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Effects of Total Sleep Deprivation on the First Positive Lyapunov Exponent of the Waking EEG

Dai-Jin Kim*, Jaeseung Jeong**, Jeong-Ho Chae*,
Soo Yong Kim**, Hyo Jin Go*, In-Ho Paik*

Abstract Sleep deprivation may affect the brain functions such as cognition and, consequently, dynamics of the EEG. We examined the effects of sleep deprivation on chaoticity of the EEG. Five volunteers were sleep-deprived over a period of 24 hours. They were checked by EEG during two days, the first day of baseline period and the second day of total sleep deprivation period. EEGs were recorded from 16 channels for nonlinear analysis. We employed a method of minimum embedding dimension to calculate the first positive Lyapunov exponent. For limited noisy data, this algorithm was strikingly faster and more accurate than previous ones. Our results show that the sleep deprived volunteers had lower values of the first positive Lyapunov exponent at ten channels (Fp1, F4, F8, T4, T5, C3, C4, P3, P4, O1) compared with the values of baseline periods. These results suggested that sleep deprivation leads to decrease of chaotic activity in brain and impairment of the information processing in the brain. We suggested that nonlinear analysis of the EEG before and after sleep deprivation may offer fruitful perspectives for understanding the role of sleep and the effects of sleep deprivation on the brain function.

Keywords: Sleep deprivation, chaos, EEG, The first positive Lyapunov exponent

1. Introduction

The adverse consequences on cognitive performance of sleep deprivation have been well documented in recent reviews (Hart et al., 1987; Pilcher and Huffcutt, 1996; McCarthy ME and Waters WF, 1997). Increase in lapse frequency, slowing of response times, or other cognitive dysfunctions occurring in individuals working at night could lead to failure to perceive and respond to critical visual and auditory information correctly (Corsi-Cabrera et al., 1996).

Total sleep deprivation is known to cause various degrees of monotonic decrease in performance of a very broad range of variables including vigilance, reaction time, arithmetic computations, short-term and long-term memory, psychomotor tasks, and logical reasoning tasks. The longer the time period of previous wakefulness is, the greater the decrease in the performances is (Horne 1978; Corsi-Cabrera et al., 1996).

After a normal period of wakefulness (16

* Department of Psychiatry, College of Medicine, The Catholic University of Korea, Seoul
** Department of Physics, Korea Advanced Institute of Science and Technology, Taejon, Korea
Correspondence to : Jaeseung Jeong
Tel : (042) 869-2569, Fax : (042) 869-2510
E-mail : jsjeong@sensor.kaist.ac.kr

hours), EEG power is significantly increased and interhemispheric correlation is decreased. After a normal sleep of night, the values are recovered in the morning. Extended hours of wakefulness exacerbate the EEG changes. The changes in the waking EEG are dependent on the amount of previous sleep or wakefulness. These findings indicate that accumulating hours of wakefulness is reflected not only in the sleeping EEG, but also in the waking EEG (Lorenzo et al 1995; Corsi-Cabrera et al 1992). The absolute powers of the whole spectrum with open eyes and of theta and beta bands with closed eye increases after total sleep deprivation (Corsi-Cabrera et al 1996). According to Torsmall and Akerstedt (1987), delta and theta bands in the absolute power increase, as sleepiness is deeper for the train drivers. These studies, however, did not report apparently which region of electrical activity in brain contributes to cognitive dysfunction predominantly. There are many limitations on applying linear analysis to the investigation of the cognitive dysfunction after sleep deprivation because of the absence of an identified metric that quantifies complex behavior of the brain.

Recent progress in the theory of nonlinear dynamics has provided new methods for the study of time-series data from human brain activities. In the dynamical aspect, the brain is assumed to be a dissipative dynamical system. The distinct states of brain activity had different chaotic dynamics quantified by nonlinear invariant measures such as correlation dimensions and Lyapunov exponents (Babloyantz and Destexhe 1987; Babloyantz 1988; Röschke and Aldenhoff 1991; Fell et al 1993). Therefore, we can investigate the brain function by understanding the dynamical properties of the brain using nonlinear analysis of EEG.

Nonlinear analysis of the EEG to estimate the correlation dimension and/or the first positive Lyapunov exponent is very useful in making relative comparisons of different physiological states (Rapp 1993). Many investigations with these methods have revealed possible medical applications for nonlinear analysis and have given rise to the possibility that the underlying mechanisms of the brain function may be explained by nonlinear dynamics (Babloyantz and Destexhe 1986; Babloyantz and Destexhe 1987; Frank et al 1990; Pritchard et al 1991; Pritchard et al 1993; Pritchard et al 1994; Stam et al 1994; Besthorn et al 1995; Stam et al 1995; Stam et al 1996). Pathological conditions such as epileptic seizures, coma, and dementia showed decreased chaotic activities of EEG, whereas normal attentional states tended to increased chaotic activities by the estimation of the correlation dimension (Frank et al 1990; Prichard et al 1994; Stam et al 1995; Rapp et al 1989).

In this paper, we investigate the cognitive dysfunction after sleep deprivation using nonlinear analysis of EEG. We estimate the first Lyapunov exponents of the EEG and compare the values in the whole brain region before and after sleep deprivation. The changes of dynamical properties of EEG at different channels may give the fruitful key to understand the role of sleep and the effect of sleep deprivation on the brain function.

In Section 2, we explain the procedure for reconstructing brain dynamics from an EEG and for analyzing the EEG by nonlinear methods and algorithm for determining the proper embedding dimension and for compensating for both noise contamination and edge effects. The first Lyapunov exponent is also defined and discussed. Section 3 briefly presents the procedure for recording data. Section 4 shows the differences in the values of the first Lyapunov exponent before and after sleep deprivation. In section 5, we discuss our results in the dynamical and physiological view. Our conclusions are given in Section 6.

2. Theory and Algorithm

In nonlinear analysis, the brain may be considered as a dissipative dynamical system. A dynamical nth-order system is defined by a set of n first-order differential equations. The states of the system can be represented by points in an n-dimensional space, where the coordinates are simply the values of the state variables x1, x2, x3, ···, xn. The phase space is the set of all possible states that can be reached by the system. In dissipative systems, as time increases, the trajectories converge to a low-dimensional indecomposable subset called an attractor (Eckmann and Ruelle 1985).

In experiments, one cannot always measure all the components of the vector giving the state of the system. Therefore, we have to reconstruct brain dynamics from a one-dimensional EEG by using delay coordinates and the Takens' embedding theorem. Takens showed that an attractor, which is topologically equivalent to the original data set, can be reconstructed from a dynamical system of n variables x1, x2, x3, ···, xn by using the so-called delay coordinates $y(t)=[x_j(t),\ x_j(t+T),\cdots,\ x_j(t+(d-1)T)]$ from a single time series xj and by performing an embedding procedure, where d is the embedding dimension. The purpose of time-delay embedding is to unfold the projection back to a multivariate state space that is a representation of the original system (Takens 1981; Eckmann and Ruelle 1985).

Lyapunov exponents estimate the mean exponential divergence or convergence of nearby trajectories of the attractor. Lyapunov exponents are usually ordered in a descending fashion from L1 (the highest value) to Ln (the lowest value). A system possessing at least one positive Lyapunov exponent is chaotic. This fact reflects the sensitive dependence on the initial conditions (Fell et al 1993).

We applied a reconstruction procedure to each EEG segment. For the time delay T, we used the first local minimum of the average mutual information between the set of measurement $v(t)$ and $v(t+T)$ (Fraser and Swwiney, 1986).

We estimate the first positive Lyapunov exponent with minimum embedding dimension method. In classical algorithms, we calculate a nonlinear invariant measure by increasing the embedding dimension until the value of the invariant measure is saturated. It requires a very large number of computations. In our new algorithm, we calculate the first positive Lyapunov exponent L1 in the minimum embedding dimension.

We determine the minimum embedding dimension by using the calculation method, presented by Kennel et al. (1992), which is based on the idea that in the passage from dimension d to dimension d+1, one can differentiate between points on the orbit that are "true" neighbors and those on the orbit which are "false" neighbors. A false neighbor is a point in the data set that is a neighbor solely because we are viewing the orbit (the attractor) in too small an embedding space ($d<d_{\min}$). When we have achieved a large enough embedding space ($d\geq d_{\min}$), all neighbors of every orbit point in the multivariate phase space will be true neighbors. We demonstrated that for limited noisy data, our algorithm was strikingly faster and more accurate than previous ones (Jeong et al. 1997; Jeong et al., 1997).

We calculate the first positive Lyapunov exponent L1 by applying a modified version of the Wolf algorithm (Wolf et al., 1985) and by following a proposal by Frank et al. (1990). Essentially, the Wolf algorithm computes the initial vector distance di of two nearby points and evolves its length at a certain propagation time. If the vector length df between the two points becomes too large, a new reference point is chosen with properties minimizing the

replacement length and the orientation change. Now, the two points are evolved again and so on. After m propagation steps, the first positive Lyapunov exponent results from the sum over the logarithm of the ratios of the vector distances divided by the total evolving time:

$$L_1 = \frac{1}{m} \sum_{i=1}^{m} \frac{\ln \frac{df_i}{di_i}}{\mathrm{EVOLV} \cdot dt \cdot \ln 2} \quad (\mathrm{bits/sec}) \quad (1)$$

where dt, di, and df are the sampling interval, and the initial and the final separations between the points in the fiducial trajectory and in the nearest-neighbor trajectory separated in time by ith EVOLV step, respectively (Wolf et al., 1985).

By using the weight function proposed by Frank et al. (1990), we improve the L1 estimate by widening the search to allow replacements to be well-aligned points lying further apart but still within the region of linear dynamics:

$$W(r, \theta) = \left(\alpha + \beta \left(\frac{b-r}{b-a} \right)^{\gamma} \right) \cdot \cos\theta \quad (2)$$

where b and a are distances over which the dynamics is assumed to be linear and to be noise-dominated, respectively, r is the radial separation between the candidate and the evolved benchmarks, and θ is the angular separation between the evolved displacement and the candidate replacement vectors. The numeric parameters α, β, and γ control the relative importance of the proximity to the alignment priority.

As suggested by Principe and Lo (1991), we use the information contained in the power spectrum of the signal segment for the proper evolving time EVOLV. We select the 1/e spectral frequency - the frequency that divides the power spectrum in the ratio of 1/e - as the frequency to be used to obtain the number of points for the EVOLV step. Realistic values for the average additive noise levels can be extracted from the curves of the correlation integral function which are used to calculate the correlation dimension. Intermediate knees in the correlation integral are related with noise contamination. The value of r for which the knee starts to appear can be used as an estimate of the noise scale. The criteria to establish the maximum scale are derived from the upper boundary of the scaling region in the correlation function (Principe and Lo, 1991).

3. Methods

Five female volunteers, who are between 27 and 30 years old, participated in the experiment. Subjects were right-handed with neither central nervous system disorder nor use of medication known to affect sleep or EEG. They were free of sleep complaints and had normal sleep habits, as assessed by questionnaire. EEG was recorded during resting wakefulness with closed eyes under two conditions: 1) in the morning after deep sleep for baseline 2) in the morning (between 8:00 and 10:00 A.M.) after total sleep deprivation for one night.

The EEGs were recorded from the 16 scalp loci of the international 10-20 system. With the subjects in a relaxed state with closed eyes for 32.768 seconds of data were recorded and digitized by a 12-bit analog-digital converter in an IBM PC. Recordings were made under the eyes-closed condition in order to obtain as much stationary EEG data as possible. The sampling frequency was 500 Hz. Potentials from 16 channels (F7, T3, T5, Fp1, F3, C3, P3, O1, F8, T4, T6, Fp2, F4, C4, P4, and O2) against "linked earlobes" were amplified on a Nihon Kohden EEG-4421K using a time constant of 0.1 sec. All data were digitally filtered at 1-35 Hz in order to remove the

residual EMG activity. Each EEG record was checked by inspection to be free from electrooculographic and movement artifacts and to contain minimal electromyographic activity. Whenever a decrease in vigilance was detected on the ongoing EEG, the technician instructed the subject to open her eyes, and a short pause was allowed, if needed, to minimize drowsiness.

The data were analyzed by using SPSS (6.0 release version). Results of group data are expressed as mean ± standard deviation (SD). Group differences between before and after sleep deprivation states were evaluated by using Wilcoxon matched-pairs signed-ranks test. A two-tailed probability of less than 0.05 was considered to be statistically significant.

4. Results

The first step in our analysis was to construct phase space using the delay coordinates. We used the time delays calculated by the method of mutual information to reconstruct the attractor. Time delays of 26-46 ms and embedding dimensions of 11-23 were used for the subjects.

The L1 were calculated for all subjects in all channels. The proper evolving time (EVOLV) was selected by using 1/e spectral frequency and was about 220 ms. The calculation of the L1 naturally depends on the time over which a trajectory is evaluated. After about 200 propagation steps, the values converge at an interval of ±1.0% around the final value of the L1.

The average values of the L1 and the standard deviations for the subjects under two conditions: 1) in the morning after deep sleep 2) in the morning after sleep deprivation for one night are summarized in Table 1. Sleep-deprived states had lower average values of the L1 at ten channels, i.e., Fp1, F4, F8, T4, T5, C3, C4, P3, P4 and O1 compared with baseline values.

5. Discussion

We investigate the effects of sleep deprivation on brain function by estimating the change of dynamical properties of EEG between before and after sleep deprivation. Until now there are several studies on the EEG in sleep deprivation with linear methods. Corsi-Cabrera et al. (1992) reported that sleep led to higher interhemispheric correlation, lower intrahemispheric correlation and lower absolute power for all frequency bands as compared to values for presleep, while sleep deprivation led to opposite results, that is, lower interhemispheric correlation and higher intrahemisphreic correlation for all frequency bands and higher absolute power of the faster spectral bands. These results suggest that sleep increases the temporal coupling between both hemispheres and enhances local functional differentiation within each hemisphere, whereas sleep deprivation tends to produce a loss of interhemispheric coupling and a more homogeneous organization within each hemisphere. Cajochen et al. (1995) assessed EEG power density and self-rated fatigue with sleep-deprived subjects. They reported that power density in the frequency range of 6.25-9.0 Hz increased monotonically in the course of sustained wakefulness. This finding corroborates earlier reports of increased EEG power density in the theta/ alpha frequency range across prolonged periods of wakefulness. Barbato et al. (1995) reported that spontaneous blink rate increased and relative power of alpha EEG decreased following total sleep deprivation for one night. For their study, a possible working hypothesis is that increased blink rate after sleep deprivation could reflect an increased central dopamine activity. Lorenzo et al. (1995) have demonstrated that waking EEG activity is modified depending on the

amount of previous sleep or wakefulness. It is reported that sleep deprivation may lead to a deterioration in performance of the vigilance task and a linear increase of the power that was more prominent on the theta band (on central than temporal derivations and on the left than the right side with open eyes). Their study showed that sleep loss led to a generally linear decrease in alpha and an increase in theta wave and that performance errors were usually accompanied by a slowing of the EEG during sleep loss. However, they did not show clearly which region of electrical changes in the brain mainly contribute to cognitive dysfunction.

Our results indicated that the chaoticity of EEG decreased at several regions of the brain, that is, central, parietal, left prefrontal, left posterior temporal, left occipital, right frontal and right anterior frontal area. This means that the sleep-deprived brain processes informations deficiently and the neural networks are less flexible in these areas. These regions of the brain include the reticular activating system, thalamus, striatum, temporoparietal cortex and frontal cortex (Mesulam, 1981). These regions coincide with the key structure of attention and arousal. We suggested that these areas should be the key to the etiology of cognitive decline by sleep deprivation. Additionally, we also tested the cognitive function by evaluating a systematized neuropsychological test before and after sleep deprivation in this experiment. Therefore our suggestion will be substantiated by our future works. We will investigate the relation between the performance and the chaotic properties of EEG in all channels in the next paper. We expect that nonlinear analysis will give us a deeper understanding of role of sleep in ways which are not possible by conventional power spectral analysis.

6. Conclusion

In this study, our result demonstrated that total sleep deprivation for one night affects the dynamical properties of the brain. Our result shows that the sleep-deprived female volunteers had lower values of the first positive Lyapunov exponent at 10 channels compared with baseline values.

Our result is a preliminary finding, because the number of subjects is so small. Although our present study is in a fundamental stage of development, its clear result encourages further investigation of the chaoticity and complexity of the brain in sleep-deprived states. Especially, nonlinear measures of the electrophysiological activity in the brain may offer unique and fruitful perspectives for understanding important features of the role of sleep and the effects of sleep deprivation on the brain function.

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Location	after deep sleep (N=5)		after sleep deprivation (N=5)		z	*P*
	Mean	SD	Mean	SD		
F3	3.9180	0.901	2.4300	0.313	-0.753	NS
F4	4.1580	0.535	3.5980	0.357	-2.023	0.0433
F7	4.4940	0.565	3.7720	0.377	-1.826	NS
F8	4.5380	0.122	3.5760	0.332	-2.023	0.0433
Fp1	4.2360	0.406	2.2700	0.589	-2.230	0.0431
Fp2	4.0380	0.662	3.2880	0.244	-1.480	NS
T3	4.1240	0..323	3.4580	0.330	-1.826	NS
T4	4.6420	0.689	3.4920	0.268	-2.023	0.0431
T5	4.3000	0.293	3.2900	0.224	-2.023	0.0431
T6	4.3040	0.382	3.2960	0.381	-1.753	NS
C3	4.0540	0.365	2.1760	0.162	-2.023	0.0431
C4	4.5209	0.448	3.3180	0.120	-2.023	0.0431
P3	4.2940	0.392	2.4040	0.346	-2.023	0.0431
P4	4.5640	0.369	3.5640	0.182	-2.023	0.0431
O1	3.8580	0.289	2.4860	0.454	-2.023	0.0431
O2	3.9260	0.730	3.8160	0.055	-0.404	NS

NS : Not significant (Wilcoxon matched-pairs signed-ranks test)

Table. 1 The average values of the L1 and the standard deviations for the subjects under two conditions: 1) in the morning after deep sleep 2) in the morning after sleep deprivation for one night.

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(*The Catholic University, **Korea Advanced Institute of Science and Technology)

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