Dynamics of human sleep EEG

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Abstract

Several investigators of EEG time series reported a rejection of the null hypothesis of linear stochastic dynamics for epochs longer than 10 s. We examine whether this rejection is related to nonlinearity or to nonstationarity. Our approach is a combination of autoregressive modeling and surrogate data testing. It is shown that the fraction of subsegments, for which the null hypothesis has to be rejected increases with the length of the subsegments and can be related to fluctuations of the AR-coefficients on time scales in the range from 2–30 s.

Key words: sleep EEG, nonlinearity, nonstationarity, AR-model, surrogate data

1 Introduction

During the last decades many attempts were made to apply methods from nonlinear dynamics to analyze EEG time series. It turned out, however, that the assumption that the EEG can be described by a deterministic chaotic system do not hold in almost all cases. Today most researchers describe the EEG as a stochastic process. It is, however, still an open question to which extent one has to consider nonlinear effects.
If the EEG is given as a time series $x(t), x(t+\Delta t), \ldots, x(t+N\Delta t)$, a very general ansatz for a discrete time model of order m is given by

$$x(t+\Delta t) = f\left(x(t), x(t-\Delta t), \ldots, x(t-m\Delta t), \vec{p}, \vec{\xi}\right) \ , \tag{1}$$

where $\vec{p}$ is a parameter vector, and $\vec{\xi}$ represents a normal-distributed white noise term. The system is considered as nonstationary, if the parameters depend on time and as nonlinear, if the function f is nonlinear. Note that this distinction is not unique. In certain cases it might be possible to transform

a linear model with time dependent parameters into a stationary nonlinear model by increasing the model order m.
For time series of human sleep EEG, the surrogate data analysis revealed evidences for a rejection of the null hypothesis of a linear stochastic process (see e.g. [2,4,6]). These analyzes were performed on segments with a length larger than 10s (16 s in [4] and 20.5, 41, 82 and 164 s in [2]). Because the null hypothesis includes also stationarity, its rejection can be both due to nonlinearity and nonstationarity and the test cannot distinguish between these two possibilities.
We study the relation between nonlinearity and nonstationarity by modeling the dynamics on short time scales by linear autoregressive (AR-) models. It was shown in [3] that autoregressive modeling of short segments of a duration of 1 s can catch essential features of human sleep EEG.
The possible influence of nonstationarity is investigated by studying how the rejection rate of the null hypothesis depends on the length of the subsegments.

2 Data and Methods

The sleep EEG (C_3-A_2-derivation) of 4 healthy male subjects were recorded during 8 h in the sleep laboratory. Data were stored on a PC with a resolution of 128 Hz. For details of the recording and signal conditioning see [6]. Sleep stages were scored visually according to the criteria of Rechtschaffen and Kales [5]. 361 segments with a duration of 1-min (7680 points) were selected after visual inspection for lack of obvious nonstationarity due to artefacts and arousals. Only 1-min segments containing a definite sleep stage (REM sleep, stage 2 or slow-wave sleep (SWS, stage 3 and 4)) were considered. The segments from sleep stage 2 were divided in two classes: Class 2A consists of segments with sharp waves crossing the $-100\mu V$ threshold, which are mostly K-complexes, while the second class, 2B, comprises segments with small or no sharp waves.
For the further analysis the segments were divided into subsegments with length ranging from $T = 1 - 30$ s and AR-models were estimated using Burgs algorithm:

$$x(t + \Delta t) = \sum_{i=1}^{m} a_i x\left(t - (i-1)\Delta t\right) + \sigma_r \xi \ . \tag{2}$$

The parameter vector $\vec{p}$ is spanned by the AR-coefficients a_i and the standard deviation of the residuals σ_r. The fitted AR-model represents the null hypothesis for the corresponding subsegment. To avoid over-fitting on the short segments the model order was estimated using Akaike's information criterion (AIC) [1]. However, to compare the fluctuations of the AR-coefficients (Fig. 3)

the order of the model was fixed to six.
For every subsegment 100 surrogate data segments were produced by iterating the fitted model with randomly shuffled residuals. The model was tested using the correlation entropy based on the correlation sum

$$C(r) = \frac{2}{N \cdot (N-1)} \sum_{i=1}^{N} \sum_{j=i+1}^{N} \Theta(r - ||\vec{X}_i - \vec{X}_j||) \; .$$

$\vec{X}_i = (x(i), x(i-\tau), \ldots, x(i-(m-1)\tau))$ are the state vectors and $||\cdots||$ denotes the maximum norm. The delay time τ was chosen as $\tau = 3\Delta t$ with Δt the sampling time. The embedding dimension was fixed to $m = 3$. The length scale r was set to $r = 0.5\sigma_x$, whereas σ_x is the standard deviation of the samples of the corresponding subsegment. There is a tradeoff between the embedding dimension and the length scale due to the finite segment length.
The correlation entropy is defined by

$$H = -\ln C(r) \; .$$

It characterizes the marginal distribution of the $\vec{X}_i$.
The correlation entropy of the original data set was compared to the entropy of the surrogate data. If the entropy of the original data set was larger or smaller than the entropy of all of the 100 surrogate data sets the null hypothesis had to be rejected with a probability $p < 0.02$ for false rejection.

3 Results

Firstly we tested subsegments of length $T = 1s$. The fraction of original segments for which the null hypothesis was rejected is smaller than 2% in all four cases (table 1) and therefore smaller than the false rejection rate due to statistical fluctuations. Therefore we found no significant nonlinearity on this time scale. Interestingly neither sharp waves nor sleep spindles or slow waves are causing a significant rejection of the null hypothesis. However, their occurrence can be detected in the coefficients of the autoregressive models of the corresponding subsegments.

If the length of the subsegments is enlarged, the fraction of rejected segments increases. The first reason for this increase is that the distribution of the values of H_{surr} becomes more narrow. Its standard deviation σ_{surr} decreases approximately proportional to $T^{-1/2}$. This phenomenon can be observed also for surrogate data tests in stationary nonlinear systems. A second possible reason for this increase is that the expectation value of the difference $\Delta H = \langle H_{surr} \rangle - H_{orig}$ itself becomes larger. This can be interpreted as an

segment length T [s]	REMS	Stage 2A	Stage 2B	SWS
1	0.35%	0.52%	0.43%	0.22%
2	0.58%	2.79%	0.80%	0.41%
4	0.82%	10.08 %	2.78%	1.15%
10	3.8%	16.12%	13.37%	4.37%
30	8.77%	88.95%	49.48%	18.85%
60	19.3%	86.05%	67.71%	44.26%

Table 1
Fraction of subsegments for which the null hypothesis is rejected ($p < 0.01$).

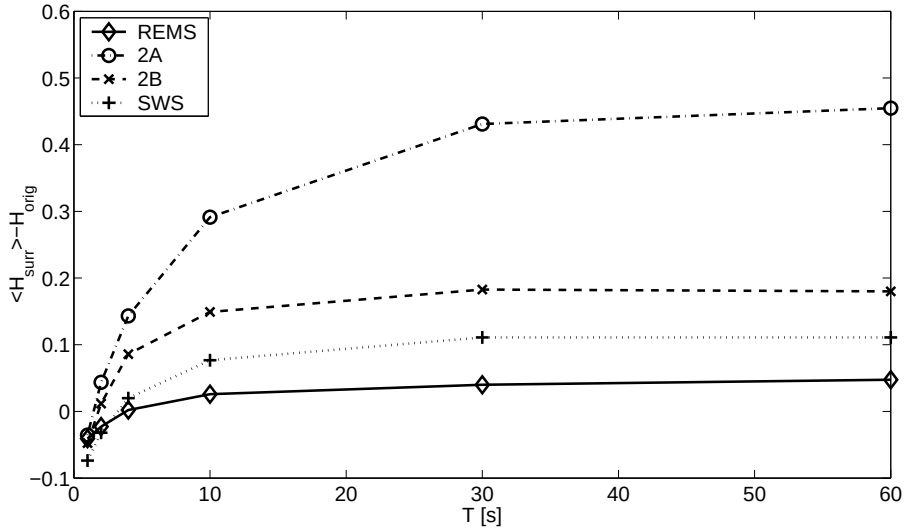

Fig. 1. Expectation values of $\langle H_{surr} \rangle - H_{orig}$ for different segment lengths.

effect of nonstationarity, because for a sufficiently large sample from a stationary system this difference is independent of the length of the segment. Fig. 1 shows the averaged behavior of these differences for the four groups depending on the length of the subsegments.

It is clearly seen that the largest differences ΔH can be observed for the two classes from sleep stage 2, which might be related to the nonstationarity introduced by the occurrence of K-complexes and sleep spindles. However, also in slow wave sleep and REM sleep significant nonstationarity is detected.

Fig. 2 shows the time course of ΔH over the night for one of the four subjects estimated from non-overlapping 60-s segments. REM sleep and SWS are characterized by values of ΔH which lie almost inside the interval spanned by the surrogates.

Because we could not the reject the null hypothesis for 1-s subsegments, AR-models (2) are a sufficiently good description for these subsegments. The nonstationarity concluded from the surrogate data analysis should be reflected in the fluctuations of its parameters. Fig. 3 shows the ratio between significant part of this fluctuations and the statistical fluctuations for the coeffi-

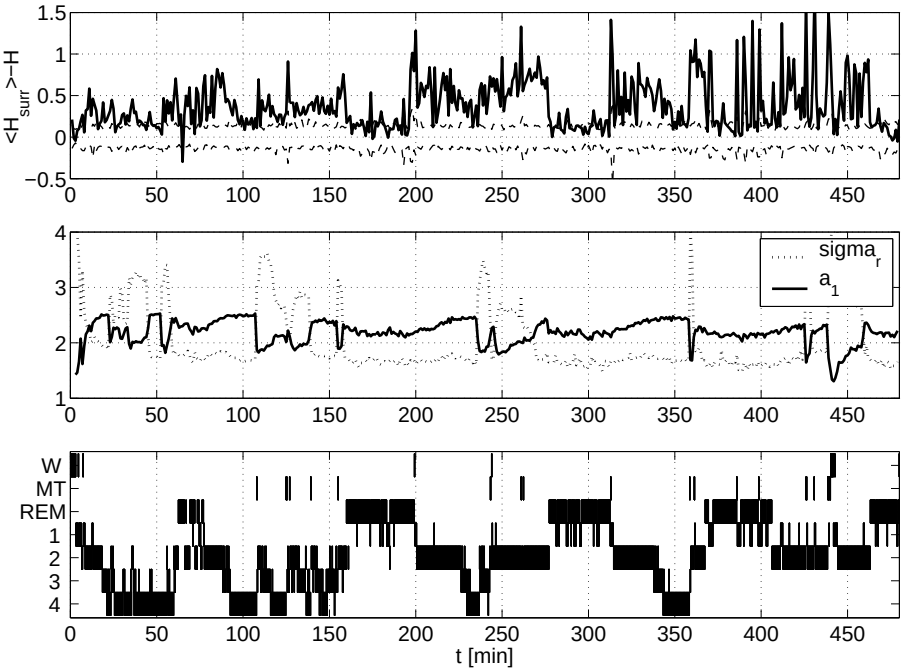

Fig. 2. Upper panel: ΔH of original data (solid line) and maximal and minimal ΔH of 100 surrogates (dotted) for non-overlapping 60-s segments. Middle panel: σ_r and a_1 from 1-s subsegments averaged over 60-s segments. The large jumps are artefacts due to varying levels of tonic muscle activity. Lower panel: hypnogram.

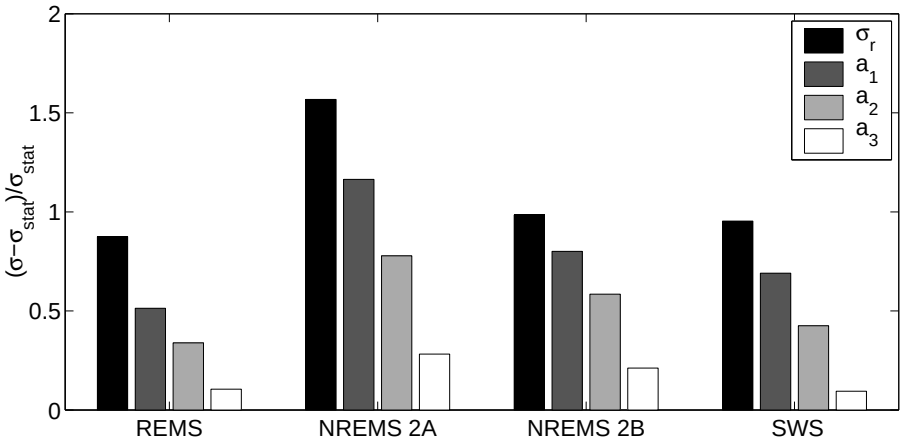

Fig. 3. Difference between the standard deviation of the distribution of four 1-s AR parameters in the 1-min segments (the standard deviation of the residuals σ_ξ and the three first AR-coefficients) and the standard deviation of its estimates normalized to the standard deviation of the estimates.

cients a_1, a_2, a_3 and σ_r. It was estimated by averaging for every sleep stage the difference between the standard deviation of the parameters from the 60 1-s subsegments of every 1-min segment and the standard deviation for the estimator of the respective parameter normalized to the latter. The largest fluctuations are found in sleep stage 2A corresponding to the results of the surrogate data analysis.

4 Conclusions

Our results demonstrate that the nonlinear signatures found in segments of human sleep EEG with durations longer than $10s$ should be explained rather by fluctuations of the AR-coefficients of the 1-s subsegments than by stationary nonlinearities. These parameter fluctuations render the EEG segments non-stationary on these time scales. However, this does not exclude the possibility that the fluctuations themselves might be described by at least approximately stationary processes on time windows of a duration up to several minutes within a definite sleep stage.
This leads to the picture of a hierarchy of three time scales: A first time scale up to 1 s contains the typical EEG patterns like spindles, K-complexes or alpha waves and can be modeled by AR-models. A second time scale up to several minutes, sometimes called microstructure of sleep [7] is characterized by the fluctuations of the AR-parameters. A third time scale from minutes to hours (macrostructure) can be related to the sleep stages and is reflected by the slow dynamics of the mean AR-parameters as it is shown in the middle panel of Fig. 2.

References

[1] H. Akaike, A new look at the statistical model identification, IEEE Trans. Auto. Control 19 (1974) 716–723.

[2] J. Fell, J. Röschke, K. Mann, C. Schäfer, Surrogate data analysis of sleep electroencephalograms reveals evidence for nonlinearity, Biol. Cybern. 75 (1) (1996) 85–92.

[3] J. Pardey, S. Roberts, L. Tarassenko, J. Stradling, A new approach to the analysis of the human sleep/wakefulness continuum, J. Sleep Res. 5 (1996) 201–210.

[4] E. Pereda, A. Gamundi, R. Rial, J. Gonzalez, Non-linear behaviour of human EEG: fractal exponent versus correlation dimension in awake and sleep stages, Neurosci. Lett. 250 (2) (1998) 91–94.

[5] A. Rechtschaffen, A. Kales, A manual of standardized terminology, technics and scoring system for sleep stages of human subjects., Public Health Services, NIH Public. 204.

[6] Y. Shen, E. Olbrich, P. Achermann, P.F. Meier, Dimensional complexity and spectral properties of the human sleep EEG, Clin. Neurophysiol. (2002), in print.

[7] M. G. Terzano, L. Parrino, Origin and significance of the cyclic alternating pattern (CAP), Sleep Med. Rev. 4 (1) (2000) 101–123.