Dynamics of parameters of neurophysiological models from phenomenological EEG modeling

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Abstract

We investigate a recently proposed method for the analysis of oscillatory patterns in EEG data, with respect to its capacity of further quantifying processes on slower (< 1 Hz) time scales. The method is based on modeling the EEG time series by linear autoregressive (AR) models with time dependent parameters. Systems described by such linear models can be interpreted as a set of coupled stochastically driven oscillators with time dependent frequencies and damping coefficients. It is an open question to which extent the estimated frequencies and dampings correspond to true properties of oscillatory eigenmodes in the underlying networks. The present study investigates this relationship using simple neural network models to generate artificial data with controllable properties. We demonstrate that the method detects changes of the eigenmodes induced by slow parameter changes of the network very well.

Key words: EEG, oscillations, data analysis, sleep, slow oscillation, thalamocortical network

1 Introduction

The EEG was traditionally analysed visually by looking for characteristic patterns such as alpha waves, sleep spindles, slow waves in sleep, spikes and spike waves in epilepsy, and other specific patterns in pathological conditions. Today computer-based spectral analysis is still the main method of analysis using the spectral power of activity in

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characteristic frequency bands. This method is simple and fast, but also unspecific regarding the physiological causes of the underlying oscillations.
We here propose a method, which allows to automatically detect and model oscillatory patterns traditionally used in EEG analysis, but is further capable to quantify processes on slower (< 1 Hz) time scales related to the temporal organisation of these oscillatory patterns. These might reflect modulatory influences on the rhythm-generators causing waxing and waning oscillatory patterns (gamma, delta) or their appearance and disappearance (e.g., the 4-second "period" of sleep spindle occurrence). The slow parameter dynamics unraveled this way may yield additional insight into properties of the generating neural circuits on top of conventional methods.
For the analysis of the EEG data we here restrict ourselves as a first step to single channel data modelled by linear autoregressive (AR-)models with time dependent parameters. The method can be extended to multivariate data by using state space models instead (see e.g. [1]). Systems described by such linear models can be interpreted as a set of coupled stochastically driven harmonic oscillators with time dependent frequencies and damping coefficients. Specific oscillatory patterns show up in the data, when the damping becomes smaller than an appropriately defined threshold. The method has been been applied recently to the characterisation of oscillations in human sleep EEG [2,3].
However, it is an open question to which extent the estimated frequencies and damping coefficients correspond to properties of the underlying networks. A first attempt in this direction [4] for wake EEG was criticised by referring to the high dimensional and nonstationary character of the EEG dynamics [5]. While the nonstationary character is explicitly taken into account in our approach we also argue that at times when the EEG is dominated by a certain oscillatory pattern the estimated frequencies and dampings might indeed have physiological meaning. A first attempt to establish such a relationship was made by Liley [6] who studied the effect of benzodiazepines on alpha and beta oscillations in the waking EEG.
The present study investigates the relationship between the frequencies and damping coefficients estimated from the EEG time series and the parameters of the underlying network using a simple neural network model to generate artificial data with controllable properties. The artificial data is then analysed using our method and the resulting theoretical and empirical frequencies and damping coefficients are compared. We demonstrate that the method in this simple case detects changes of properties of the eigenmodes in the network induced by slow parameter changes pretty well, but that also unexpected phenomena can occur.

2 Data analysis

Short segments of an EEG signal $x(t)$ sampled at discrete times t_n are modelled by autoregressive models, AR(p), of order p using the Burg algorithm (see e.g. [1]). The AR(p)-model

$$x(t_n) = \sum_{i=1}^{p} a_i x(t_{n-i}) + \epsilon(t_n) \tag{1}$$

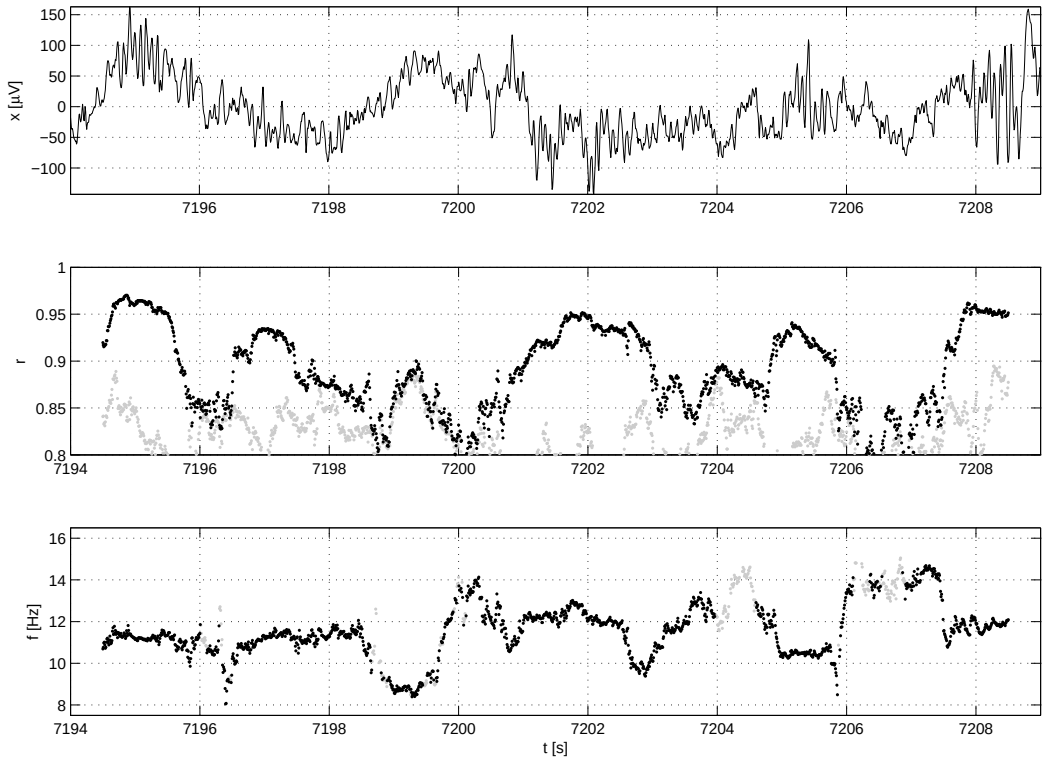

Fig. 1. 15 second segment from NonREM sleep stage 2. EEG data from C3-A2 derivation (top), absolute values r (middle) and frequencies f of the two least damped poles (black and grey) of an AR(8)-model.

linearly predicts $x(t_n)$ based on previous measurements with $\epsilon(t_n)$ denoting the residual errors. Frequencies $f_k = \phi_k/(2\pi\Delta)$ and damping coefficients $\gamma_k = 1/\tau_k = -\Delta^{-1} \ln r_k$, where $\Delta = t_n - t_{n-1}$ denotes the sampling interval, are then estimated from the poles z_k of the AR-model which correspond to the eigenmodes of the estimated linear system:

$$z^p - \sum_{k=1}^{p} a_k z^{p-k} = \prod_{k=1}^{p} (z - z_k) \qquad z_k = r_k e^{i\phi_k} \ . \tag{2}$$

The order p of the model determines the maximum number of poles z_k. In principle the order p of the AR-model and the segment length for the fit are free parameters of the method. The longer the segment used for the fit the better the estimates of the frequency and damping, but the lower the temporal resolution. A similar tradeoff holds for the model order: the higher the model order the more oscillatory modes might be potentially captured but the statistic becomes progressively worse because the number of parameters increases. Moreover, spurious oscillatory modes might occur for too high model orders and the detection of oscillatory events becomes harder due to the worse statistics.

Figure (1) exemplarily shows a reasonable analysis of a 15 seconds segment from NonREM sleep stage 2 (C3-A2 derivation, sampling frequency 128 Hz), cf., also [3]. The analysis was performed using AR(8) models on overlapping 1-s segments. The black curves for r and f correspond to the least damped eigenfrequency of the system, i.e., the mode with maximal r. Note that only one frequency band is displayed, the frequencies of the other poles are not shown.

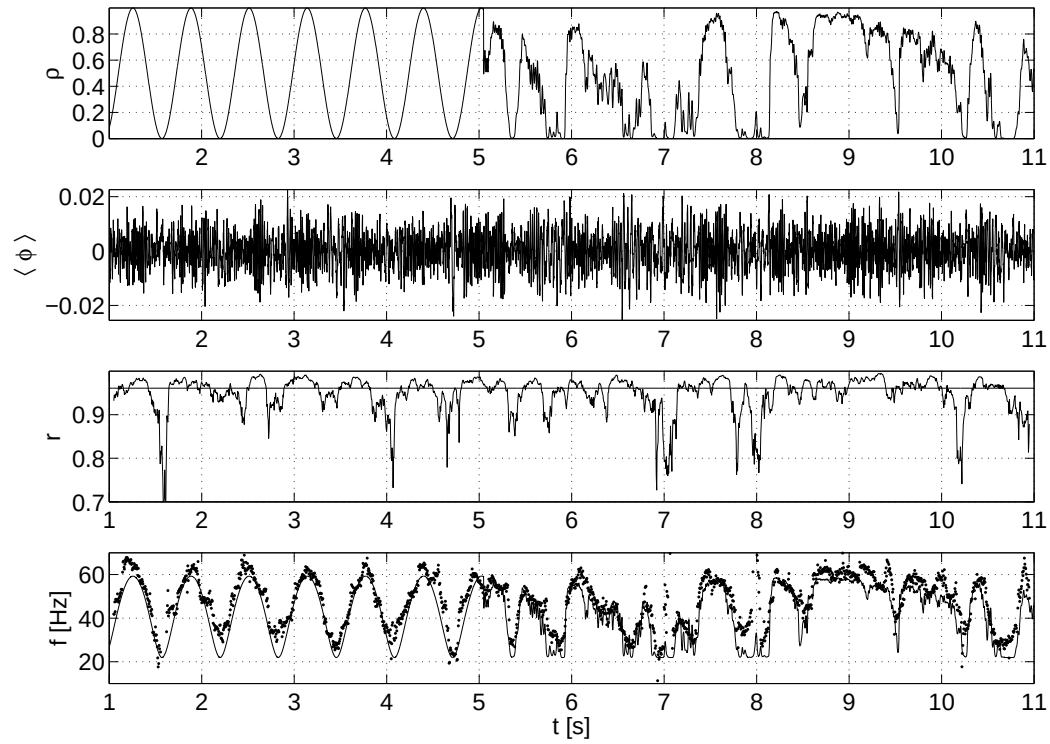

Fig. 2. Result of the analysis of the artificial data (segment length 100 ms) for the modulation of the inhibitory–excitatory coupling K. Open loop control (4) from second 1 to 5 and closed loop control (5) later. Shown are: The time course of the parameter ρ_K (top), the mean potential $\langle\phi\rangle$ (second), the absolute values r (third) and the frequencies f (bottom) of the poles of the fitted AR(8)-models. The solid black lines correspond to the r value and frequency analytically derived from the model in Eq. (9).

3 The neural network model

In order to get an impression of the quality of pole parameters derived from our method, we tested the algorithm with artificial data from a neural network of $N = 180$ excitatory and inhibitory cells with potentials ϕ_i and ψ_i, respectively:

$$\tau_1 \frac{d\phi_i}{dt} = -\phi_i + I_1 + J \sum_j w^1_{ij} f_1(\phi_j) - K \sum_j w^2_{ij} f_2(\psi_j) + \eta_i \tag{3}$$

$$\tau_2 \frac{d\psi_i}{dt} = -\psi_i + I_2 + \sum_j w^3_{ij} f_1(\phi_j) \ . \tag{4}$$

The mean excitatory potential $\langle\phi\rangle := \sum_{i=1}^N \phi_i/N$ was used as an artificial EEG signal in subsequent analyses with a sampling interval $\Delta = 0.001$s. Model parameters used are $I_1 = I_2 = 0$, $\tau_1 = \tau_2 = 10$ ms, $f_1 = \tanh$, $f_2 = \mathrm{id}$. Coupling matrices $(w^\alpha_{ij})_{ij}, \alpha = 1, 2, 3$ are $GWN(1./N, \sigma)$ with $\sigma = 0$ for simplicity. The η_i are i.i.d. $GWN(0, 1)$-processes per step at a step-size of 0.1ms. For strong enough excitatory and inhibitory couplings the model exhibits a Hopf-bifurcation. The network is of the same type as the networks used for modeling alpha oscillations [7], but at the moment the model parameters are arbitrarily chosen with no concrete physiological reference.

Network properties are slowly modulated near the bifurcation via the coupling constants J and K in (3). Specifically, we assume that $J = J_0\rho_J$ and $K = 3(.75 + 4\rho_K)$, where ρ_J and ρ_K are slow temporal parameters. Such modulations vary the frequencies and dampings of network oscillations in a predictable way, see below. We considered three cases:

(1) Varying K – open-loop control: $\rho_K = .5 + .5\cos(\omega_0 t)$ with $\omega_0 = 1$ and $J = 1.2 = const.$

(2) Varying K – closed loop control: here changes in ρ_K are self-generated by the network using an additional slow bistable loop added to the model (3) and (4):

$$\tau_{31}\frac{dz_1}{dt} = -z_1 + I_3 + J_{33}f_3(z_2) + J_{31}\sum_{j=1}^{N} f_1(\phi_j) \tag{5}$$

$$\tau_{32}\frac{dz_2}{dt} = -z_2 + z_1 \quad \rho_K = .5(f_3(z_2) + 1.0)\ . \tag{6}$$

$z_1,\ z_2$ low-pass the excitatory mean activity and the excitatory feedback $J_{33}f_3(z_2)$ in (5) introduces hysteresis in the added loop which by means of ρ_K feeds back to the main system (see Fig. 2, where $I_3 = -0.5, \tau_{31} = \tau_{32} = 30, J = 1.3, J_{31} = 1.5, J_{33} = 30, f_3 = \tanh$ for $t > 5$).

(3) Varying J - open loop control: $J = 1.8\rho_J$, $\rho_J = .5{+}.5\cos(\omega_0 t)$ with $\omega_0 = 0.25$ and $\rho_K = 0.5$.

4 Analytic expressions for Frequency and Damping

The simple network model (3,4) allows for the derivation of analytic approximations for the frequency and damping in order to compare them with the result from the data analysis. The following approximations have to be made

Mean-field approximation: Systems of the form (3,4) with random coupling coefficients satisfy a law of large numbers [8,9]. In the asymptotic limit every single excitatory or inhibitory potential approaches the value of the mean arbitrarily closely. For finite systems the means of the excitatory and inhibitory potentials satisfy the stochastic differential equation

$$\tau_1\frac{d\langle\phi\rangle}{dt} = -\langle\phi\rangle + I_1 + JEw_{ij}^1 f_1(\langle\phi\rangle) - KEw_{ij}^2 f_2(\langle\psi\rangle) + \eta_1 \tag{7}$$

$$\tau_2\frac{d\langle\psi\rangle}{dt} = -\langle\psi\rangle + I_2 + Ew_{ij}^3 f_1(\langle\phi\rangle) + \eta_2\ . \tag{8}$$

In (7) and (8), $\langle\phi\rangle$ and $\langle\psi\rangle$ are the mean potentials of the E- and I-cells, $Ew_{ij}^n, n = 1, 2, 3$ are the expectation values of the couplings, and η_1 an η_2 are effective noise-sources with standard deviation of the order $\frac{1}{\sqrt{N}}$. The absolute value of the effective

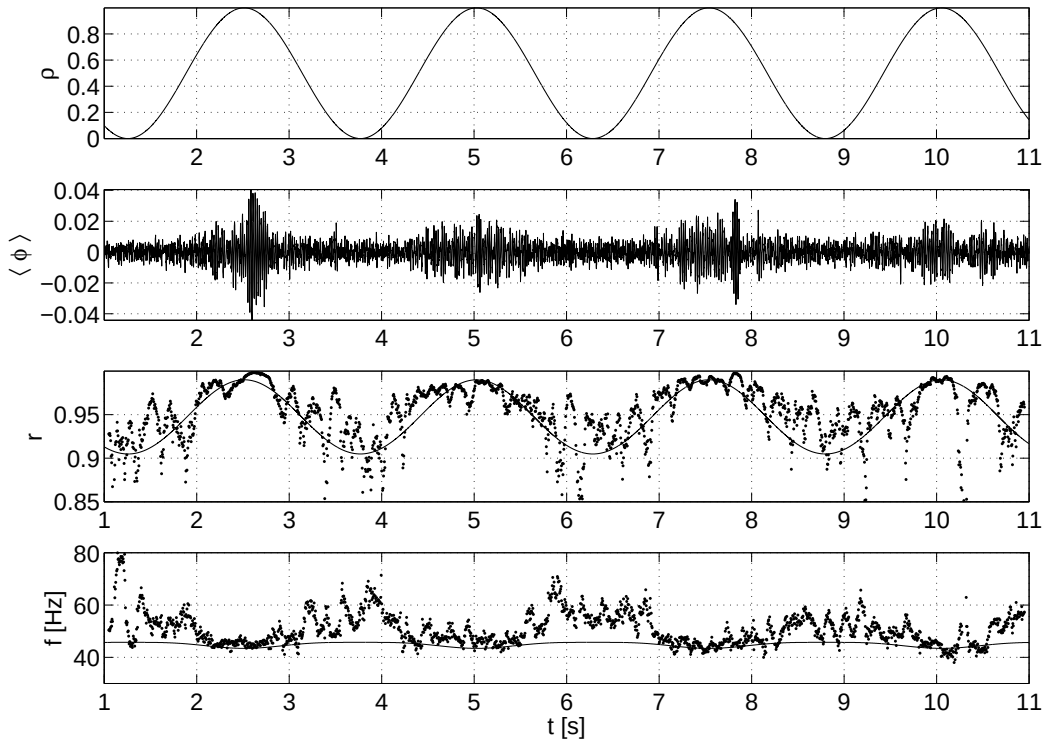

Fig. 3. Same as in Fig. 2 but with modulation of the excitatory–excitatory coupling J and only open loop control.

noises is not relevant here, but they vanish for $N \to \infty$. Accordingly, the whole system behaves like a single stochastic oscillator in the limit of large system size.

Adiabatic approximation: The time-dependent parameter ρ_x, $x = J$ or K, varies on a slower time scale than the mean potentials $\langle\phi\rangle$ and $\langle\psi\rangle$. We can then solve (7) and (8) assuming that ρ_x is constant leading to solutions parameterised by $\rho_x(t)$.

First, we look for the stationary solution of (7,8) without noise. In the present case with $I_1 = I_2 = 0$ this is simply $\phi^0 = \psi^0 = 0$. Second, we study the influence of the noise by linearising the system around the stationary solution. This results in a stochastically driven harmonic oscillator which corresponds to an AR(2)-model for discrete time. Using standard methods one obtains for the frequency $\omega^2 = (2\pi/f)^2$ and the damping γ the expressions

$$\omega^2 = \frac{\hat{K}}{\tau_1\tau_2} - \frac{1}{4}\left(-\frac{1}{\tau_1} + \frac{1}{\tau_2} + \frac{\hat{J}}{\tau_1}\right)^2 \tag{9}$$

$$\gamma = \frac{1}{2}\left(\frac{1}{\tau_1} + \frac{1}{\tau_2} - \frac{\hat{J}}{\tau_1}\right) . \tag{10}$$

with $\hat{J} = J f_1'(\phi^0)$ and $\hat{K} = f_1'(\phi^0) f_2'(\psi^0) K$. For $\hat{J} \geq 1 + \frac{\tau_1}{\tau_2}$ i.e. $\gamma < 0$, and $\hat{K} > \frac{\tau_1}{\tau_2}$ the stationary solution becomes unstable leading to a stable limit cycle (Hopf bifurcation). From (9) and (10) it is apparent that variation of ρ_K – and thereby $\hat{K}$ – does not influence the damping but only the frequency of the oscillation. In contrast ρ_J modulates both, frequency and damping, although the impact seen on frequency is often only small, cf., Fig. 3.

5 Numerical Results from Simulated data

The results for artificially generated data sets are shown in Fig. 2 and Fig. 3. In Fig. 2 the inhibitory-excitatory coupling (i.e., K and $\hat{K}$) was modulated introducing a time dependent frequency. The first half of the data set was generated with *open loop control* and the second half under *closed loop* conditions as described earlier. An AR(8)-model was fitted to overlapping segments of 0.1 seconds length. There is a good agreement between the time course of the frequencies estimated form the data and the analytical result as shown in the lowest panel in Fig. 2. However, also the absolute values r of the poles, which are related to the damping of the oscillations, appear modulated by the slow parameter ρ_K. From the analytical result Eq. (10) one would have expected to observe no influence. The reason for this modulation is unclear. It might result from nonlinearities in the network which are not considered in the theoretical parameter estimates; it could also reflect some synchronisation effects between the $N = 180$ E and I-cells in the network and it could further result from the frequency dependent bias of the fitting procedure.

In order to study time-dependent damping constants, too, the excitatory–excitatory coupling was modulated in a further simulation. Parameters were chosen such that the impact of the modulation on the frequency of the oscillations was only weak. The results are shown in Fig. 3. Apparently, the method is able to detect the modulation of the damping well. However, the oscillation generated by the modulation of the damping constant (Fig. 3, third frame) seems to be superimposed by a faster, pretty noisy oscillation, leading to jerky excursions of r (and f), an effect already present also in Fig. 2. The origin of this oscillation is again not clear. Preliminary analyses showed that it is related to several phenomena. First, there is a correlation between the instantaneous amplitude of the signal and the estimated value of r. The time scale of this fluctuations is governed by the damping constant itself. Second, there is an effect of the spurious correlations introduced by the overlap of the analysed segments. However, more work is needed to resolve this issue in detail.
Furthermore, note that the frequency can be estimated only reliably when the damping is sufficiently small, i.e. $r > 0.95$. This corresponds to the threshold levels for oscillatory events derived empirically in our earlier work [2,3].

6 Conclusions

We have shown in a simple model that the modulation of the frequency and the damping constant of the resonance of an excitatory-inhibitory network can be detected from the mean activity by applying a recently developed method for the analysis of oscillatory patterns in EEG data. This result supports the idea to relate frequency changes in typical oscillatory patterns of human EEG such as observed during sleep for sleep spindles or delta oscillations [3] to modulations of coupling constants or other parameters in the underlying thalamocortical networks.
The presented result can serve as a starting point for further tests of the proposed

method of EEG data analysis.

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