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A Nonlinear Approach to Brain Function: Deterministic Chaos and Sleep EEG

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Summary: In order to perform a nonlinear dimensional analysis of the sleep electroencephalogram (EEG), we applied an algorithm proposed by Grassberger and Procaccia to calculate the correlation dimension D_2 of different sleep stages under Lorazepam medication versus placebo. This correlation dimension characterizes the dynamics of the sleep EEG and it estimates the degrees of freedom of the signal under study. We demonstrate that slow-wave sleep depicts a much smaller dimensionality than light or rapid eye movement (REM) sleep, and that Lorazepam does not alter the EEG's dimensionality except in stage II and REM. **Key Words:** Deterministic chaos—Dimensionality—Lorazepam.

The daily experience of the electroencephalographer dealing with sleep and its abnormalities has led to a classification of the spontaneous electrical activity of the central nervous system (CNS) during sleep in different stages (1). However, the differentiation between different sleep stages, i.e. between light sleep (stage I) and rapid eye movement (REM) sleep, cannot be exactly performed. One reason might be that the time history of the electroencephalogram (EEG) during a certain sleep stage is not predictable over a longer time period. Even if a typical EEG pattern is detectable, it is impossible to estimate the future behavior of the EEG. This means that similar causes in the sense of similar EEG states or similar EEG patterns do not produce similar effects. This is the reason why some authors considered the EEG to be a stochastic process (2–5), which, for instance, might be the expression of band-pass-filtered signals from hidden noise generators. In this sense the unpredictability of the sleep EEG is a basic feature of its statistical character.

To the contrary, in recent years it has become clear (6–9) that unpredictability does not damage the causality principle of natural philosophy. Under selected conditions, nonlinear dynamical systems that can be described by deterministic mathematical models are able to generate so-called deterministic chaos. Such chaotic systems show a sensitive dependence on initial conditions, which means that different states of a system that are arbitrarily close initially will become macroscopically separated after sufficiently long times (10–14). Regardless of a prescription of the system's dynamics in terms of differential equations, the behavior of such chaotic systems is not predictable over longer time periods (15). In this sense the unpredictability of the EEG is a basic phenomenon of its chaotic character.

The commonly used method for investigating the behavior of dynamical systems is to measure their attractors in phase space and to compute their correlation dimension D_2. This dimensionality estimates the complexity or the degrees of freedom of the investigated signal (16).

The nonlinear EEG analysis described in this paper exceeds the gain of information from conventional signal analysis. The dimension analysis is a description of the dynamical properties of a system and, as is shown in the present paper, time histories with apparently similar spectra do not necessarily have the same dimensionality.

The main aim of the present paper is to demonstrate that the EEG during different sleep stages is a deterministic process. The highly nonlinear character of the EEG should not be confused with noise properties. The application of this method to the sleep EEG has shown that different sleep stages correspond to significantly different correlation dimensions. The more the sleep moved toward slow-wave sleep, the lower the correlation dimension D_2.

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As an example, we will demonstrate how the theoretical aspects of deterministic chaos led to new approaches in pharmacopsychiatry. In normal healthy probands the influence of Lorazepam does not alter the dimensionality of slow-wave sleep, but it does alter stage II and REM sleep.

METHODS

The concept of attractors

A dynamical system is defined by a set of first-order differential equations. It is well known that the nonlinearity of these differential equations is a necessary (but not sufficient) condition for the generation of deterministic chaos (8).

In most cases it is impossible to specify the solution of such differential equations in a closed formula. In order to get better insight into the properties of dynamical systems, investigations of such systems are performed in the so-called phase space. Every instantaneous state of a system is represented by a single point in the phase space. The sequence of such states over the time scale defines a curve in the phase space, called a trajectory. As time increases, the trajectories either penetrate the entire phase space or they converge to a lower dimensional subset, called an attractor.

Different kinds of attractors are known. A closed curve, e.g. the phase-space representation of a sinusoid, is called a limit cycle. The sum of two sine waves with incommensurable frequencies results in a torus in phase space. Two arbitrarily close points of a limit cycle will continue to stay close as time increases. However, a subset of a phase space is called a strange attractor if initially arbitrarily close points get macroscopically separated after sufficiently large time intervals. In this case, similar causes do not produce similar effects. This important property is called sensitive dependence on initial conditions, which means unpredictability is a basic feature of strange attractors. On the contrary, the phase-space representation of noise never converges to a lower dimensional subset. Noise always penetrates the entire phase space. Therefore a stochastic process cannot define any attractor.

One of the most commonly used methods to describe and characterize an attractor is to compute its correlation dimension D_2 (17). The correlation dimension is introduced in information theory (18) and is a generalization of the Hausdorff (or fractal) dimension D_0. Moreover, it estimates the information dimension D_1 when $D_0 > D_1 > D_2$.

A necessary condition for the computation of dimensionality is to construct the phase space. For the analysis of experimental data, the proposal of Takens (19) is usually followed, which spans the phase space by the time-shift method. This means that an n-dimensional phase space was spanned by $x(t)$, $x(t + t_d)$, $\ldots$, $x(t + (n - 1)t_d)$. In our investigations we always used a time lag t_d, which was the first zero in the corresponding autocorrelation function and embedded the signals into phase spaces of up to 15 dimensions. For details of the theory see Röschke and Basa (20–22) and Röschke and Aldenhoff (23).

In order to estimate the correlation dimension D_2, one computes the correlation integral (17)

$$C(R) = \lim_{N\to\infty} \frac{1}{N^2} \sum_{i=1}^{N} \sum_{j=1}^{N} \theta(R - |\bar{x}_i - \bar{x}_j|) \qquad (i \neq j)$$

Θ: Heavyside function

Here, C(R) is a measure of the probability that two arbitrary points $\bar{x}_i$, $\bar{x}_j$ of the phase space will be separated by distance R. The main point is that C(R) behaves as a power of R for small R.

$$C(R) \propto R^{D_2}$$

Therefore plotting log C(R) versus log R allows us to calculate D_2 from the slopes of the curves. If the slopes of the graphs for increasing embedding dimensions converge to a saturation value, this limit is called the correlation dimension D_2. Because of the independence of the calculated dimensionality from the time lag t_d, one can prove at least that D_2 is not altered if another t_d is chosen (22).

For the analysis of an attractor of which the dimensionality was not previously known, it is necessary to calculate C(R) for several embedding dimensions. Assuming the dimensionality of an attractor is k, then the embedding theorem of Witney requires an embedding dimension of $2k + 1$ (14, 22, 24).

Specific properties of D_2

For a better understanding of the interpretation of the correlation dimension D_2, we will outline some features of D_2 based on theoretical data. If an attractor is a closed curve in phase space (limit cycle), its correlation dimension is $D_2 = 1.00$. A sine wave $x(t) = A \cdot \sin(2\pi f_1 t)$ is the simplest example of a limit cycle. Moreover, the sum of two sine waves with commensurable frequencies (i.e. $f_1 = 2$ Hz, $f_2 = 3$ Hz) defines a limit cycle too (Fig. 1a). To the contrary, the sum of two sine waves with incommensurable frequencies ($f_1 = 1.414$ Hz, $f_2 = 1.732$ Hz) results in a torus (Fig. 1b) with a dimensionality of $D_2 = 2.00$.

These examples demonstrate the advantage of the nonlinear dimension analysis versus conventional spectral analysis. Whereas the power spectrum of the sum of two sine waves (incommensurable frequencies

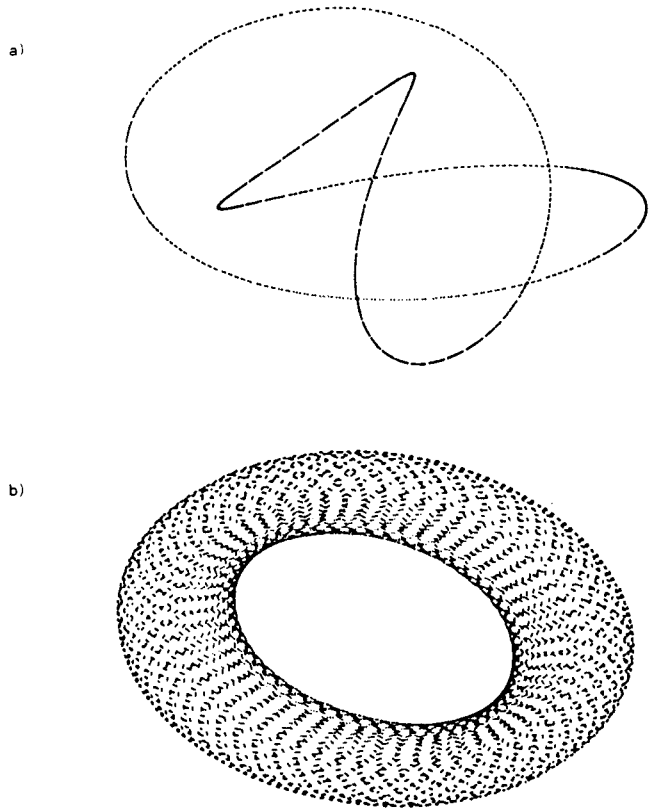

FIG. 1. **a.** The sum of two sine waves (f_1 = 2 Hz, f_2 = 3 Hz) results in a closed curve in phase space, called a limit cycle. **b.** The sum of two sine waves with incommensurable frequencies (f_1 = 1.414 Hz, f_2 = 1.732 Hz) results in a torus in phase space.

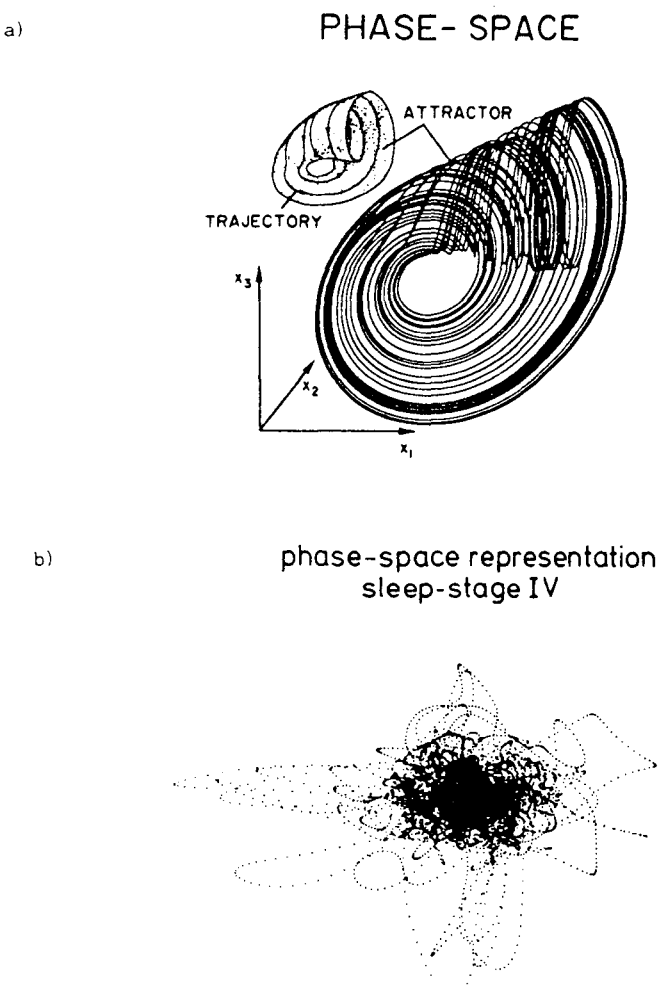

FIG. 2. **a.** An example of a strange attractor (Rössler attractor); modified after Abraham and Shaw (*Dynamics—Geometry of Behavior*, Santa Cruz, CA: Aerial Press, 1983). **b.** The two-dimensional phase-space representation of a human's sleep stage IV.

or not) remains in two different peaks in the frequency domain, the evaluation of the correlation dimension shows the differences. The calculation of D_2 from a time series consisting of a certain frequency and its harmonics gives a value of D_2 = 1.00. If the signal consists of two or three incommensurable frequencies, the correlation dimension is D_2 = 2.00 or D_2 = 3.00, respectively (24).

As mentioned above, the phase-space representation of a stochastic signal does not result in a convergence of the trajectory to an attractor. This means that the dimensionality of white noise is infinite. All strange attractors encountered up to now have a fractal dimension, which means that their dimensionality is a noninteger. Strange attractors can be identified with the properties of deterministic chaos. Figure 2 shows two strange attractors: the famous Rössler attractor, and the two-dimensional phase-space representation of an attractor of a human's sleep stage IV.

The main advantage of dimension analysis is the investigation of time series with apparently noiselike spectra. If the phase-space representation of such signals converges to an attractor, its dimensionality is a finite number. Otherwise, the investigated time series has stochastic properties and cannot be considered as a signal from a deterministic system.

An important question is whether the information one gains from the estimation of dimensionality is contained in the power spectra. In other words, is the dimensionality a parameter that increases knowledge about a system's dynamics? To answer this question we used data from a former investigation (24). We compared two time series, X_{GEA} and X_{HI}, the EEG of each measured with chronically implanted electrodes from the auditory cortex (GEA) and the hippocampus (HI) of a cat's brain during slow-wave sleep (21,24). The dimensionality of the cortex was D_2 = 5.63, and that of the hippocampus was D_2 = 4.58.

Following the proposal of Schroeder (25), we computed an autoregressive filter $A_{HI}(t)$, which estimated the spectrum of the signal X_{HI}. Therefore the convolution $Y_{GEA} = A_{HI}(t)*X_{GEA}$ converted the time series X_{GEA} into Y_{GEA}, having a spectrum resembling that of

TABLE 1. *Correlation dimensions (D_2) from lead position P_z and C_z from 12 healthy subjects without medication*

	P_z				C_z			
	II[a]	III	IV	REM	II	III	IV	REM
A	6.10	4.90	4.70	6.00	6.00	5.00	4.70	5.90
B	6.50	4.80	4.50	7.00	6.40	4.80	4.50	7.00
C	6.10	5.30	4.20	6.10	5.90	5.30	4.10	6.00
D	6.00	4.80	4.00	5.90	5.90	4.90	4.00	5.80
E	5.60	4.30	4.70	6.30	5.50	4.20	4.70	6.20
F	5.80	4.40	4.10	6.00	5.70	4.30	4.00	5.90
G	5.70	4.80	4.40	6.10	5.80	4.90	4.40	6.20
H	5.20	4.50	4.10	5.90	5.10	4.40	4.00	5.70
I	5.20	4.30	4.00	5.60	6.00	4.60	4.20	6.50
J	6.20	4.70	4.00	7.20	6.50	4.80	4.00	6.60
K	5.70	4.60	4.20	6.00	5.70	4.60	4.20	6.00
L	5.90	4.80	4.40	6.20	5.90	4.80	4.40	6.20
Mean value	5.83	4.68	4.28	6.19	5.87	4.72	4.27	6.17
SD	0.39	0.29	0.26	0.46	0.37	0.31	0.27	0.37

[a] Sleep stages II–IV and REM.

X_{HI} and vice versa (24). In other words, X_{HI} and Y_{GEA} as well as X_{GEA} and Y_{HI} had comparable power spectra. The main point was that regardless of the similar power spectra, the dimensionality of Y_{GEA} ($D_2 = 5.15$) differed from that of X_{HI} and also the dimensionality of Y_{HI} ($D_2 = 5.10$) differed from that of X_{GEA}. In other words, time series with comparable power spectra did not yield to attractors with the same dimensionality. Therefore measurement of the dimensionality is an additional tool for analyzing the dynamics of brain waves.

Experimental setup

We investigated 12 healthy male subjects, aged between 20 and 31 years (mean 24.6 ± 1.7 years). Subjects were volunteer recruits from the university student population and the general public. All were in self-reported good health with regular sleep–wake patterns. There was no evidence of hypnotic drug use or above-average alcohol or caffeine consumption. All were free of a past history or current symptoms of psychopathology as well as of any medical condition known to influence sleep.

We performed two experimental sessions: First, we recorded the sleep EEG under drug-free conditions (placebo), and second, we applied an oral dose of 2.5 mg Lorazepam at 10:30 p.m. The registration of the sleep EEG was started at 11:00 p.m. and was finished at 7:00 a.m. the next day. Surface electrodes were placed on the scalp (P_z, C_z, C_3 and C_4) and mastoid to record EEG activity, at the outer canthi to the left and right eye to record eye movements and on the chin to record submental electromyographic activity. Interelectrode impedances were all below 5 kohms.

For visual analysis according to Rechtschaffen and Kales, the sleep EEG was recorded using a Schwarzer E 12000 EEG machine. Additionally, the EEG data from P_z and C_z were digitized by a 12-bit analog-digital converter, sampled with a frequency of $f_s = 100$ Hz (50-Hz low-pass filter, 48 dB/oct.) and stored on the disk of a Hewlett Packard computer (A 900).

According to Rechtschaffen and Kales, the sleep EEG was scored by two independent judges. They determined four artifact-free time periods each of n = 16,384 data points (nearly 2:40 minutes) corresponding to sleep stages II, III, IV and REM. Within these time periods the sleep stages did not change. The choice of the artifact-free time intervals was arbitrary. The representative collections of slow-wave sleep and sleep stage II were from the first half, and the REM periods were from the second half of the all-night sleep EEG.

RESULTS

Normal probands without medication

The evaluation of the correlation dimension D_2 computed for all probands and from lead position P_z and C_z under drug-free conditions was shown in Table 1. For both electrode positions the mean dimensionality was highest during REM stage and lowest during stage IV. Significances of the calculated differences between the mean values were performed by applying the Student's *t* test. For the differences between all sleep stages, except the comparison of stage II and REM at lead position C_z, a value of $p < 0.05$ was obtained. The difference between stage II and stage REM at C_z was not statistically significant ($p < 0.1$). The comparison of the correlation dimensions between the two different lead positions resulted in nonsignificant differences. It should be mentioned that in some cases (nearly 15%) for the choice of the representative time intervals no clear convergence toward a saturation value and therefore toward a valid dimensionality could be observed. But in all cases in which an attractor exists, its correlation dimension was stable within small boundaries.

Table 1 clearly shows that the correlation dimension D_2 was highest during REM sleep and lowest during stage IV sleep. The differences between light (stage II) and slow-wave sleep (stage IV) was more than 1.5 units at both lead positions. The most impressive reduction of D_2 was observed at the transition from stage II to stage III, i.e. when slow-wave sleep arose. This transition was characterized by a decrease in the correlation dimension D_2 of 1.15 units at C_z and P_z. Between stages III and IV the differences were 0.45 units at C_z and 0.40 units at P_z, but nevertheless they were significant.

Normal probands with Lorazepam medication

The calculations of the correlation dimension D_2 from both electrode positions (P_z, C_z) and for all probands were summarized in Table 2, which also shows the mean values and the standard deviations (SD). Because of the marked sleep-inducing effect of Lorazepam, the probands did not show a sufficiently long time series of sleep stage I during the registration period from 11:00 p.m. to 7:00 a.m. Therefore we were not able to compute the dimensionality of stage I sleep under Lorazepam medication.

Again, under the influence of Lorazepam, $D_{REM} > D_{II} > D_{III} > D_{IV}$, and during all sleep stages there were no significant differences between the electrode positions P_z and C_z. Under the influence of Lorazepam a highly significant difference ($p < 0.001$) could be calculated between the dimensionalities of all sleep stages. Even the difference between stage II and REM at electrode position C_z (which was not sufficiently significant under drug-free conditions) was significant ($p < 0.001$). Again, the highest decrease of the dimensionality was observed at the transition from stage II to stage III.

The comparison of slow-wave sleep (stages III and IV) under drug-free conditions and under Lorazepam medication did not show any significant difference in dimensionality at both lead positions. However, there was a significant difference ($p < 0.01$) between the dimensionality of sleep stage II at P_z as well as at C_z. Under the influence of Lorazepam the correlation dimension D_2 was nearly half a unit smaller than under placebo conditions. For REM sleep, a significant difference ($p < 0.05$) in the dimensionality between placebo/verum could be calculated only at electrode position C_z. The dimensionality of REM sleep was lower (0.29 units) under the influence of Lorazepam. At lead position P_z this difference was not sufficiently significant ($p < 0.10$). Again, a convergence toward an attractor was not observed for the choice of the time epochs corresponding to defined sleep stages in all cases. Nevertheless, under the influence of Lorazepam a clear convergence toward a stable dimensionality could be observed in nearly 90% of all stages.

TABLE 2. *Correlation dimensions (D_2) and corresponding mean values from lead positions P_z and C_z under Lorazepam medication (2.5 mg)*

	P_z				C_z			
	II[a]	III	IV	REM	II	III	IV	REM
A	5.40	4.80	4.50	6.00	5.30	4.80	4.30	5.90
B	5.40	4.90	4.50	6.00	5.30	4.90	4.60	5.90
C	6.00	5.40	4.30	6.20	5.80	5.30	4.60	6.10
D	5.40	4.60	4.30	6.10	5.50	4.80	4.30	6.10
E	5.00	4.50	4.20	5.60	5.20	4.40	4.20	5.90
F	5.60	4.60	4.40	6.40	5.60	4.60	4.40	6.30
G	5.20	5.00	4.60	5.50	5.20	5.00	4.50	5.60
H	5.40	4.80	4.50	5.60	5.40	4.80	4.60	5.60
I	5.40	4.80	4.20	5.80	5.30	4.60	4.00	5.60
J	5.50	5.20	4.10	6.20	5.70	5.20	4.00	6.00
K	5.40	4.60	4.10	5.80	5.10	4.80	4.20	5.60
L	5.60	5.00	4.10	6.00	5.20	4.80	4.50	6.00
Mean value	5.44	4.85	4.32	5.93	5.38	4.83	4.35	5.88
SD	0.24	0.27	0.18	0.28	0.22	0.25	0.22	0.24

[a] Sleep stages II–IV and REM.

DISCUSSION

A few attempts to study the dimensionality of the EEG during wakefulness or sleep have been reported before. Babloyantz et al. (10), Rapp et al. (16,26,27) and Babloyantz (11) had already published some data about the chaotic dynamics of the brain's electrical activity. Our computations of the correlation dimension D_2 during sleep in humans confirm the exemplary results of Babloyantz et al. (10), who described for sleep stage IV a dimensionality of $D_2 = 4.37$ and for sleep stage II a correlation dimension of $D_2 = 5.03$. Graf and Elbert (28) investigated the waking EEG of a healthy subject and found a dimensionality of nearly 12 for the eyes-open condition. For the alpha-rhythm under eyes-closed conditions they computed a dimensionality of about 10. Meyer-Kress and Holzfuß (29) reported a fractal dimension of 8.7 for the waking EEG, which decreased to 5.1 after the subject kept his eyes closed and relaxed for a while.

Our computations indicate that in normal volunteers the dimensionality of the sleep EEG was much smaller than during wakefulness (28,29). Moreover, the dimensionality decreased if sleep became deeper. Under the influence of Lorazepam (2.5 mg oral application), the dimensionality during sleep stage II was reduced by nearly half a unit ($p < 0.01$). During REM sleep (at electrode position C_z), the dimensionality reduction was also significant ($p < 0.05$). During slow-wave sleep, the correlation dimension D_2 did not show any significant changes under Lorazepam medication. Taken together, these data pointed to the view that the EEG's dimensionality is a basic measure of the complexity of information processing by the brain. Complexity might be highest under intensive cognitive or emotional activity and medium in a relaxed state, i.e. during alpha activity under eyes-closed conditions. During sleep, the complexity decreased with increasing deepness of sleep from light to slow-wave sleep. The relatively high dimensionality of REM sleep might be explained by the increased information processing of the brain due to dreaming. The reduction of the REM dimensionality under the influence of Lorazepam is in accordance with the view that Lorazepam reduces dreaming. Moreover, Lorazepam not only decreases

the percentage of REM sleep but also alters the quality of REM sleep to lower complexity.

The question arises as to how specific the single number criterion D_2 characterizes the different sleep stages. The choice of the time periods, each a representative collection of a certain sleep stage, was due to the distribution of the sleep cycles during the night. The slow-wave sleep episodes, as well as those of sleep stage II, were acquired from the first half, whereas the acquisition of the REM episodes were from the second half of the night. In order to estimate the intraindividual consistency of the correlation dimension, we computed D_2 from different time periods of the first and the second half of the night, each an unambiguous collection of slow-wave sleep, sleep stage II and REM sleep data. We found out that in some cases no clear convergence of the slopes of the curves of log C(R) versus log R to a saturation value could be observed, which means that in some cases the EEG of a certain sleep stage did not show enough stable attractor property. Therefore, no valid correlation dimension for these time intervals could be computed. But in all cases in which the EEG unambiguously converged to an attractor, the dimensionality of this attractor was very stable within small boundaries (<5%). Consequently, if a clear attractor exists for a certain sleep stage, its dimensionality is a valid measure for the complexity of the EEG signal under study.

We considered as the main finding of this investigation that the EEG measured at different sleep stages did not show properties of stochastic signals. Usually the slow-wave sleep activity is called a state of hypersynchrony, whereas this synchrony cannot be observed during sleep stage II, and it is also impaired during REM sleep. This view is supported by the fact that the dimensionality during slow-wave sleep was minimal and that less synchronized stages correspond with higher dimensionality.

The evaluation of the dimensionality for the first time allows one to estimate a degree of synchrony from a single recording. According to the method described in this paper, a correlation or coherency analysis (always needing two different lead positions minimally) is no longer required. Moreover, the correlation dimension is a measure of synchrony and complexity and the degrees of freedom of a signal. It is not yet clear what the physiological correlate of the different degrees of freedom might be. In analogy to Basar (30–32) the higher dimensional dynamical properties of the CNS during sleep stages II and REM can be considered a result of weakly coupled oscillations of various neuronal networks with independent frequencies. Vice versa, the lower dimensional slow-wave sleep might be an expression of strongly coupled oscillations or the partial inactivation of previously active neuronal networks. In this sense the high synchrony (or low dimensionality) during slow-wave sleep might be a self-organizing effect that switches uncoordinated neuronal activity to coupled oscillations (31,32).

The computation of the correlation dimension or the evaluation of the degrees of freedom also estimates the lower boundary of the number of independent variables that are necessary to modulate the dynamics of the investigated system. In this sense, it is necessary that for any automatically performed analysis of the sleep EEG a set of five to eight independent variables should be investigated. Whereas slow-wave sleep should be characterized sufficiently by four or five variables (i.e. the alpha-, beta-, theta-activity or so), REM sleep or sleep stage II investigations require an extension of up to six or seven independent variables (i.e. the additional activity of higher frequency components). Any lower dimensional system would not be good enough to approximate the brain's electrical activity during sleep.

Further studies should answer the question of how the dimensionality and dynamics of the EEG may be changed under sleep disturbances, such as in depressive or psychotic disorder. Also the question of how pharmaceuticals with different psychotropic effects influence the dynamical properties of the CNS is an important one. Even considering the enormous computational expense, the evaluation of the correlation dimension seems to be a promising tool to gain further insight into brain function and the regulation of sleep.

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