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Estimation of the Dimensionality of Sleep-EEG Data in Schizophrenics

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Summary. Deterministic chaos could be regarded as a healthy flexibility of the human brain necessary for correct neuronal operations. Several investigations have demonstrated that in healthy subjects the dimensionality of REM sleep is much higher than that of slow wave sleep (SWS). We investigated the sleep-EEG of schizophrenic patients with methods from nonlinear system theory in order to estimate the dynamic properties of CNS. We hypothesized that schizophrenics would reveal alterations of their dynamic EEG features indicating impaired information processing.

In 11 schizophrenic patients, the EEG's dimensionality during sleep stages II and REM was reduced. We suggest that such lower dimensional chaotic processes might be associated with an overloading of neuronal networks during sleep and therefore the psychopathology of schizophrenics might be due to impaired complexity of their EEG's dynamics.

Key words: Sleep EEG – Deterministic Chaos – Dimensionality – Schizophrenia

Introduction

Neuropsychiatric research has to deal with the dilemma that the brain, the most complex biological system, seems to contradict the causality principle of natural philosophy. The basic assumption that similar causes do produce similar effects can hardly be proven for brain output under experimental conditions. The electroencephalogram (EEG) in particular has often been suspected to be a stochastical process rather than a meaningful signal. In recent years, the theory of nonlinear dynamic systems have provided some new methods to handle complex systems (Ehlers et al. 1991). Under selected conditions, nonlinear dynamic systems are able to generate so-called deterministic chaos. Such systems show a sensitive dependence on initial conditions which means that different states of a system, being arbitrarily close initially will become macroscopically separated after a sufficiently long time. Regardless of a description of a dynamic system in terms of differential equations, its behavior is not predictable over longer time periods. In this sense, the unpredictability of the EEG might be a basic phenomenon of its chaotic character. Using the tools of this approach the dynamics of a complex system can be reduced to a small number of coherent states or "attractors" and thereby it allows one to discriminate between stochastic and deterministic processes. The value of this method has been demonstrated successfully in different complex systems (Babloyantz 1991; Babloyantz et al. 1985; Destexhe et al., 1988; Pool 1989; Röschke and Aldenhoff 1991). With respect to the brain's electrical activity even this method is limited since the high level of complexity of brain function during wakefulness overrides the power of computer algorithms. However, during sleep various states can be characterized by significantly different dimensional "attractors" (Babloyantz et al. 1985). The methodical approach seems to be particularly suited for the investigation of schizophrenia which often coincides with sleep disturbances. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R) (APA 1987) schizophrenic disorder is characterized by several essential disturbances in form and content of thoughts, including features like delusions, hallucinations, loosening of associations, etc. In schizophrenia conventional sleep analysis revealed alterations during rapid-eye-movement sleep (REM) and Non-REM sleep, without demonstrating a specific disturbance pattern such as suggested by clinical experience (Keshavan et al. 1990). Polysomnographic abnormalities in schizophrenic patients are impaired sleep continuity (Kupfer et al. 1970), reduced total sleep time (Reich et al. 1975), reduced amounts of SWS (Caldwell and Domino 1967; Feinberg et al. 1969; Traub 1972), REM latency (Feinberg et al. 1965; Kupfer et al. 1970, Stern et al. 1969) and defective REM rebound (Gillin et al. 1974; Zarcone et al. 1975) following REM deprivation. However, several issues such as deficits in

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SWS or expectd abnormalities in REM sleep of schizophrenics remain controversial (Keshaven et al. 1990). Dreams, or other forms of mental activity, occur in all stages of sleep (Foulkes 1962). A particularly fascinating phenomenon is the striking similarity between dreaming and psychotic perception (Llinas and Paré 1991; Rifant 1979). Therefore, we performed dimension analysis during different sleep stages of unmedicated schizophrenic patients and compared it with those of normal controls.

Methods and Materials

We investigated 11 unmedicated acute schizophrenic inpatients (9 male, 2 female) who had not received any neuroleptic medication for the past 3 months (Table 1).

Patients were aged between 22 and 34 years (mean 28, SD 4) and were diagnosed according to DSM-III-R. Severity of illness was characterized by BRPS, SANS, SAPS, BRMS and GAS (for details see Table 1). The control group was balanced in sex, age, and education. 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 of current symptoms of psychopathology, as well as of any medical condition known to influence sleep. Subsequently to a night of adaptation to sleep laboratory conditions the sleep-EEG was registered from 11.00 PM until 7.00 AM next day. Surface electrodes were placed on the skull (P_z, C_z, C_3, C_4) and mastoid to record electroencephalographic activity, at the outer canthi on the left and right eye to record eye movements and on the chin to record submental electromyographic activity. Interelectrode impedances were all below five kOhms. Visual analysis of the sleep-EEG was performed according to Rechtschaffen und Kales (1968) by two independent judges. 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 (0.53 Hz high-pass filter; 50 Hz low-pass filter) and stored on the disk of a Hewlett Packard Computer (A-900). From the sleep-EEG four artifact-free time epochs ($n = 16384$ data points) were selected, each an unambiguous representative collection of one of the sleep stages

Table 1. Characterisation of the patient's group in terms of diagnosis according to DSM-III-R criteria and the score of different rating scales

Patient	Diagnosis (DSM-III-R)	Age/Sex	BRPS	SANS	SAPS	BRMS	GAS
A	295.11	23/m	46	93	0	14	35
B	295.32	34/m	52	81	47	26	30
C	295.31	27/f	54	43	34	19	25
D	295.34	28/m	50	73	45	11	40
E	295.34	28/m	47	70	44	14	45
F	295.31	33/m	47	62	47	25	25
G	295.30	26/m	40	42	35	21	28
H	295.10	25/m	43	62	48	28	35
I	295.31	30/m	48	13	25	12	45
J	295.10	33/f	40	18	45	24	30
K	295.30	22/m	42	79	17	12	35
Mean value		28	46	58	35	19	34
Standard deviation		4	5	26	15	6	7

II, III, IV and REM. According to the physiological sleep profile the representative collections of SWS were selected from the first, the REM periods from the second half of the all night sleep-EEG.

One of the commonly used attempts to investigate the behavior of dynamic systems is to measure their attractors in phase space and to compute their correlation dimension (Grassberger and Procaccia 1983) D_2 which is introduced in information theory (Renyi 1970) and is a probabilistic type of dimension. The dimensionality of a dynamical system is the number of state variables that are used to describe the dynamics of the system (Parker and Shua 1989). It is also related to the number of active coherent modes modulating a physical process and therefore it estimates the complexity and the degrees of freedom of the investigated system (Farmer et al. 1983). Moreover the correlation dimension D_2 estimates the information dimension D_1. It is the case that $D_0 \geq D_1 \geq D_2$ (Grassberger and Procaccia 1983). A necessary condition for the computation of dimensionality is to construct a phase space which is generally identified with a topological manifold. Following the time shift method proposed by Takens (1981) one of a possible phase space is spanned by $x(t), x(t+t_d), x(t+2t_d), \ldots$ Here $x(t)$ is the sample of the time series and t_d is a fixed time increment which in most cases was chosen equal to the first zero of the autocorrelation function (ACF) to $x(t)$ (Destexhe et al. 1988). We embedded the signals into phase spaces of up to 15 dimensions (Röschke and Aldenhoff, 1992). It should be emphasized that in cases were no zero crossing of the ACF was observable, we used the first local minimum of the ACF as time increment t_d. Every instantaneous state of the system is therefore represented by a single point $\bar{x}_k$ in phase space. The sequence of such states over the time scale defines a curve in the phase space, called trajectory. As time increases the trajectories either penetrate the entire phase space or they converge to a lower dimensional subset, an attractor. By computing the correlation integral C(R)

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

one can calculate the dimensionality by plotting log C(R) versus log R because C(R) behaves as a power of R for small R.

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

C(R) is a measure of the probability that two randomly chosen points $\bar{x}_i, \bar{y}_j$ of the phase space will be separated by distance R and θ is the Heaviside function ($\theta(z) = 1$ for $z > 0$ and $\theta(z) = 0$ for $z \leq 0$). In fact we did not calculate all possible distances $|\bar{x}_i - \bar{x}_j|$, but chose 820 (= 5% of 16384 data points) equally distributed points as reference points and calculated the differences between these reference points and all other points of the phase space representation. If the slopes of the graphs (log C(R) vs log R) for increasing embedding dimensions converge to a saturation value, this limit is called the correlation dimension D_2. According to Theiler (1986) we ensured that the correlation function counts only those pairs of phase space points which are not close in space only because they are close in time. Therefore we avoid an improvement of the convergence of the correlation algorithm. The computation of the slopes were performed by calculating the linear regression of five successive points and shifting this window over the log R axis. Because of the independence of the calculated dimensionality from the time lag t_d one has to prove at least that D_2 would not be altered if another t_d is chosen. Usually we chose $t_d = \{30\,\text{ms}, 40\,\text{ms}, 50\,\text{ms}\}$ for sleep stage I and REM-sleep, and $t_d = \{50\,\text{ms}, 60\,\text{ms}, 70\,\text{ms}\}$ for the other stages. It should be emphasized that in some cases no clear convergence of the slopes of the curves of log C(R) vs 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 dimensionality for these time intervals could be computed. But in all cases in which the EEG independent on the choice of t_d unambiguously converged to an attractor, the dimensionality of this attractor was very stable within small boundaries. Consequently, if an attractor exists for a certain sleep stage, its dimensionality is a valid measure for the complexity of the EEG signal under study.

Table 2. Mean values and standard deviation of conventional sleep EEG parameters of 11 unmedicated schizophrenics (patient) and the control group (control)

	TST	SOL	SEI	%REM	%I	%II	%SWS	REM-Lat.
Patient:	412.7	27.7	0.88	22.7	6.7	40	20.2	65.1
($n = 11$)	51.7	26.6	0.08	7.0	4.8	9.9	7.5	42.0
Control:	429.2	21.0	0.90	18.9	7.3	44.3	22.2	106.0
($n = 11$)	32.3	10.8	0.07	5.0	3.7	10.7	6.7	44.7

Table 3. Individual correlation dimensions from position P_z and C_z of 11 schizophrenic patients corresponding to sleep stages. (s.d. means standard deviation)

Patient	Position C_z				Position P_z			
	II	III	IV	REM	II	III	IV	REM
A	5.2	4.8	4.2	5.6	5.3	4.6	4.2	5.5
B	5.2	5.0	5.0	6.0	5.0	5.0	4.6	6.2
C	5.6	5.3	4.5	6.3	5.4	5.4	4.4	6.1
D	5.5	4.5	4.2	5.6	5.3	4.5	4.2	5.7
E	5.6	5.6	5.0	5.8	5.6	5.4	5.0	5.8
F	–	4.6	4.2	6.0	4.5	4.5	4.3	6.2
G	5.9	4.7	4.6	6.2	5.8	4.5	4.6	6.2
H	5.5	5.1	4.4	5.5	5.7	5.2	4.2	5.5
I	4.9	4.6	4.3	6.1	5.2	4.5	4.5	5.9
J	5.6	4.6	4.4	5.8	6.0	4.6	4.3	5.8
K	4.6	4.4	3.9	5.2	4.6	4.4	4.0	5.2
Mean	5.36	4.84	4.43	5.83	5.31	4.78	4.39	5.83
SD	0.39	0.37	0.33	0.33	0.47	0.39	0.27	0.31

Table 4. Correlation dimension D_2 calculated from lead position C_z for both schizophrenic and control group ($n = 11$; 16384 data points; SD = ± standard deviation)

	Sleep stage			
	II	III	IV	REM
Schizophrenia	5.36	4.84	4.43	5.83
SD	0.39	0.37	0.33	0.33
Control group	5.89	4.70	4.25	6.18
SD	0.38	0.30	0.29	0.36
Significance	$P < 0.01$	n.s.	n.s.	$P < 0.05$

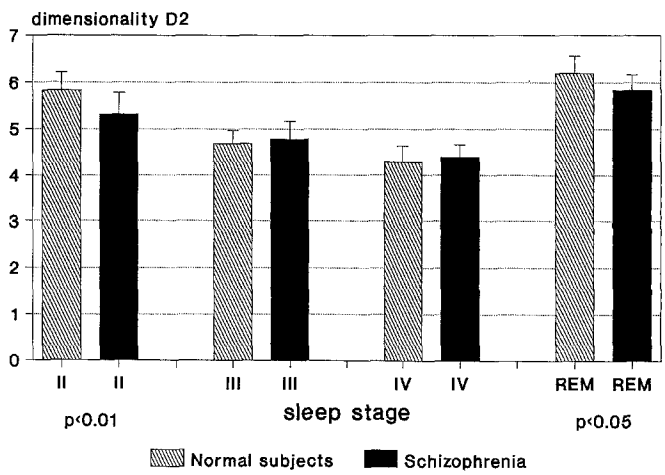

Fig. 1. Mean values and standard deviation of the correlation dimension D_2 during different sleep stages in psychosis and in the control group from lead position P_Z

Results

a) Conventional Sleep Architecture

Table 2 shows the results of the conventional sleep scoring procedure according to Rechtschaffen and Kales (1968) of the patients and the control group. Except for REM latency ($P < 0.05$, two-tailed t-test) no statistically significant differences concerning the parameters total sleep time (TST), sleep onset latency (SOL), and the percentage (%SPT) of sleep stages I, II, REM and SWS were detectable.

b) Dimensionality estimation

The results of our investigations are shown in Table 3 which for lead position C_Z and P_Z shows the individual values of the correlation dimensions D_2 corresponding to identical sleep stages. Because of too many movement artifacts in the sleep-EEG of nearly half of the schizophrenics during stage I, the patients did not show a sufficiently long time period of sleep stage I during the recording time. Therefore, we were not able to compute the dimensionality of sleep stage I in psychotic patients. For both schizophrenics and the control group the mean values of the correlation dimension from position C_Z are summarized in Table 4.

It should be emphasized that the evaluation of the dimensionality for lead position P_z shows the same differences (Fig. 1). For both positions the dimensionality of the sleep-EEG was highest during stage REM and lowest during stage IV. In general, the dimensionality decreased with the deepening of sleep. For control subjects and schizophrenic patients dimensionality differed significantly between sleep stages ($P < 0.005$; t-test; two-tailed). The direct comparison of different sleep stages between schizophrenics and control group reveals a slightly higher dimensional attractor during sleep stages III and IV in psychotic patients at electrode position P_Z as well as at C_Z. However, these differences did not reach statistical significance. On the contrary, there was a significant decrease of the correlation dimension during sleep stage II ($P < 0.01$, t-test, two-tailed) and REM-sleep ($P < 0.05$, t-test, two-tailed) in schizophrenics at position P_Z as well as at C_z.

Discussion

The pioneering work of Babloyantz et al. (1985) on the chaotic dynamics of the brain's electrical activity during sleep demonstrated that certain sleep stages can be characterized by dynamic properties resulting in different dimensionality. Lopes da Silva (1991) stated that it is clear that neuronal networks producing EEG signals are complex systems with nonlinear dynamics. He further mentioned that in cases when a rather high dimensional attractor is found, one has to be cautions with respect to the conclusion that the corresponding network can be described as a chaotic attractor. In these cases, it might be difficult to distinguish an apparently high dimensional attractor from filtered noise. Pijn et al. (1991) constructed a control signal having the same power spectrum as the original data set by replacing the phase angle of each frequency component by a random number. They concluded that a signal can only be distinguished from random noise if the corresponding dimensionality is clearly lower than that of the control signal. For data sets of $N = 4096$ samples they draw the conclusion that for certain conditions of an epileptic seizure a chaotic attractor of low dimension with D_2 in the range of $2 \leq D_2 \leq 4$ was present. On the other hand it has been shown that a filtered EEG rhythm does not have the same dynamics as a filtered white noise with the same spectrum (Soong and Stuart, 1989). Following a proposal of Schroeder (1984) we computed an autoregressive filter $A_{EEG}(t)$, which estimated the spectrum of an EEG signal (X_{EEG}) of 16384 data points. X_{wn} was the time series of a white noise. Therefore, the convolution $Y_{EEG} = A_{EEG}(t) * X_{wn}$ converted the white noise into Y_{EEG}, having a spectrum resembling that of X_{EEG}. The main point was that regardless of similar power spectra we could not observe the same dimensionality of X_{EEG} and Y_{EEG}. The control signal Y_{EEG} revealed higher values of D_2 or did not lead to any convergence of the curves (log C(R) vs log R). Therefore, we concluded that the electrical activity during sleep should not be mistaken for noise-like or stochastical processes. Moreover, the existence of finite dimensional attractors is a striking argument that the unpredictability of the EEG might be a consequence of its chaotic but nevertheless deterministic character (Röschke 1992). Exemplarily for a normal healthy subject Babloyantz described for sleep stage IV a dimensionality of $D_2 = 4.37$ and for sleep stage II a dimensionality of $D_2 = 5.03$. For the EEG of Creutzfeld-Jacob disease Babloyantz (1991) calculated a much lower dimensionality of $D_2 = 3.8$ and for a "petit mal" of epileptic seizures $D_2 = 2.05$ was calculated (Destexhe et al. 1988).

Our computations, evaluated on a group of 11 healthy subjects, confirm the primary results of Babloyantz et al. (1985). We could demonstrate that the deeper the sleep, the lower the correlation dimension varying in healthy subjects between $D_2 = 4.25$ (stage IV) and $D_2 = 5.89$ (stage II). In schizophrenia the dimensionality during sleep stages II and REM were statistically significant decreased, slow wave sleep depicted a slight, statistically nonsignificant increase of the EEG's dimensionality.

In 1989 Pool published an article dealing with the nonlinear dynamic approach to the understanding of various body functions and discussed whether "it is healthy to be chaotic". The most interesting argument was that the appearance of deterministic chaos may provide a necessary flexibility to the healthy brain and other systems of the body to operate in a physiological way. Conversely, he concluded, mainly pathophysiological mechanisms, e.g. mental disorders, may be associated with a loss of this chaotic flexibility. Freeman and Skarda (1985) have argued that deterministic chaos may serve the brain as a ready state keeping it primed to accept new inputs. The normal neuronal background activity of the brain, he states, is chaotic and mental activity takes places as an imposition of patterns on this chaotic brackground.

To our knowledge no data about the dimensionality or complexity of the sleep EEG in schizophrenic disorder have been published up to now. Koukkou et al. (1992) reported about a higher correlation dimension in the awake EEG of acute schizophrenics compared to healthy controls. Dieckmann et al. (1985) reported a higher entropy or, in other words, less order in the brain activity of schizophrenic patients during wakefulness, which in their opinion may be due to the diminished ability of schizophrenics to maintain attention and to their unsteadiness of will.

According to a provocative article of Crick and Mitchison (1983), without any experimental evidence up to now, the function of dream sleep is to enable the brain to erase spurious associations on a regular basis. The authors proposed that the function of dream sleep is to remove certain undesirable modes of interaction in networks of interconnected cells in the cerebral cortex. As mentioned above no experimental evidence for the Crick-Mitchison theory has been demonstrated up to now. We argue that the EEG's relatively high dimensionality during sleep stages II and REM and the relatively low dimensionality during deep sleep support the Crick-Mitchison theory about the function of dream sleep. The dynamic EEG attributes in terms of dimensionality clearly demonstrate a higher complexity and therefore a higher level of information processing (Gallez and Babloyantz 1991) of the brain during EEG episodes known to be highly correlated with dreaming. Interestingly the dimensionality of sleep stages III and IV is not significantly altered in psychotic patients. Although a reduced amount of slow wave sleep is discussed controversial in schizophrenia (for review see Keshavan et al. 1990), the quality of SWS seems to be the same in psychosis and normal healthy subjects. In our opinion a disturbance of the dynamic process to store and to erase associations during sleep probably might be associated with schizophrenic's thought disorders. This association might be based on the dynamic behavior of neuronal networks. A major difficulty of neuronal networks is that they become overloaded if an attempt is made to store simultaneously too many different patterns or associations of patterns. Overloading will result in unpredictable phenomena (parasitic states) like bizarre associations (fantasy), generations of always the same output, whatever the input is (obsession) or response to usually inappropriate inputs (hallucinations).

In order to avoid network overloading (and therefore to avoid schizophrenic symptoms) Crick and Mitchison postulated a mechanism which tunes the cortical system in the sense of removing parasitic states, a process they called "reverse learning" and which might be associated with dream sleep. This process may serve an essential function: it allows the orderly processing of memory and helps to reduce fantasy and obession. One of the most attractive aspects of the Crick-Mitchison theory is its heuristic resort to neural net behavior in computerized brain simulations (Hobson 1990). Newman and Evans (1965) described human dream processes as analogous to computer program clearance. They proposed that the dream process might be likened in function to the systematic program clearance which is absolutely necessary where computer programs are being continously evolved to meet changing circumstances. In their view, the primary function of sleep is probably to allow such a clearing process to get under way without interference from external information.

Callaway (1970) argued that schizophrenic's cognitive disturbances resulted from the interference of extraneous information on central processing. Such misinformation might be caused by the schizophrenic's impairment of reverse learning mechanism proposed by Crick and Mitchison. Callaway and Naghdi (1982) suggested a model which characterized the basic disorders of information processing in schizophrenia. The authors were concerned with information processing by persons whose associations were disordered, whose ideas and perceptions seemed to develop without adequate cross-checking. They postulated two sorts of interactive information processing: one is made up of automatic parallel processes and remains normal in schizophrenia, whereas the other uses controlled serial processes and is deficient. In this sense the schizophrenic symptom of delusions become more understandable in terms of underlying cognitive processes (Hoffmann 1987). A delusion is not a simple false belief; all of us fall a prey to false beliefs at times because of misinformation. A delusion is a false belief that is inescapable even in the face of the most overwhelming evidence to the contrary. The inescapability of delusions can be understood as a kind of parasitic state that expresses itself as a belief or inference. One possibility for the germination of such parasitic states may be caused in the impaired ability of the schizophrenic brain to erase spurious associations during dream sleep.

Assuming Freeman is right and that deterministic chaos might be a healthy flexibility of the human brain necessary for correct neuronal operations (Skarda and Freeman 1987), the decreased dimensionality of the EEG during certain sleep stages in schizophrenia reflects a reduction of the degrees of freedom, reduced complexity of neuronal activity and therefore impaired information processing (Gallez and Babloyantz 1991) in psychosis. This reduction in the degrees of freedom of the chaotic EEG of sleep stages II and REM in psychosis might be associated with the impaired functional mechanism proposed by Crick and Mitchison. Our results suggest an alteration of the CNS's workload during sleep which may injure the brain's ability of correct information processing. Further investigations, i.e. the calculation of the first positive Lyapunov-exponent, should yield a useful extension of the characterisation of EEG attributes in terms of nonlinear system theory. This number estimates the mean exponential expansion or contraction of a flow in phase space, expressing the sensitive dependence on initial conditions.

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