Effect of Total Sleep Deprivation on the Dimensional Complexity of the Waking EEG

Jaeseung Jeong PhD,[1] Dai-Jin Kim MD,[2] Soo Yong Kim PhD,[3] Jeong-Ho Chae MD PhD,[2] Hyo Jin Go MD PhD,[2] and Kwang-Soo Kim MD PhD[2]

[1]Department of Diagnostic Radiology and Child Study Center, School of Medicine, Yale University CT, 06520-8042, USA; [2]Department of Psychiatry, College of Medicine, The Catholic University of Korea, Seoul, South Korea; [3]Department of Physics, Korea Advanced Institute of Science and Technology,Taejon, 305-701 South Korea

Study Objectives: Sleep deprivation can affect the waking EEG that may reflect information processing of the brain. We examined the effect of total sleep deprivation (TSD) on nonlinear dynamics of the waking EEG.
Design: Paired-group design
Setting: A sleep disorders laboratory in a hospital.
Participants: Twenty healthy male volunteers
Interventions: Waking EEG data were recorded from subjects with eyes closed after (a) an 8-hour night's sleep and (b) TSD for 24 hours. The dimensional complexity (D2), as a nonlinear measure of complexity, of the EEG after a full night sleep were compared with those of the EEG after TSD.
Measurements and Results: The sleep-deprived states had lower D2 values at three channels (P4, O2, and C3) than normal states.
Conclusions: TSD results in the decrease of complexity in the brain, which may imply sub-optimal information processing of the cerebral cortex. We suggest that the investigation of the relation between nonlinear dynamics of the waking EEG induced by TSD and cognitive performance may offer fruitful clues for understanding the role of sleep and the effects of sleep deprivation on brain function.
Key words: Sleep deprivation; waking human EEG; nonlinear analysis; dimensional complexity

INTRODUCTION

SLEEP DEPRIVATION HAS BEEN A VALUABLE TOOL FOR THE STUDY OF SLEEP MECHANISMS, as well as an activator used in the clinic for detecting otherwise hidden epileptic activity. A lot of previous research on cognitive performance after sleep deprivation has been reported that total sleep deprivation (TSD) may cause monotonic decrease in performance of a broad range of variables including vigilance, reaction time, short-term and long-term memory, arithmetic computations, psychomotor tasks, and logical reasoning tasks.[1-5] Increase in lapse frequency, slowing of response times, and/or other cognitive dysfunction occurring in sleep-deprived subjects can lead to failure to perceive and respond to critical visual and auditory information correctly.[4,6]

The effects of TSD are reflected on the waking electroencephalogram (EEG) which is the activity of cortical neuron assemblies situated just below the surface of the scalp during wakefulness.[4,7-11] Corsi-Cabrera et al.[4] reported that absolute powers of the whole spectrum with eyes open, and of theta and beta bands with eyes closed, are higher after TSD and increase linearly with hours of wakefulness. Torsmall and Akerstedt[7] showed that delta and theta bands in absolute power of waking EEGs increase, as sleepiness is deeper. Lorenzo and his colleagues[11] measured the reaction time and the number of errors in a vigilance task. They observed that the increase in reaction time was correlated positively with hours of prior wakefulness and with the increase in theta absolute power during relaxed wakefulness. These findings can be interpreted that lack of sleep built up by extended hours of wakefulness may affect information processing of the brain, which modifies the waking EEG as well as the sleep EEG. It is, thus, important to investigate the relation between dynamical properties of waking EEG induced by sleep deprivation and brain function.

Recent progress in the theory of nonlinear dynamics and chaos has provided new methods for the time series analysis from human brain activities. In particular, nonlinear dynamical analysis of the EEG to estimate the dimensional complexity (D2) or the first positive Lyapunov exponent (L1) has proven to be very helpful in making relative comparisons of different physiological states, especially during sleep. Babloyantz et al.[12] first reported that EEG data obtained from the human brain showed deterministic chaos for sleep stages 2 and 4. Several studies have shown that nonlinear parameters like the D2 and L1 depended on the sleep stage; D2 and L1 decreased from the "awake" stage to sleep stage 1—4 and increased during rapid eye movement (REM) sleep.[12-19] Surrogate data analyses for detecting nonlinearity by comparing discriminating statistics of the original time series—such as D2 or L1—with those of its surrogate data which are a phase-randomized versions of the original time series found nonlinearity in the EEG during slow-wave sleep (SWS)[18,20] and REM.[20] Moreover, alterations in the nonlinear dynamical proper-

ties of sleep EEG in patients with depression and schizophrenia were found. Significant differences in D2 and L1 were detected during REM sleep in schizophrenia[21-23] and during SWS in depression,[23,24] indicating that altered nonlinear dynamics of the EEG during sleep may disturb information processing in depression and schizophrenia.

Most previous studies on examination of the waking EEG changes induced by sleep deprivation have applied conventional linear methods to the EEG analysis. The lone exception being the detection of low-dimensional dynamic self-organization in stage 4 sleep after partial sleep deprivation described in Cerf et al.[25] The aim of this study is to examine the effect of TSD on the waking EEG by using nonlinear methods. We used the D2 as an identified metric that estimates the degree of freedom of systems. We regarded the D2 as an operationally defined measure of complexity to quantify complex behavior of the brain instead of using it as an absolute measure to differentiate between periodic, chaotic or stochastic dynamics. We were also interested in the localization of the electrical activity in the brain affected by TSD by measuring the EEG from 16 channels after a normal sleep and TSD. The nonlinear dynamics of the EEG from 16 channels induced by TSD was compared with that after a full night's sleep.

In the materials and methods section, we explain the experimental procedures and the algorithm used to estimate the D2. The Results section shows the differences in the D2 values of the EEG from 16 channels before and after TSD. We discuss our results in the dynamical and physiological view in Discussion section.

METHODS

Subjects

Twenty normal male volunteers (23.4±1.9, mean±standard deviation) participated in the study and signed informed consent forms. In order to exclude the effect of hormone or sex, we only included male subjects. Subjects were right-handed and healthy. They took no medications and had no neurological or sleep-related disorders. All subjects had normal sleep habits including normal sleep duration and schedule, no daytime nap habits, no excessive daytime sleepiness, assessed by a one-week sleep log and by clinical interview and questionnaire. Subjects were selected if they usually went to sleep between 23:00h and 24:00h and if they usually slept eight hours per night (mean sleep duration: 476.5±14.5 min). We monitored their EEG twice at one-week intervals before experiments.

Procedure

In experiment, subjects slept in a sound-proof, temperature-controlled room. Their EEG data were measured at 07:00h just after an eight-hour night sleep. The EEG data were recorded with eyes closed to obtain as long stationary EEG data as possible. A 24-hour schedule of sleep deprivation began on morning awakening following the night. Subjects remained awake in the laboratory together with two experimenters throughout the whole course of the 24 hours. Every two hours, variations of alertness were assessed respectively by means of subjective ratings of sleepiness (the Stanford Sleepiness Scale). The EEG recordings were again taken with eyes closed at 07:00h next day.

EEG Recording

The EEGs were recorded from 16 scalp loci of the international 10-20 systems. With the subjects in a relaxed state with eyes closed, 32.768 sec of data (16,384 data points) were recorded at each monitoring stage and digitized by a 12 bit analog-digital converter in an IBM-PC. The sampling frequency was 500Hz. Potentials from 16 channels (Fp_1, F_3, F_7, T_3, T_5, C_3, P_3, O_1, Fp_2, F_4, F_8, T_4, T_6, C_4, P_4, O_2) against "linked earlobe" were amplified on a Nihon-Kohden EEG 4421K recorder using a time constant of 0.1 sec. The EEG data were digitally filtered at 1-35 Hz to remove the residual EMG activity. Each EEG records were judged by visual inspection to be free from movement and electro-oculographic artifacts, and to contain minimal electromyographic (EMG) activity. An experimenter checked the data collection to assure that the subject was awake. If there was any indication of a drop in alertness, we asked the subject to open his eyes and take a short break to improve his alertness before resuming data acquisition.

Nonlinear Dynamical Analysis

The time series of the EEG is not predictable over long time periods. This means that similar causes in the sense of similar EEG states do not produce similar effects. However, recent nonlinear dynamical theory has shown that unpredictability of a time series may not result from stochastic processes. Under selected conditions, nonlinear dynamical systems can generate so-called deterministic chaos. Such systems show a sensitive dependence on initial conditions, indicating that different states of a system, being arbitrarily close initially, will become exponentially separated in sufficiently long times.[26] The behavior of these deterministic chaotic systems is not predictable over long time periods. Nonlinear dynamical analysis assumes that the EEG may be generated from deterministic systems. In this sense, the unpredictability of the EEG might be a basic phenomenon of its deterministically chaotic characteristic.

In order to get better insight into the properties of dynamical systems, the nonlinear analysis is performed in phase-space. We transform a one-dimensional time series into a multi-dimensional phase space. In a hypothetical system governed by n variables, the phase space is n-dimensional. Each state of the system corresponds to a single point in the phase space whose n coordinates are the values assumed by the governing variables for this specific state. If the system is observed through time, the sequence of points in the phase space forms a dynamical trajectory. This trajectory fills a subspace of the phase space, which is called the system's *attractor*.

The reconstruction of the attractor in the phase space is performed through the technique of plotting delay coordinates. Let an observed time series x(t) be the output of a differentiable dynamical system f ton an m-dimensional manifold M. In order to unfold the projection back to a multivariate phase space that is a representation of the original system, we use the delay coordinates y(t) =[$x_j(t)$, $x_j(t+T)$,…, $x_j(t+(d-1)T)$] from a single time series $x_j(t)$ after performing an embedding procedure. y(t) is one point of the trajectory in the phase space at time t, x(t+iT) are the coordinates in the phase space corresponding to the time-delayed values of the time series, T is the time delay between the points
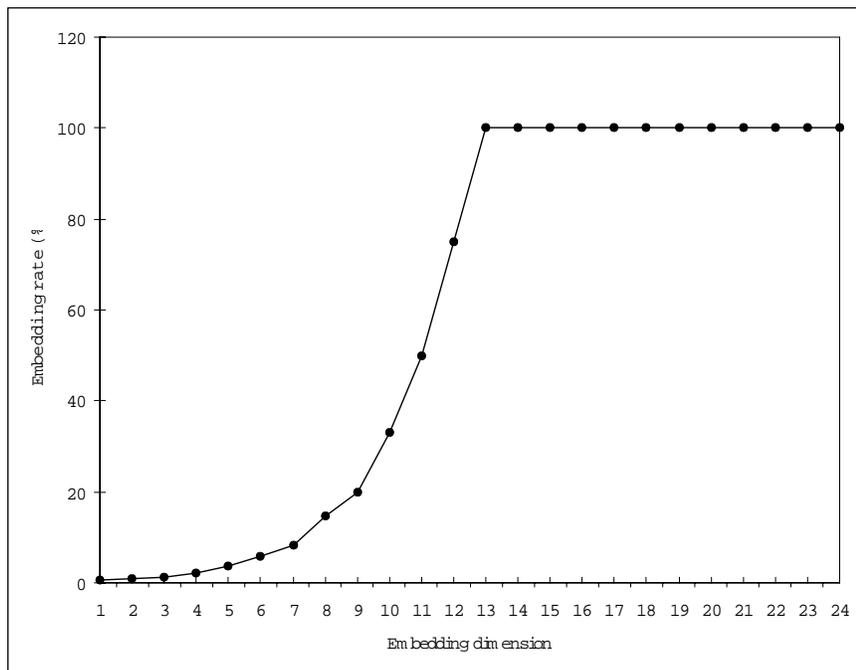
Figure 1—The embedding rate as a function of embedding dimension for 16,384 EEG data points at T6 in a subject. The optimal minimum embedding dimension for calculating the D2 was selected as 13 in this case.

of the time series considered, and *d* is the embedding dimension.[26] Takens showed that an attractor reconstructed by using delay coordinates from a single time series *xj* and by performing an embedding procedure is topologically equivalent to the original system.[27]

The choice of an appropriate time delay T and embedding dimension *d* are important for the success of reconstructing the attractor with finite data. For the time delay T, we used the first local minimum of the average mutual information between the set of measurement x(*t*) and x(*t*+T). Mutual information measures the linear and nonlinear dependence of two variables, in contrast to auto-correlation, which measures only linear dependence.[28]

We used the minimum (optimal) embedding dimension in the reconstruction procedure. The algorithm for estimating the minimum embedding dimension 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 that are false.[29] 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 ≥ d_{min}), all neighbors of every orbit point within the multivariate phase space will be true neighbors. The detailed algorithm is presented in the paper of Jeong et al.[30]

We defined the embedding rate as the ratio of true neighbors to neighbors in the embedding dimension. Figure 1 shows a typical example of the embedding rate as a function of the embedding dimension for 16,384 EEG data points in a subject. The proper minimum embedding dimension was selected as 13 in this case. We also showed the increase in the efficiency and accuracy of our method relative to the old one.[31]

One of the important mathematical quantities characterizing

an attractor is its dimensional complexity (D2), which is a metric property of the attractor that estimates the degree of freedom. It determines the number of independent variables that are necessary to describe the dynamics of the original system. For instance, in the case of steady-state behavior, the D2 of the attractor is zero and the D2 of the periodic attractor is one; in chaotic states, the D2 usually takes on noninteger values. The larger the D2 of the attractor, the more complicated the behavior of the nonlinear system. The D2 is thus a measure of complexity of the process being investigated and characterizes the distribution of points in the phase space.

We evaluate the D2 of the attractors from the EEG by using the Grassberger-Procaccia algorithm (GPA).[32] In this algorithm, the D2 is based on determining the relative number of pairs of points in the phase-space set that are separated by a distance less than *r*. It is computed from

$$D2 = \lim_{r \to 0} \lim_{N \to \infty} \frac{\log C(r,N)}{\log r}$$

where the correlation integral C(N, r) is defined by

$$C(r) = \frac{1}{N^2} \sum_{\substack{i,j=1 \\ i \neq j}}^{N} \theta(r - |\vec{x}_i - \vec{x}_j|)$$

where x_i and x_j are the points of the trajectory in the phase space, N is the number of data points in the phase space, the distance *r* is a radius around each reference point x_i and θ is the Heaviside function, defined as 0 if x<0, and 1 if x≥0. For small *r*, scaling property is exhibited: C(N, r) ∝ r^{D2}. For self-similar (fractal) attractor the local scaling exponent is constant, which region

Table 1—Means (standard deviations) of the D2 values from 16 channels before and after TSD

Channel	Before sleep deprivation	After sleep deprivation	T	p
F3	6.86±1.36	7.41 ± 1.59	-1.000	0.332
T4	7.59±1.46	8.03 ± 1.13	-0.749	0.464
F7	8.09±1.07	7.92 ± 1.56	0.429	0.674
C4	7.66 ± 1.48	7.63 ± 1.94	0.047	0.963
Fp1	8.31 ± 1.51	7.54 ± 1.53	1.684	0.112
F8	7.01 ± 1.51	7.48 ± 1.37	-1.157	0.264
T3	9.20 ± 1.50	9.43 ± 1.44	-0.305	0.764
*P4	6.39 ± 1.35	5.62 ± 1.29	2.177	0.045
T5	7.09 ± 1.41	6.70 ± 1.49	0.815	0.427
*O2	7.66 ± 1.66	6.29 ± 1.32	3.366	0.004
*C3	8.32 ± 1.47	7.07 ± 1.63	2.311	0.035
T6	7.67 ± 1.58	7.07 ± 1.52	1.115	0.281
P3	6.51 ± 1.24	6.89 ± 1.49	-0.670	0.513
Fp2	7.99 ± 1.51	8.40 ± 1.49	-0.794	0.439
O1	7.23 ± 1.24	6.50 ± 1.48	1.870	0.080
F4	6.57 ± 0.99	6.98 ± 1.53	-0.870	0.397

*: statistically significant (p<0.05, paired t-test)
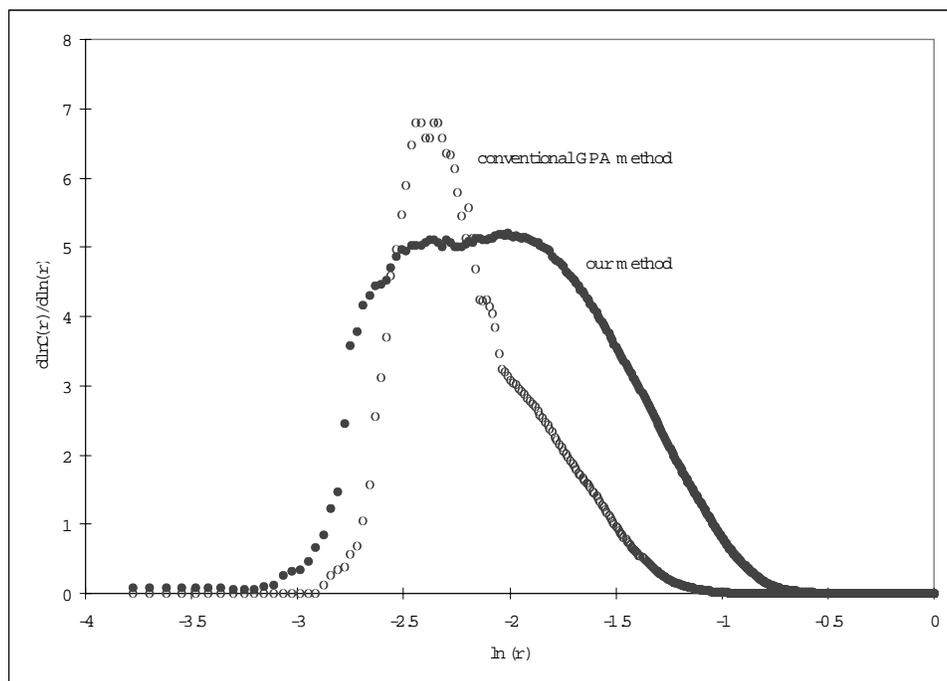

Figure 2—Comparison of the slope estimate at the minimum embedding dimension of 13 with the slightly modified method (·) for an EEG at T6 in the subject with a higher embedding dimension of 32 as obtained by the classical algorithm (o) for the same data. The slightly modified method produce a larger scaling region, indicating convincing scaling property in the EEG.

is called scaling region. If this plateau is convincing enough, this scaling exponent can be used as an estimate of the correlation dimension. One plot $C(N, r)$ vs. r on a log-log scale, and the correlation dimension is given by the slope of the log $C(r)$ vs. log r curve over a selected range of r. The slope of the correlation integral curve in the scaling region is estimated by a least-squares fitting method. A slightly modified version of the GPA was used to prevent over-contributing early terms from the start in the correlation integral and to compensate for the oscillation of the scaling region caused by the lacunarity of the attractor or finite sample oscillations caused by the limited amount of data.[33]

Statistical Analysis

Results of group data were expressed as mean±standard deviation (SD). Group differences between recordings taken after a normal sleep and TSD were statistically analyzed using paired t-test (SPSS version 6.0). A two-tailed probability of less than 0.05

was considered as significant.

RESULTS

The first step in our analysis was to reconstruct phase space using the delay coordinates. Time delays of 26—36 ms and embedding dimensions of 11—19 were used for the subjects. The slope of the correlation integral curve in the scaling region was estimated by a least-squares fitting method. The average D2 values from 16 channels and their standard deviations before and after TSD are summarized in Table 1. This demonstrates that sleep-deprived states had lower average D2 values at channel P_4, O_2, and C_3 than in normal states (p<0.05). The differences between the average values were about 0.6—2.1 units. This result indicates that TSD leads to the decrease of complexity of the EEG.

DISCUSSION

We estimated the D2 values of the waking EEG after eight-hour night sleep and 24-hour TSD to examine the effect of TSD on nonlinear activity of the waking EEG. It was shown that the waking EEG after TSD had lower D2 values at three channels (P_4, O_2, and C_3) than normal waking EEG. This result indicates that TSD lead to the decrease of complexity in the EEG thereby reflecting a change in information processing of the brain.

As previous studies showed, our result clearly demonstrated that the waking EEG reflects TSD. Several studies using linear methods reported that TSD resulted in a linear increase of power with eyes open, which was more prominent on the theta band, on central rather than on temporal derivations, and on the left rather than the right side. TSD also led to a decrease of alpha 1 absolute power with eyes closed.[8,10,11,34] In addition, Corsi-Cabrera et al.[8] reported that sleep deprivation led to 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 deprivation tends to produce a loss of interhemispheric coupling and a more homogeneous organization within each hemisphere. Although we cannot find any relation between linear and nonlinear properties of the waking EEG after TSD from this preliminary study, we can stress that nonlinear activity of the waking EEG is also modified by TSD.

The clear distinction of the nonlinear dynamics of the waking EEG before and after TSD based on dimensionality suggests a reduction of the degrees of freedom in conjunction with a change from the normal to the sleep-deprived state. A loss of complexity appears when biological systems become functionally impaired.[35] One of explanations for the reduction of the dimensionality is the inactivation of previously active networks or a loss of dynamical brain responsivity to the environmental stimuli.[14] Previous studies presented that reaction time in the vigilance task in sleep-deprived state was also longer than that in normal state.[7,11] Torsvall and Akerstedt reported that train drivers fail to respond to external signal during the night.[7] Thus, we can infer from these findings that decreased complexity of the waking EEG after TSD may be also associated with a loss of dynamical brain responsivity to the environmental stimuli.

Our result indicated that the complexity of EEG decreased at the left central, right parietal, and right occipital areas of the brain. This means that the neural networks are less sensitive in these areas, and the sleep-deprived brain processes information sub-optimally. In left central area (C3), Corsi-Cabrera group[4,11] also detected the absolute power changes in waking EEG after sleep deprivation. These regions of the brain include the reticular activating system, thalamus, striatum, temporoparietal cortex, and frontal cortex, which are the essential structures of attention and arousal. We suggest carefully that these areas may be related to the etiology of cognitive decline by sleep deprivation.

This result is, however, somewhat different from those of previous studies on sleep EEG after sleep loss. Cajochen et al.[36] demonstrated that the enhancement of slow wave EEG activity (delta and theta bands) during sleep following extended wakefulness was most pronounced in frontal cortical areas. Horne also reviewed that frontal cortical areas in particular are affected by sleep deprivation.[37] Recent support for this result may also be derived from the observation that rCBF in the anterior cingulate and orbitofrontal cortex as assessed by PET were negatively correlated with EEG delta activity during sleep.[38] This discrepancy may be from the difference between waking and sleep EEG. This warrants a more detailed analysis of the sleep and waking EEG after sleep deprivation to precisely localize the brain region affected by sleep deprivation. In addition, we propose that the relation between a deterioration in performance of the vigilance task and nonlinear dynamics of the waking EEG induced by sleep deprivation should be investigated to understand the role of sleep in information processing by the brain in ways which are not possible by conventional power spectral analysis.

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