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State Space Oscillator Models for Neural Data Analysis

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Abstract

Neural oscillations reflect the coordinated activity of neuronal populations across a wide range of temporal and spatial scales, and are thought to play a significant role in mediating many aspects of brain function, including attention, cognition, sensory processing, and consciousness. Brain oscillations are typically analyzed using frequency domain methods such as nonparametric spectral analysis, or time domain methods based on linear bandpass filtering. A typical analysis might seek to estimate the power within an oscillation sitting within a particular frequency band. A common approach to this problem is to estimate the signal power within that band, in frequency domain using the power spectrum, or in time domain by estimating the power or variance in a bandpass filtered signal. A major conceptual flaw in this approach is that neural systems, like many physiological or physical systems, have inherent broad-band "1/f" dynamics, whether or not an oscillation is present. Calculating power-in-band, or power in a bandpass filtered signal, can therefore be misleading, since such calculations do not distinguish between broadband power within the band of interest, and true underlying oscillations. In this paper, we present an approach for analyzing neural oscillations using a combination of linear oscillatory models. We estimate the parameters of these models using an expectation maximization (EM) algorithm, and employ AIC to select the appropriate model and identify the oscillations present in the data. We demonstrate the application of this method to univariate electroencephalogram (EEG) data recorded at quiet rest and during propofol-induced unconsciousness.

I. INTRODUCTION

Oscillations are ubiquitous in neural systems and reflect the coordinated activity of neuronal populations across a wide range of temporal and spatial scales. Neural oscillations are thought to play a significant role in mediating many aspects of brain function, including attention [1], cognition, sensory processing, and consciousness [2]. Accordingly, neurocognitive and psychiatric disorders, as well as altered states of consciousness, have been associated with altered or disrupted brain oscillations. Neural oscillations can be recorded in both animal and human models, at scales spanning individual neuron membrane potentials, neural circuits, and non-invasive scalp electromagnetic fields. Thus, neural

oscillations can provide an important mechanistic scaffold to link observations across different scales and models. Characterizing such mechanistic links could facilitate the development of specific and physiologically-principled brain dynamic biomarkers.

Brain oscillations are typically analyzed using frequency domain methods such as nonparametric spectral analysis, or time domain methods based on linear bandpass filtering. A typical analysis might seek to estimate the power within an oscillation sitting within a particular frequency range, for instance, an alpha oscillation whose frequency is between 8 and 12 Hz. A common approach to this problem is to compute the power between 8 to 12 Hz, in frequency domain using the power spectrum, or in time domain by estimating the power or variance in a bandpass filtered signal. A major conceptual flaw in this approach is that neural systems, like many physiological or physical systems, have inherent broadband "1/f" dynamics, whether or not an oscillation is present. Calculating power-in-band, or power in a bandpass filtered signal, can therefore be misleading, since such calculations do not distinguish between broadband power within the band of interest, and true underlying oscillations. In this paper, we present a novel approach for analyzing neural oscillations using a combination of linear oscillatory models. We estimate the parameters of these models using an EM algorithm, and employ the AIC to select the appropriate model, which serves to identify the oscillations present in the data. We demonstrate the application of this method to electroencephalogram (EEG) data recorded at quiet rest and during propofol-induced unconsciousness.

II. Methods

A. Model Formulation

We wish to construct a state space model that can parametrically represent oscillations at different frequencies, as well as "1/f" dynamics. As a working example, we consider the case of the propofol-induced frontal EEG, which has a combination of slow (0.1 to 1 Hz) and alpha (8 to 12 Hz) oscillations. We can represent these oscillations as distinct components that are observed in noise:

$$y_t = x_{slow,t} + x_{alpha,t} + v_t \quad (1)$$

Each oscillatory component can be modeled using a second-order autoregressive (AR(2)) process, written in state space form as:

$$y_t = [1 \ 0 \ 1 \ 0] \begin{bmatrix} x_{slow,t} \\ x_{slow,t-1} \\ x_{alpha,t} \\ x_{alpha,t-1} \end{bmatrix} + v_t \quad (2)$$

$$x_t = \begin{bmatrix} a_1 & a_2 & 0 & 0 \\ 1 & 0 & 0 & 0 \\ 0 & 0 & a_3 & a_4 \\ 0 & 0 & 1 & 0 \end{bmatrix} x_{t-1} + \begin{bmatrix} w_{s,t} \\ 0 \\ w_{a,t} \\ 0 \end{bmatrix}, \quad (3)$$

where

$$v_t \sim \mathcal{N}(0, \sigma_v^2) \quad w_{s,t} \sim \mathcal{N}(0, \sigma_{ws}^2) \quad w_{a,t} \sim \mathcal{N}(0, \sigma_{wa}^2).$$

We note that the AR(2) form can represent oscillations with different amplitudes, and different levels of resonance or bandwidth. In addition, the AR(2) spectrum has a "1/f" profile at frequencies above the oscillatory frequency. More generally, we can imagine a number of alternative scenarios where one or both of these oscillations are absent. For instance, in the awake resting state, frontal EEG channels would not be expected to have an alpha oscillation, nor a slow oscillation. In this instance, broad-band "1/f" dynamics might be present, and could be modeled using an AR(1) process. Alternatively, a combination of AR(1) and AR(2) dynamics could also be represented in this state space form.

In the analysis to follow, we will consider all of these possibilities, and use model comparison with AIC to choose the most appropriate model given the observed data. As a shorthand, we will refer to the model described in equations (2) and (3) as an "AR(2+2)" model, since there are two AR(2) processes in this model. Similarly, we will refer to the combination of AR(1) and AR(2) processes as an "AR(1+2)" model. We will also consider models with a single dynamic component represented by either AR(1) or AR(2) state dynamics. We note that these models are technically autoregressive moving average (ARMA) models due to the presence of observation noise in equation (3), but we will refer to the models according to their underlying state dynamics to emphasize their oscillatory structure.

B. Estimation Algorithm

We estimate the AR parameters and noise covariances for these models using the expectation maximization (EM) algorithm for state space models described by Shumway and Stoffer [3]. Briefly, the complete data likelihood can be written for the state space model in equations (2) and (3), and closed-form expressions for the M-step can be derived. The E-step is carried out using the Kalman filter, fixed-interval smoother, and covariance smoothing algorithms [4,5]. We use our prior knowledge of the EEG spectrum under propofol anesthesia to set initial values for the AR parameters, such that the initial slow oscillation peak frequency is set to 1 Hz, the initial alpha oscillation peak frequency is set to 10 Hz, with the poles for both oscillation set initially at a radius of 0.99 from the origin in the complex plane. The EM algorithm was run for 200 iterations on every model. All models reached a plateau in the log likelihood by this point.

C. Data Analysis and Model Comparison

To evaluate the performance of the models and estimation method, we analyzed EEG recorded during induction of propofol-induced unconsciousness in healthy volunteers between the ages of 18 to 36 years. The study was conducted at Massachusetts General Hospital and was approved by the Human Research Committee. The EEG signals were recorded at sampling frequency of 5000 Hz, downsampled to 200 Hz. We analyzed a single frontal channel of EEG, comparing an awake, eyes-closed baseline state to a propofol-induced unconscious state. In the propofol-induced unconscious state, the frontal EEG contains large slow (0.1-1 Hz) and alpha (8-12 Hz) oscillations. In the baseline state, these oscillations are absent, although a slow drift may be observed.

We used the Akaike Information Criterion (AIC) to select from among the AR(1), AR(2), AR(1+2), and AR(2+2) models described earlier [6]. To account for the state space formulation of the model, we included the AR parameters, state noise variances, and observation noise variance as parameters in the AIC, all of which were estimated by the EM algorithm.

A common approach for decomposing a signal into frequency-dependent components is to apply bandpass filters. We therefore compared the performance of our state space models with bandpass filtered signals. To represent the slow wave, we used an 100 order Hamming-windowed low pass FIR filter with a 1 Hz cutoff frequency. To represent the alpha wave, we used an order 100 Hamming-windowed bandpass FIR filter with a passband between 8 and 12 Hz. We created a reconstructed bandpass signal by summing the bandpassed slow and alpha components.

III. Results

Table I shows the AIC values for the different models considered, for both the baseline condition and the propofol-induced unconscious condition. For the baseline case, the AIC is lowest for the AR(1) model, whereas for the propofol condition, the AIC is lowest for AR(2+2) model, allowing us to select these models for each respective condition. The spectra for the fitted models are shown in Figure 1, alongside a nonparametric multitaper spectral estimate of the observed data (3 Hz spectral resolution, 26 tapers over 10 seconds of data).

There is no discernible alpha oscillation in the case of resting state, supporting the model selection of AR(1). The residuals between the modeled observed data and the collected data in resting state (i.e., the innovations) had a magnitude of 4.9348 uV or less. In this AR(1) case, there is only one hidden state that is nearly identical to the recorded data. The AR(1) model for this baseline condition is:

$$\begin{aligned} x_{slow,t} &= 0.9953x_{slow,t-1} + w_{slow,t} \\ w_{slow} &\sim \mathcal{N}(0, 5.1593) \end{aligned}$$

The spectrum of the AR(2+2) model representing the propofol-induced unconscious case is shown in Figure 1, again alongside a multitaper spectral estimate of the observed data. The

slow and alpha oscillation components of the AR(2+2) model show a close correspondence to the peaks observed in the multitaper spectrum. The AR(2+2) model has independent hidden states representing the slow and alpha oscillations, making it possible to separate the oscillations in the time domain. Figure 2 shows the slow and alpha oscillation time series estimated under this model. The residuals or innovations have a magnitude of 0.2198 uV or less. The AR models for each component in this propofol case are

$$\begin{aligned}x_{slow,t} &= 1.961x_{slow,t-1} - 0.9627x_{slow,t-2} + w_{slow,t} \\w_{slow} &\sim \mathcal{N}(0, 0.0062) \\x_{alpha,t} &= 1.7826x_{alpha,t-1} - 0.9139x_{alpha,t-2} + w_{alpha,t} \\w_{alpha} &\sim \mathcal{N}(0, 1.6368)\end{aligned}$$

Characteristic features of the oscillations, such as their peak frequency, can be calculated directly from the model parameters [7]. The peak frequency of each AR(2) component is given by

$$\omega = \arccos \frac{a_1(1-a_2)}{4a_2},$$

where a_1 is the first time lag parameter and a_2 is the second. The peak frequency of the slow wave under propofol is 0.9475 Hz and the peak frequency of the alpha oscillation is 11.6952 Hz. The slow component during resting state has a peak frequency of 0 Hz by the definition of an AR(1) process, since an AR(1) process has a single real-valued pole at zero frequency. The poles of the AR model components determine the shape of the power spectrum and the peak frequencies. The radius of the pole determines the prominence of the peak frequency and in effect the damping of the oscillatory process. In the AR(2) model for slow wave oscillations under propofol, the radius of the poles is 0.99 and in the model for alpha, the radius is 0.956, showing that the slow wave is slightly more prominent, as expected.

We compared the performance of our state space model with the more conventional bandpass filtered approach. Figure 3 shows the multitaper spectra for the bandpass slow and alpha components, the reconstructed signal, and the residuals, alongside the residuals for the state space model. Although bandpass filtering identifies oscillations similar to those obtained using the state space model, the residuals under the bandpass model show prominent low frequency oscillatory structure. By comparison, the residuals from the state space model are approximately 60 dB smaller and less structured. In the bandpassed model, the power and structure in the residuals are a consequence of leakage outside the arbitrary bandpass cutoffs, which our method avoids by using AR models to represent the component oscillations. Figure 4 shows the component oscillations, reconstructed signals, and residuals for the bandpass method in time domain, alongside comparisons to the state space. Similar to the frequency domain analysis, we see significant time domain residual structure under bandpass filtering that is absent under the state space model.

IV. Discussion

Our results illustrate how state space models, structured in a particular form consisting of a combination of first and second-order systems, can be used to identify oscillatory structure in neural time series. A typical approach taken in neuroscience analyses is to compute the power within a spectral band, or variance in a bandpass filtered signal. A major limitation of this approach is that broad band noise spanning the frequency range of interest cannot be distinguished from an oscillation. Our approach addresses this limitation by explicitly modeling potential oscillatory components, and then selecting amongst a set of models with different oscillatory configurations to determine the composition of the signal. Under this approach, power attributed to a specific oscillatory component can be calculated, independent of other broadband components, and the component oscillatory time series can be separated.

Our approach differs from traditional AR or ARMA modeling by specifying a structure for the AR parameters to represent either drift (AR(1) or AR(2) with real poles) or oscillatory terms (AR(2) with complex poles). Although a more general AR or ARMA model can certainly identify such components, our experience is that the spectral representations of such models can be highly variable as the model order increases [8]. By positing a specific form for the putative drift terms or oscillations, we can reduce the number of model parameters required to represent such features, and increase the robustness of the model estimates. Moreover, the structure of our proposed models is more easily interpreted in terms of specific oscillatory components than a more general AR or ARMA structure.

Other authors have recognized the need to identify oscillations distinct from broadband noise in neural data. Voytek, et al., use a curve fitting procedure in frequency domain that models $1/f$ and oscillatory components in the power spectrum [9]. This method works well to model the spectrum and identify peaks in the spectrum. Our proposed method has the advantage that oscillatory components can be separated in the time domain. The linear systems approach we have described may also provide more specificity in detecting oscillations, since the temporal structure or impulse response implied by the AR components is specific to oscillators. Matsuda and Komaki [10] have described a very similar oscillatory model, albeit composed of separate sinusoidal and cosinusoidal components intended to facilitate phase estimation. Their work has focused on phase estimation, rather than identification and separation of oscillations. Nonetheless, their approach is highly consonant with ours.

Empirical mode decomposition (EMD) and independent component analysis (ICA) are popular methods that might also be used to separate oscillatory components within a signal. EMD can separate out oscillatory signals centered at different frequencies [11]. However, there are often multiple intrinsic mode functions (IMFs) in the same frequency range that could be difficult to select or interpret. Moreover, the convergence criteria for EMD is arbitrary and the frequency content of individual IMFs can be difficult to specify or control. ICA usually requires multiple channels, and assumes that the underlying time series are independent [12]. The assumption of independence may not be reasonable in many

neuroscience problems, in which we typically observe temporally and spatially correlated signals generated by highly interconnected brain structures.

Future work to characterize and compare the statistical properties of this method and others will be crucial for neuroscience and clinical applications. In the meantime, our results suggest that state space models can provide more precise characterizations of neural oscillations.

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Fig. 1.
Power spectra of Recorded EEG Data and AR Component Equations of the models selected by AIC.

Fig. 2. Estimated Slow and Alpha Oscillation Time Series Using the AR(2+2) model During Propofol-induced Unconsciousness with 95% Confidence Interval

Fig. 3. Power spectra of Recorded EEG Data, Oscillations from Bandpass Filtering, and Model Residuals During Propofol-induced Unconsciousness.

Fig. 4. Oscillation Components Isolated using Bandpass Filters Compared to Oscillation Components Estimated using the State Space Model.

TABLE I

AIC for Frontal EEG

Model	Number of Parameters	Rest	Propofol
AR(1)	3	6683.3	6664.6
AR(2)	4	6685.4	3728.8
AR(1+2)	6	6690.8	3567.8
AR(2+2)	7	6693.3	3407.4