p=0.028); and delta T6 (p=0.043) relative power; and a decrease in alpha Fp1 (p=0.040), F3 (p=0.013), Fp2 (p=0.033), T4 (p=0.050), T6 (p=0.018), O2 (p=0.011) and Oz (p=0.025) and beta (p=0.022) absolute power. These outcomes show that the EEG power spectra, after sleep deprivation, exhibit site-specific differences in particular frequency bands and corroborate for the premise of local aspects of brain adaptation after sleep deprivation, rather than global.

KEY WORDS: sleep deprivation, qEEG, power spectral analysis, cortical activity.

Respostas eletrencefalográficas após uma noite de privação de sono

RESUMO - Privação total ou parcial de sono causa efeitos deletérios em diferentes funções cognitivas e psicomotoras, que podem estar relacionados às mudanças eletrofísiológicas frequentemente observadas. No presente estudo, investigou-se os efeitos de uma noite de privação de sono nas respostas eletrencefalográficas de repouso. O protocolo experimental consistiu na coleta e gravação dos dados do qEEG de onze sujeitos jovens e saudáveis antes (momento baseline) e após (momento 2) uma noite de privação de sono. Todos os dados sofreram transformação logarítmica, que evidenciou um aumento significativo nas potências relativas de teta T6 (p=0,041), O2 (p=0,018) e OZ (p=0,028); e delta T6 (p=0,043). As bandas de freqüência mais rápidas alfa Fp1 (p=0,040), F3 (p=0,013), Fp2 (p=0,033), T4 (p=0,050), T6 (p=0,018), O2 (p=0,011), Oz (p=0,025) e beta (p=0,022) apresentaram reduções significativas na potência absoluta. Os resultados demonstram que o espectro de potência exibe diferenças específicas em freqüências de bandas diferentes, e corroboram para a premissa de que o cérebro, após privação ou perda de sono, promove ajustes e adaptações locais, ao invés de globais.

PALAVRAS-CHAVE: privação de sono, qEEG, análise do espectro de potência, atividade cortical.

Sleep is essential to proper brain functioning and the lack of it results in performance impairment during everyday cognitive tasks[1-7] Sleep deprivation (SD) seems to exert local and specific effects on the brain, rather than global. Recent electroencephalographic and neuroimaging studies revealed that the prefrontal cortex (PFC) is more responsive to sleep deprivation then other brain areas[2,8-12], which is expected since the frontal region has a greater restorative need than other areas of the brain. In fact, during non-REM sleep, in the rebound sleep after SD, the increase in delta-wave frequency (1-4 Hz, slow wave activity related to deep sleep) occurs first in the PFC and later on other areas of the cortex[13,14], suggesting the importance of the initial recovery in this region. A few recent studies[2,3,8] perceived that, contrary to all expectations, after sleep deprivation, the PFC was significantly more activated during a different tasks

supporting the hypothesis of local dynamic compensatory changes in the brain electrical activity after sleep deprivation.

Studies concerning quantitative electroencephalography (qEEG) are still contradictory and have divergent results. The majority of EEG investigations reports the effects of SD on the rebound sleep, and considering the extended literature on sleep, very few studies have tried to observe electrophysiological changes during prolonged wakefulness[15-19]. EEG is a useful and sensitive tool related to cognitive processes of vigilance and, therefore, it can be used to understand the consequences of sleep loss in the brain electrical activity and functional organization while individuals are still awake. It has been demonstrated by EEG spectral analysis that SD alters cortical activity during prolonged wakefulness[15-19]. Spectral power analysis[17] has revealed an increase in the absolute power of theta, alpha and beta bands after sleep deprivation; but the same authors[18] observed later a reduction in alpha, although theta remained increased. Some studies also reported a reduction in the absolute power of all bands, except for delta, and a significant increase in theta absolute power in frontal and temporal areas[11]. Corsi-Cabrera[6] observed an increase in alpha and beta absolute power in the occipital area. As noted, studies involving EEG are still very much at odds, probably due to different methodologies and individual variability.

Therefore, the aim of this study was to observe and attempt to elucidate the electrophysiological changes followed by one night of sleep deprivation.

METHOD

Subjects – The sample consisted of 11 individuals, 4 males and 7 females, with ages varying between 21 and 40 years (30.8±6.0 years). All subjects were healthy, right-handed, non-smokers, and were not making use of any psychoactive or psychotropic substance at the time of the test. To ensure that subjects did not present any impairment of their physical and mental health, a questionnaire was applied. The questionnaire also aimed at identifying food intake, body temperature, fatigue, and drugs use, among others. Subjects were not allowed to consume any alcoholic beverages or caffeine. The Edinburgh inventory was used to assess laterality and exclude left-handed individuals from the experiment. All subjects signed a consent form, where the experimental condition was thoroughly described. The Psychiatric Institute's Ethics Committee approved the experiment.

Study and procedures – Electrophysiological recording occurred in two different times: pre- and post-sleep deprivation (which itself took all night long). Subjects arrived at the laboratory by 8:00 p.m. and performed the first qEEG recording (time 1- baseline). During the whole period of sleep deprivation (night), volunteers played games, watched videos and carried out recreational activities, and were also fed each three or four hours. Two experimenters who were required to ensure continuous wakefulness of the subjects monitored sleep deprivation continuously. Subjects abstained from smoking or drinking xanthine-containing beverages (coffee, tea, cola or soft drinks). No concurrent treatment was allowed during the study. At 7:00 a.m. (time 2- after sleep deprivation) of the subsequent morning, all subjects repeated the same identical qEEG recording.

Electroencephalogram recording – The study design respected the International Pharmaco-EEG group guidelines. Subjects were seated comfortably in a sound-light-attenuated room, while EEG was collected from 20 monopolar derivations for five minutes (eyes closed, alert but resting). Data were collected with eyes closed in order to observe the cortex electrical activity without any external stimuli, minimizing possible visual artifacts. Electrodes were positioned according to the International 10/20 System (referred to linked earlobes with ground at FPZ). All electrode impedances were kept below 5 kΩ. The signal was amplified with a gain of 22,000, analogically filtered between 0.01 Hz (high-pass) and 100 Hz (low-pass), and sampled at 240 Hz using a Braintech-3000® *(EMSA-Medical Instruments,* Rio de Janeiro, RJ, Brazil) EEG acquisition system. The EEG was recorded by means of the software *ERP Acquisition* (Delphi 5.0®, *Borland-Inprise),* developed at the Brain Mapping and Sensorimotor Integration Lab, employing the following digital filters: *notch* (60 Hz), high-pass of 0.3 Hz and low-pass of 25 Hz. Visual inspection was employed for detection and elimination of artifacts. Eye-movement (EOG) artifact was monitored with a bipolar electrode montage using two 9-mm-diameter electrodes attached superior to and on the external cantus of the right eye.

Data analysis – At least two minutes of artifact-free data were extracted from the EEG's total record for quantitative analysis. A Matlab 5.3® *(The Mathworks Inc.,* Massachusetts, USA) routine was implemented to perform a spectral analysis. For each of the 20-monopolar derivations, absolute and relative powers (μV2) were computed for the delta (1.0–3.5 Hz), theta (4.0–7.5 Hz), alpha (8.0–12.0 Hz), and beta (13–25 Hz) frequency bands.

Statistical analysis – Data were expressed as mean±s.d. The statistical software SPSS for Windows was used for all data analysis. Preliminary descriptive examinations revealed that none of the evaluated EEG indexes had a normal distribution. To obtain a better approximation of a normal distribution, data from the power spectra were submitted to a log transformation (LOG 10, log absolute power). Significance levels were set at $p \le 0.05$.

RESULTS

One night of sleep deprivation caused a significant power decrease in the frontal, temporal and
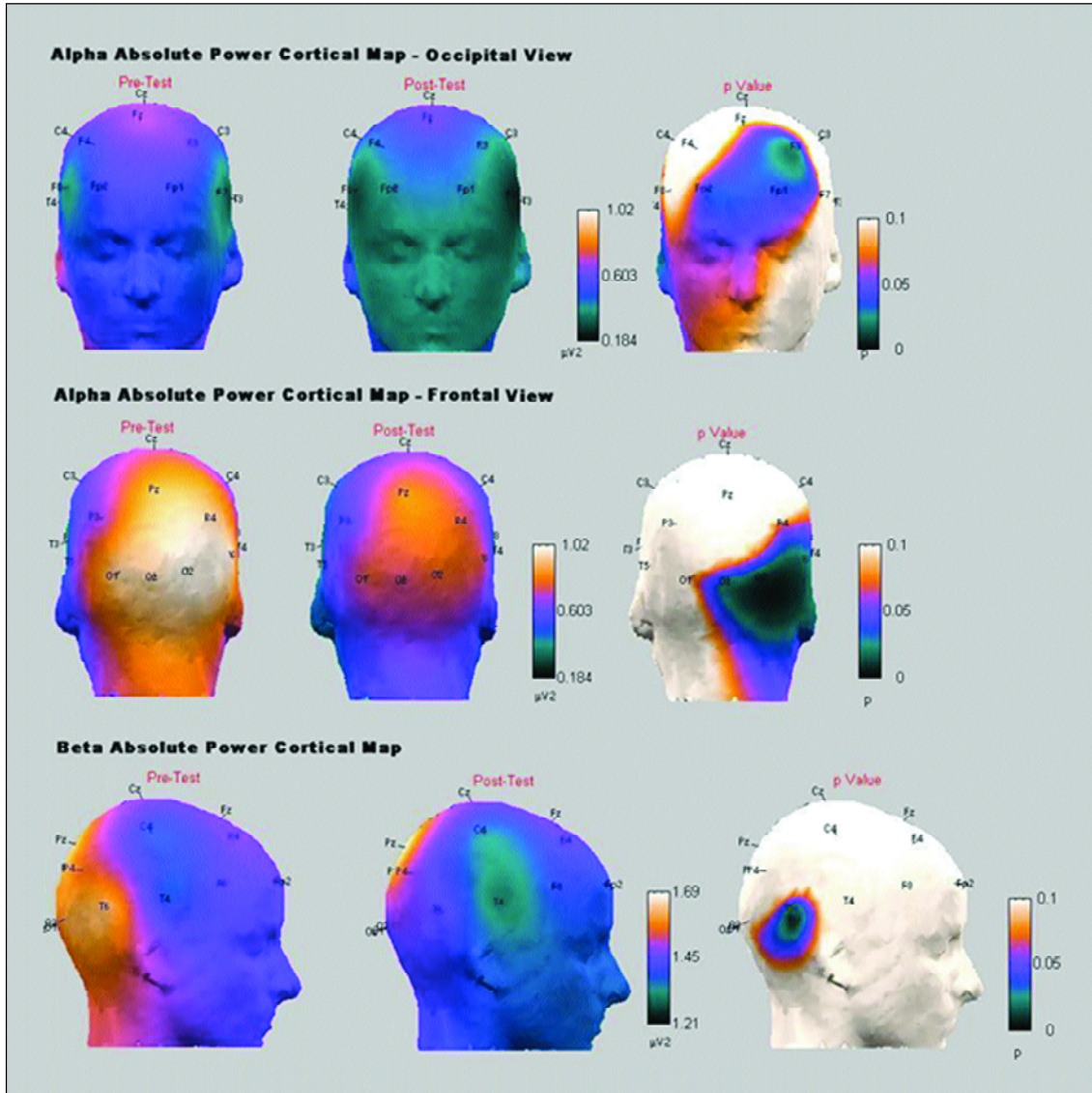
Fig 1. Alpha absolute power cortical map showing both frontal and occipital views. One night of sleep deprivation caused a significant power decrease in frontal, temporal and occipital areas in this frequency band. Electrodes Fp1 (p=0.040), F3 (p=0.013), Fp2 (p=0.033), T4 (p=0.050), T6 (p=0.018), O2 (p=0.011) and Oz (p=0.025) illustrate remark - able reductions. The beta absolute power cortical map also presents a significant power decrease in temporal region, specially, in the T6 electrode (p=0.022) after one night of sleep deprivation (*p≤0.05).

occipital areas of the alpha frequency band, in the following electrodes: Fp1 (p=0.040), F3 (p=0.013), Fp2 (p=0.033), T4 (p=0.050), T6 (p=0.018), O2 (p=0.011) and Oz (p=0.025). The beta frequency band also experienced power reduction in the temporal area (p= 0.022). Temporal delta (p=0.043) and temporal-occipital theta T6 (p=0.041), O2 (p=0.018) and OZ (p= 0.028), instead, exhibited power increases after SD. Cortical maps represented by Figures 1 and 2 represent the significant electrophysiological results found in the present study

DISCUSSION

The real implication of sleep is still not entirely clear and therefore the effects of sleep deprivation or sleep loss also cannot be fully understood yet. The present study demonstrated that one night of sleep deprivation caused specific electrophysiological changes, such as relative delta and theta power increase, and absolute alpha and beta power reduction. Our findings are in agreement with previous results[5,6,11,18,20] which observed the same alterations in the brain electrical activity. Electrophysiological alterations such as
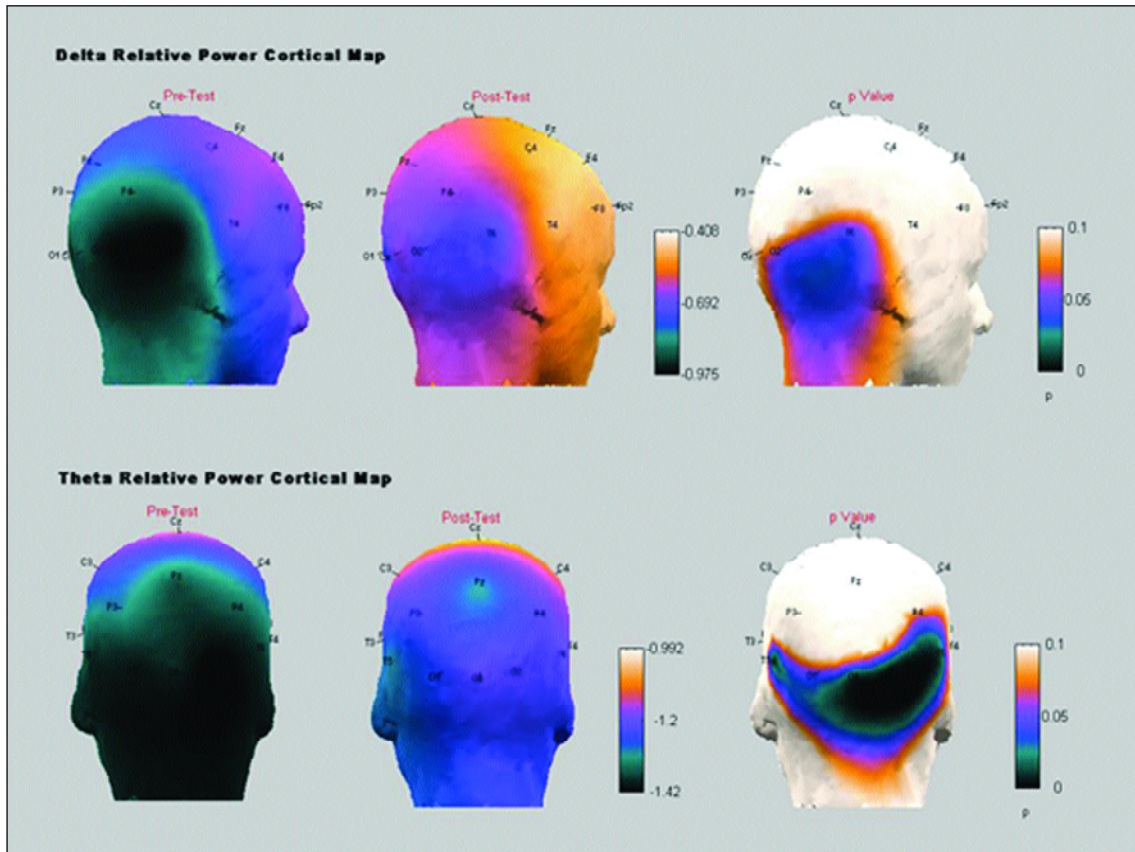
Fig 2. Theta relative power cortical map. The occipital view of this frequency band exhibits the power increase observed after one night of sleep deprivation in the following electrodes: T6 (p=0.041), O2 (p=0.018), and Oz (p= 0.028). An also significant power augmentation was observed in the delta band. The delta relative power cortical map, in particular electrode T6 (p=0.043) displays the power changes observed after one night of sleep deprivation (*p≤0.05).

an increase in theta relative power in occipital and temporal areas, and in delta relative power in temporal regions were expected since the subjects were feeling each time sleepier and less alert (no scales were used, subjects simply were frequently asked about their subjective feelings of sleepiness throughout the whole experiment). It is clear that the amount of theta becomes more pronounced as the period of continuous wakefulness pro g resses; despite the fact that cognitive workload (e.g., task demands) requirements did not change. Increased theta activity is found[21] to correlate with increased workload, as well as increased fatigue. However, the widespread augmentation[22] of theta activity has more traditionally been associated with impaired alertness. In addition, these generalized low-frequency EEG patterns have been associated[23] with decrements in vigilance tasks. Although changes in EEG theta activity can be interpreted as something other than an indication of an individual state of alertness (for example, a correlation has been observed between theta activity and

cognitive workload), experimental evidences have shown that theta is in fact a trustworthy indicator of great central nervous system inhibition associated[24] with low arousal and diminished information processing. Based on the present knowledge about sleep loss and sleep deprivation, physiological arousal and perf o rmance; both EEG and behavioral findings observed in the present study are coherent with the lite rature. In sleep-deprivation paradigms, it has been well established[22] that prolonged wakefulness is accompanied by systematic power increases in theta activity. Contextual stimulation is known to attenuate[25] the effects of sleep deprivation, and thus, the impact of sleepiness on alertness may have been reduced modestly preventing greater delta power increase (e.g., frank sleep episodes), while still not altogether eliminating its appearance in slow-wave EEG. This could help explain why delta power did not show a large increase all over the scalp, but instead, was limited to the temporal area (T6 electrode).

Alpha power during waking EEG is also of part i c-ular interest for the research on sleep deprivation, sleep loss and sleepiness. Throughout active wake-fulness (eyes open), alpha power is usually low, but if the individual is severely fatigued[26], it might increa-se. However, in resting conditions (eyes closed), alpha power is high also when the subject is fully rested. Several studies observed that during the transition f rom resting conditions with eyes closed to sleeping, a gradual reduction in alpha power occurs, all togeth-er with an increase in theta power[26-28]. So, if the sub-jects experience progressively higher sleepiness sen-sation as the hours of wakefulness proceed, the ob-served reduction in alpha power is expected. Con-sequently, reduced alpha power during waking peri-ods may indicate a high motivation for sleeping; indeed it has been already found[28] that subjective sleepiness during sleeplessness periods correlates neg-atively with alpha power and positively with theta power during the waking EEG after sleep depriva-tion. Also, the early stages of drowsiness, experienced h e re by the subjects after prolonged wakefulness, a re characterized by what it's called an "alpha pow-er dropout". The trains of alpha waves become less and less continuous and the last alpha fragments give place to a low voltage pattern of mixed slow fre-quencies (e.g., delta and theta). Alpha activity coun-teracts with theta activity; i.e., a slowing of posteri-or alpha is substituted gradually by the posterior the-ta activity (and delta), as the motivation to sleep in-c reases. These adjustments in the spectrum towards slower frequency bands were better observed here by the temporal activity changes (T6 electrode), since we found an alpha and beta power reduction and an accordingly a theta and delta power increase in this area. Since these changes are very common in the elderly[29] or in patients suffering from dementia, we can only speculate about the substantial correla-tion between these EEG abnormalities observed after sleep deprivation (spectrum shift to slower fre q u e n-cies) and a cognitive deficit.

Some investigators also observed that the alpha power reduction after sleep deprivation may be relat-ed to a reduction of activation in sub-cortical brain structures with general cortical activation properties, e.g., brain stem, midbrain, hypothalamus and other parts of the limbic system, since these structures show a positive correlation between regional cerebral blood flow and cortical alpha power[30] in resting con-ditions. Moreover, the positive correlation between beta absolute power increase (e.g., temporal region) and the hours of prolonged wakefulness might be interpreted as an index of eff o rt and temporary acti-

vation. A speculative interpretation may be that the observed beta power increase reflects processes underlying an increasing effort to maintain wakeful-ness, i.e., the motivation to stay awake, that coinci-des[16] with an increased feeling of sleepiness.

In conclusion, the present study demonstrated that one night of sleep deprivation was associated with: (1) increased resting EEG theta and delta rela-tive power with a specific occipital/temporal focus, (2) decreased resting EEG beta absolute power in the temporal area and (3) decreased resting EEG alpha absolute power centered in the frontal, temporal and occipital areas of the cortex. The electrophysiolo-gical adjustments of the brain after sleep depriva-tion or sleep loss are still not entirely clear, but it is highly plausible to be local, rather than global, pro-cesses. Future re s e a rch is still necessary to replicate these findings and maybe correlate qEEG changes with behavioral responses. The processes underlying these electroencephalographic changes are connect-ed to each other and might reflect the performance impairments that commonly accompany the loss or deprivation of sleep, leading us to our choice to go to sleep.

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