The sleeping brain as a dynamical system - insights from time series analysis of the human sleep EEG

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***Abstract*—The human sleep EEG is studied using a dynamical system approach to the sleeping brain. The dynamics of the sleeping brain is governed by multiple time scales: ranging from the typical EEG oscillations at $1-30$ Hz up to the $\approx$ 24h circadian rhythm. Starting point is the fastest time scale - the sleep oscillations. They are described by modeling the EEG using adaptive linear models. The slower dynamics appear then as time dependence of the parameters of these models.**

***Index Terms*—sleep oscillations**

I. The sleeping brain as a dynamical system

The sleeping brain is a complex dynamical system. It shows a rich repertoire of internally generated dynamic patterns and transitions between states. A particular property of the sleeping brain is that it exhibits dynamics on very different time scales. A sketchy overview is given in Table I. According to the two process model of sleep regulation the interaction between the circadian rhythm and the homeostatically regulated sleep pressure S determines the transitions between sleep and waking [1] while an ultradian process controls the alternation between NonREM and REM sleep.

The slow oscillation of the membrane potential of cortical neurons between "up" (depolarization) and "down" (hyperpolarization) states giving rise to the slow waves in deep sleep and K complexes in light sleep [2]. Between these two time scales some authors assume an additional process leading to an alternation between phasic and tonic activity, the so called cyclic alternating pattern (CAP) [3]. The different time scales are not independent. The sleep stages, for instance, are defined with respect to the occurrence of certain oscillatory patterns [4]. The central marker for sleep homeostasis is the slow wave activity that is usually defined as the spectral band power in the delta frequency band $\approx 0.5-4.5$ Hz.

In the following a general framework will be proposed that allows to analyze systematically the connection between the different time scales by reconstructing appropriate state spaces using parameters of the models of the faster time scales as state vectors for the slower dynamics. After discussing the character of the human sleep EEG as a time series the general framework of the analysis following [5] will be introduced and applied to the human sleep EEG starting from the fastest time scale in sleep EEG - the time scale of the sleep oscillations that is modeled by linear models. It will be discussed, how the sleep oscillations are described by these models and how the parameter dynamics, i.e. the dynamics on the next slower time scale is related to the temporal organization of these sleep oscillations.

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TABLE I
THE DIFFERENT TIME SCALES OF THE SLEEP DYNAMICS

$\approx$ 24 h	Circadian rhythm and sleep homeostasis
$\approx 60-90$ minutes	ultradian process alternation between REM and NonREM sleep
several seconds to several minutes	transitions between NonREM sleep stages
several seconds	Cyclic alternating patterns (CAP)
$\gtrsim$ 1s	Slow oscillations between "up" (high firing rate) and a "down" (no firing) states
$1-20$ Hz	The typical sleep oscillations: delta oscillations, sleep spindles, alpha and theta oscillations

II. The characteristics of the human sleep EEG as a time series

The electroencephalogram (EEG) is resulting the summed postsynaptic activity of large numbers of neurons primarily of the cortex. For clinical purposes one looks usually for particular patterns in the EEG, which are thought to represent some kind of synchronized activity. From the viewpoint of data analysis the EEG has traditionally been described as a stochastic process often characterized by its spectral properties.

After the discovery of the phenomenon of deterministic chaos, i.e. the fact that low dimensional nonlinear deterministic systems could generate aperiodic signals it was speculated that also the EEG might be a chaotic signal because the brain as the underlying dynamical system is deterministic and nonlinear. By applying the Grassberger-Procaccia algorithm [6], [7] to the human sleep EEG several researchers (e.g. [8], [9], [10], [11], [12], [13]) found that the correlation dimension varied with the sleep stages being lower in deeper sleep, higher in light sleep and highest in REM sleep. This appealed very much to the intuition that the dynamics during deep sleep should be less complex compared to the dynamics in light sleep or even the dynamics during REM sleep and the awake state. Nevertheless, already from the beginning these results have been questioned for several reasons: From a general point of view it seems very unlikely that such a complex system as the brain should produce an activity which can be described by low-dimensional dynamics. Moreover, the interpretation that finite values of the correlation dimension were a sign of low dimensional chaos had been challenged by the finding that also correlated noise exhibiting power law spectra $P(f) \propto f^{-\beta}$, can produce finite estimates for the correlation dimension [14]. Furthermore, in [15] a low pass filter to white noise was applied and it was shown that the resulting time series

produced finite estimates of the correlation dimension. These studies demonstrated that finite correlation dimensions can result already from linear stochastic processes. To avoid these spurious finite estimates, Theiler [16] proposed a correction to the original GP algorithm, which reduces the effects of linear correlations. He showed that this correction removes the scaling regions that lead to finite dimensions for colored noise. In [17] it was shown that the usually reported values for the dimensional complexity of the human sleep EEG are indeed an effect of a missing correction for temporal correlations, also known as "Theiler window" [16], [18]. A proper application of the algorithms on the human sleep EEG would result in finding the embedding dimension as dimension estimate, i.e. would reveal that the human sleep EEG shows "stochastic behavior" in the sense of [19]. If one, nevertheless, sticks to a smaller "Theiler window" one can still ask, which property of the data is reflected by a dimensional complexity measured this way. The answer for the human sleep EEG was that dimensional complexity measures essentially the amount of delta power. This result can be directly related to the finding of [14]: The power spectrum of the the sleep EEG between 1 and 20 Hz can be fitted by a power law function except the peaks due to spindle and sometimes theta or alpha activity. In [17] it was shown that the corresponding exponent, the logarithm of the delta power and the dimensional complexity estimated from EEG segments of 1minute duration are strongly correlated. Thus it is very likely that similar reasons as in [14] found for fractional Brownian motion are responsible for the dimensional complexity estimates in the human sleep EEG. This does not mean, however, that the human sleep EEG is best described as fractional Brownian motion, it only says that it is similar and that this similarity is reflected in the dimension estimates.

If the findings for quantities such as the correlation dimension can be explained by linear features of the EEG such as the spectral band power one may ask whether the sleep EEG in general is sufficiently well described by linear models. This type of questions is usually addressed with surrogate data analysis. This is a method for hypothesis testing with the null hypothesis being that the data can be fully described by a linear model. Surrogate data are data with the same "linear" properties, i.e. power spectrum or correlation function, as the original data, but being random otherwise. One computes a nonlinear test statistics, e.g. the dimensional complexity, on both the surrogate data and the original data and rejects the null hypothesis if the value of the test statistics is significantly different from that of the surrogates (see [20] for an overview and [21], [22] for some open problems).

In the case of human sleep EEG surrogate data analysis produced evidences for the rejection of the null hypothesis of a linear stochastic process (see e.g. [23], [13], [17]). These analyses were performed, however, on segments with a length larger than 10s. Because the null hypothesis includes also stationarity, its rejection can be both due to non-linearity on short time scales, i.e. nonlinear oscillations, or non-stationarity, i.e. an additional slow dynamics, and the test cannot distinguish between these two possibilities. By performing the surrogate data test on segments of increasing length it was argued in [24] that rejections of the null hypothesis reported previously are probably due to some additional slow dynamics on a time scale $>$ 1s such as 4s "periodicity" of the occurrence of sleep spindles [25] or the occurrence of K-complexes. The main conclusion from this analysis was that the human sleep EEG is sufficiently well described by linear models on times scales $\approx$ 1s, i.e. the time scale of the sleep oscillations, but incorporating slower dynamics needs more complicated, nonlinear models.

III. Separating the time scales

Considering the sleeping brain as a dynamical system means that its state at any time t can be described by a state variable $\tilde{\mathbf{x}}(t)$ and some transition function that describes how the future state depends on the previous state.

$$\tilde{\mathbf{x}}(t+\Delta) = \mathbf{F}(\tilde{\mathbf{x}}(t), \xi) \tag{1}$$

with Δ denoting the time difference between two observations, i.e. the sampling interval. In general the full state might be not observable. Therefore we have to introduce also an observation equation

$$\mathbf{y}(t) = h(\tilde{\mathbf{x}}(t), \nu(t)) \tag{2}$$

with $\mathbf{y}(t)$ being the observed quantity, for instance one or more EEG channels. ν is denoting the measurement noise. Taking into account a second time scale explicitly we split the original state $\tilde{\mathbf{x}}(t)$ into a new "fast" state $\mathbf{x}(t)$ and a parameter $\mathbf{p}$ with a slower dynamics

$$\begin{aligned} \mathbf{x}(t+\Delta) &= F_x(\mathbf{p}(t), \mathbf{x}(t), \xi_x(t)) & (3)\\ \mathbf{p}(t+\Delta) &= F_p(\mathbf{p}(t), \mathbf{x}(t), \xi_p(t)) & (4)\\ \mathbf{y}(t) &= h(\mathbf{x}(t), \nu(t))\ . & (5) \end{aligned}$$

At this point we made the assumption that the observed quantity, here the measured EEG, does not depend explicitly on the parameters $\mathbf{p}$, but only on the state $\mathbf{x}$. That corresponds to the idea that the EEG is generated by postsynaptic potentials and that neuromodulatory influences determine their strength but do not influence the EEG directly.

So far Eqs. (3,4) are only a rewritten form of (1). Now the basic idea is that the slow dynamics (4) depends not on all details of the fast state $\mathbf{x}(t)$, otherwise it would be fast itself, but only on slow *features* of the fast dynamics. In order to define the notion of such a feature formally we consider the fast dynamics (3) for fixed parameters $\mathbf{p}$

$$\mathbf{x}(t+\Delta) = F_x(\mathbf{p}, \mathbf{x}(t), \xi_x(t)) \tag{6}$$

Thus, a probability density $\rho(\mathbf{x}(t), \mathbf{x}(t-\Delta), \ldots, \mathbf{x}(t-m\Delta)$ exists that is invariant with respect to the dynamics and depends on the parameter vector $\mathbf{p}$. A feature f_i is now defined as an expectation value with respect to this density. Examples for such features would be the auto- or cross-correlations function up to a time delay $m\Delta$ or the power spectra. If the invariant density is unique, its features would be functions of the parameters $\mathbf{p}$. Thus the parameter dynamics (4) can be written as

$$\mathbf{p}(t+\Delta) = \mathbf{G}(\mathbf{p}(t), \mathbf{f}(\mathbf{p}), \xi_p(t)) \tag{7}$$

In order to be consistent the time scale $m\Delta$ for the definition of the features has to be much smaller than the typical time scale of the parameter dynamics (7). More details and some examples for the application of this framework can be found in [5]. The distinction between a state variable and parameters varying on a slower time scale can be iterated leading to a hierarchy of descriptions. In the following we will concentrate on the description of the fastest time scale, i.e. the sleep oscillations and then discuss, how these descriptions might lead to parameter dynamics on slower time scales.

IV. Time varying linear models

The result from [24] that short segments of the human sleep EEG ($\approx$ 1s) can be described sufficiently well by linear models and that nonlinear signatures found in longer EEG segments are probably due to nonstationarities, i.e. slow parameter changes means that in the case of the human sleep EEG linear models should be sufficient for the description of the fast dynamics (3). The most general form to write down a linear model is the state space model - here in the innovation representation:

$$\begin{aligned} \mathbf{x}(t+\Delta) &= \mathbf{A}\mathbf{x}(t) + \mathbf{K}\mathbf{e}(t) \\ \mathbf{y}(t) &= \mathbf{C}\mathbf{x}(t) + \mathbf{e}(t)\ . \end{aligned} \tag{8}$$

with the dynamical matrix $\mathbf{A}$, the observation matrix $\mathbf{C}$, prediction errors (innovations) $\mathbf{e}$ and the Kalman gain $\mathbf{K}$. The deterministic part of the dynamics is described by the matrix $\mathbf{A}$. The dimension of $\mathbf{y}_n$ is given by the number of observations, i.e. the number of EEG channels that are considered. The dimension p of $\mathbf{x}$, i.e. the dimension of the internal state is also a parameter of the model that has to be determined from the data or by other considerations (see below). The other parameters of this model are the matrices $\mathbf{A}$, $\mathbf{C}$, $\mathbf{K}$ and the covariance of the innovations. In order to study the parameter dynamics a reparametrization that allow a better interpretation of the parameters can be obtained by the diagonalization of $\mathbf{A} = \mathbf{V}^{-1}\mathbf{D}\mathbf{V}$. This leads to

$$\begin{aligned} \mathbf{z}(t+\Delta) &= \mathbf{D}\mathbf{z}(t) + \mathbf{V}\mathbf{K}\mathbf{e}(t) \\ \mathbf{y}(t) &= \mathbf{C}\mathbf{V}\mathbf{z}(t) + \mathbf{e}(t)\ . \end{aligned} \tag{9}$$

with Eigenvalues $\lambda_k = r_k \exp(-i\phi_k)$ $k = 1, \ldots, p$. Thus the models (8,9) describe stochastically driven harmonic oscillators with damping constants

$$\gamma_k = \tau_k^{-1} = -\Delta^{-1} \ln r_k \tag{10}$$

and frequencies

$$f_k = \phi_k/(2\pi\Delta) \tag{11}$$

Each Oscillator k is additionally influenced by the size of its driving noise $(\mathbf{V}^{-1}\mathbf{K}\mathbf{e})_k$ and its observability characterized by $(\mathbf{C}\mathbf{V})_k^T$. The basic idea is that an oscillatory pattern with frequency f_k is observed in the EEG if the damping γ_k is sufficiently small and the oscillator is observable. By considering (9) as the description of the fast dynamics (6) with fixed parameters, Eq. (3) corresponds to a description of the human sleep EEG as a set of stochastically driven harmonic oscillators with time dependent frequencies, dampings and possibly also time varying observability and driving noise. Is there any counterpart from the physiological side to such a description? The most direct connection to physiologically motivated models can be made by considering (8) as the linearization around a stationary state. In [26] this connection was demonstrated using a toy model of the Wilson-Cowan type [27] of two randomly coupled populations of excitatory and inhibitory populations of rate[1] neurons driven by random inputs. The system can be analyzed using a mean-field approximation. Linearizing around the fixed point solution one gets a stochastically driven harmonic oscillator with the frequency and damping depending on the excitatory-excitatory and excitatory-inhibitory coupling constants.

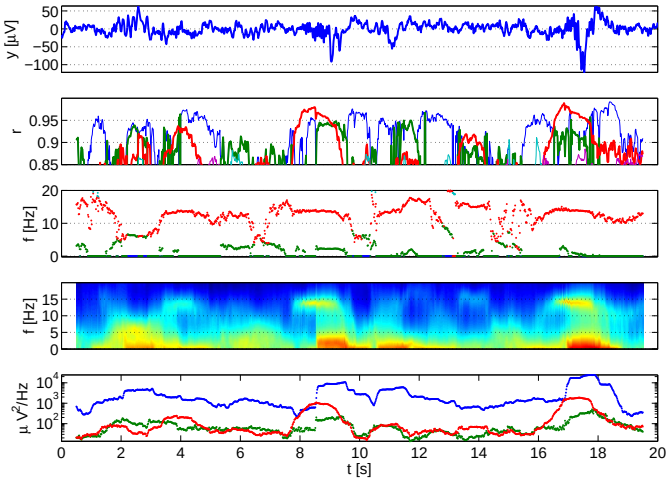

Fig. 1. 20s EEG segment (derivation C3A2) from sleep stage 2. Overlapping 1s segments were fitted with an AR(8) model. Panels from top to bottom (1): data, (2): absolute value r of the poles, (3): frequency f of the poles (4): color coded spectrogram, warmer colors denote higher power. (5): Time course of spectral band power: δ (0.5-4.5 Hz) blue, α (8.5-11 Hz) green, and σ (11-16 Hz) red. Power spectra for (4) and (5) were estimated from the fitted AR(8)-models.

Moreover, it was shown that when these coupling constants are varying in time, the corresponding time dependence of the frequencies or damping can be traced by fitting a linear model with time dependent coefficients to the network activity. This was performed by fitting an autoregressive (AR)-model [26], which is a special case of the state space model (8)(see below) on overlapping segments. A similar analysis using more realistic models such as e.g. [28] has to be done in the future.

A. Single channel data

Fig. 1 shows the result of this analysis to real EEG data from sleep stage 2. There an AR(8)-model was fitted to overlapping 1s segments. The AR(p)-model has the form

$$y(t) = \sum_{k=1}^{p} a_k y(t - k\Delta) + e(t) \tag{12}$$

[1]That means that only the firing rate and not the single spikes were modeled

Eq. (12) can be casted in the form of (8) by setting

$$A_{ik} = \begin{cases} a_k & \text{for } i = 1 \\ 1 & \text{for } i = k+1 \\ 0 & \text{else} \end{cases} \quad \mathbf{C} = \begin{pmatrix} 1 \\ 0 \\ \dots \\ 0 \end{pmatrix} \quad K_k = a_k \ .$$

The panels in Fig. 1 show different possibilities to visualize the result of this procedure. Only the relevant frequency range $0 - 20$Hz is shown. While the second and third panels show the time dependence of the frequencies and the corresponding modules the lower panels show the time course of the spectral power that contains similar information because the power spectrum $P(f)$ of an AR-model can be directly expressed by its parameters: The eigenvalues λ_k are related to the power spectrum by

$$P(f) = \sigma_\epsilon^2 \left| \frac{z^p}{\prod_{k=1}^{p}(z - \lambda_k)} \right|^2 \qquad z = e^{2\pi i f \Delta} \ , \qquad (13)$$

Note, that the order of the autoregressive model p not only determines the maximal number of oscillators $p/2$ that can be modeled, but also the number of frequency bands that could contain information that is not already contained in the other frequency bands.

Sleep spindles can be seen in Fig. 1 at $t = 9$s and $t = 17$s. While the AR-model has a pole in spindle frequency range almost for the full 20s a sleep spindle is only seen in the data, if the damping is sufficiently small, i.e. the corresponding module r is large enough. The oscillation of this module and therefore of the damping of the "spindle oscillator" can be considered as an example of the slow parameter dynamics introduced above. The two sleep spindles in this example are both associated with some slower oscillatory patterns in delta frequency range which however, produce not such a clear signature in the time course of the frequencies and modules, but could be clearly recognized in the time course of delta power.

B. Multivariate analysis

The state space model (9) is the natural starting point for the analysis of more than one channel. Using the full state space model instead of only an autoregressive model adds, however, some technical difficulties. An additional technical difficulty stems from the fact that the estimation of the parameters of the full state space model is not a linear problem anymore in contrast to the AR model and thus is more time consuming and vulnerable to local minima. Moreover, fitting a state space model to 1s raises the danger of overfitting considerably and does not lead to stable results in the sense that the time dependence of the parameters becomes sufficiently continuous. An alternative is showed in Fig. 2. There a state space model of the form (8) with a considerably higher order $p = 24$ was fitted to a 20s segment including 4 channels. In contrast to Fig. 1 the frequencies and dampings of the oscillators are now by definition constant over the whole 20s segment, because only one model was fitted for the whole segment. The typical 4s "period" of the spindle occurrence is therefore visible in the state amplitudes only. This means that, if we assume that

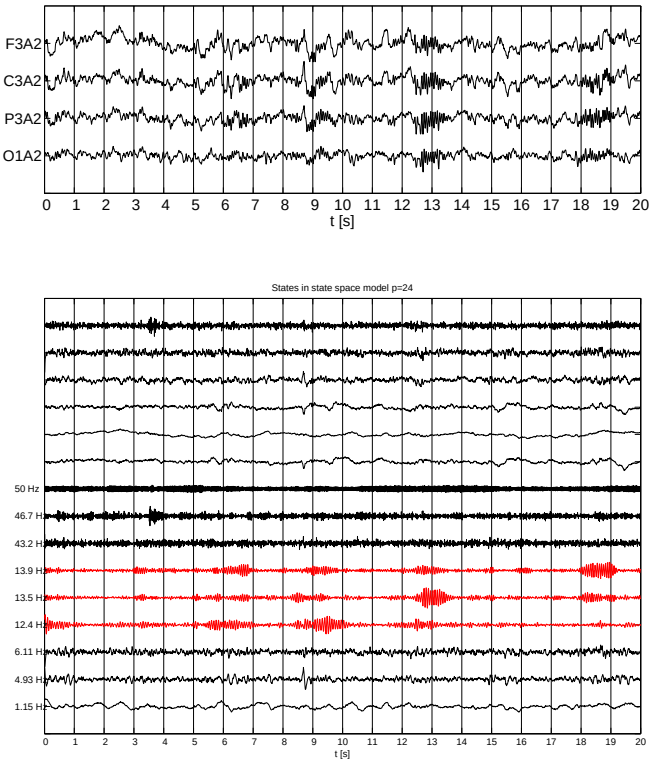

Fig. 2. A state space model of order $p = 24$ was fitted to four channels of 20s human sleep EEG of sleep stage 2.Top: Data, Bottom: Normalized states $\Re z_k + \Im z_k$ with the corresponding frequencies f_k.

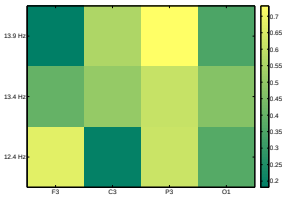

Fig. 3. Normalized coefficients of the observation matrix $(\mathbf{CV})^T$ for the four EEG channels and the three spindle frequency oscillators from Fig. 2.

this "periodicity" is due to an underlying additional dynamical process (see [25] for an extensive discussion), this state space model is not a generative model in the sense, that iterating it with random inputs would generate a signal that also shows this "periodicity", in contrast to the AR-model with time dependent coefficients shown in Fig. 1.

On the other hand, fitting the model to longer time segment allows a better frequency resolution. In the presented example one sees that the models fits three oscillators in the spindle frequency range with slightly different frequencies. As shown in Fig. 3 a closer analysis reveals that the slower spindles are stronger visible in the more frontal derivations.

V. Outlook

It was shown that a dynamics on different time scales can be modeled by dividing the original state space into fast new state variables and slow parameters. In the case of the human sleep EEG it was argued that the fast dynamics can

be modeled using linear models. Then the slower parameter dynamics can be considered as the dynamics of the frequencies and dampings of stochastically driven oscillators underlying the sleep oscillations. Moreover, there is a direct connection from this description to a description of slower dynamics using spectral band power, such as slow wave activity, the spectral band power in the δ-frequency band which is the central observable in models of sleep homeostasis [1].

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