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Estimation of Eeg Signal Dispersion During Seizure Propagation

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Abstract

Localization of the seizure focus in the brain is a challenging problem in the field of epilepsy. The complexity of the seizure-related EEG waveform, its non-stationarity and degradation with distance due to the dispersive nature of the brain as a propagation medium, make localization difficult. Yet, precise estimation of the focus is critical, particularly when surgical resection is the only therapeutic option. The first step to solving this inverse problem is to estimate and account for frequency- or mode-specific signal dispersion, which is present in both scalp and intracranial EEG recordings during seizures. We estimated dispersion curves in both types of signals using a spatial correlation method and mode-based semblance analysis. We showed that, despite the assumption of spatial stationarity and a simplified array geometry, there is measurable inter-modal and intra-modal dispersion during seizures in both types of EEG recordings, affecting the estimated arrival times and consequently focus localization.

Index Terms

Encephalogram; Signal Dispersion; Time-Frequency Analysis; Seizures

1. Introduction

Oscillatory activity in the brain is often referred to as brain waves and analyzed accordingly, but there is considerable debate on whether brain oscillations are true propagating waves [1] [2][3][4]. Propagation of neuronal activity is significantly more complex than wave propagation in other heterogeneous, anisotropic media, such as the earth or the ocean. Spatial variation of electrical activity during seizure evolution is associated with progressive activation of neuronal networks, is a combination of local synaptic and large-scale propagated activity, and depends on both the anatomical and the functional connectivity between brain areas.

The encephalogram (EEG) measures large-scale, aggregate neuronal activity (electrical potentials). EEG signals are non-stationary signals with wave-like dynamics and are modulated by brain state, e.g. sleep, behavior and disease. In brain disorders such as epilepsy, which is believed to result in part from the abnormal hyper-synchronization of neuronal networks, scalp and intracranial EEG recordings are used to diagnose, monitor and localize seizures in the brain. Intracranial (subdural) EEG recordings are invasive but measure more local and smaller scale neuronal activity. Scalp EEG is non-invasive but measures larger scale, 'global' brain activity, attenuated by the presence of the skull. The morphological heterogeneity and anisotropy of the brain causes both types of EEG signals to become distorted and degraded with increasing distances, due to the different arrival times, and thus phase distortions, of different components (modes) of the signal. Thus, the brain can be thought of as a dispersive medium, in which the phase velocity of the recorded oscillations varies with frequency.

Estimating the dispersive characteristics of a medium is particularly important for optimizing source localization. Accurate localization of the seizure focus in focal epilepsy is critical, particularly in drug-resistant cases requiring surgical resection, which relies on precise knowledge of the focus.

EEG signal dispersion during seizure evolution has not been specifically investigated before. This study focuses on the identification and preliminary estimation of dispersion in both scalp and intracranial EEG recordings during seizure propagation, in order to understand this phenomenon at a more local network scale, measured by the intracranial signal and a more global scale, measured by the scalp signal. Our data suggest that it occurs at both scales. Although we do not claim that the brain is a true waveguide, we use techniques often used for dispersion curve estimation in true waveguides, such as the ocean. We demonstrate that it is possible to estimate inter-modal dispersion parameters at specific time intervals during seizure evolution, both from the scalp and intracranial EEG spectra.

2. Overview of Wave Dispersion Estimation Methods

Time-frequency and frequency-wavenumber analyses are typically used to estimate dispersion [5][6]. They involve the analysis of either individual signals to estimate group velocity between source and receiver, or array of signals to estimate the phase velocity between receivers [7][8][9]. Consider a signal $s(t)$ that propagates in a homogeneous dispersive medium from a source to a receiver, undergoes a transformation described by $h(t)$ and is ultimately measured as signal $y(t) = s(t) * h(t)$. Due to dispersion, the source signal undergoes a phase distortion according to the phase of the transfer function: $H(f) = |H(f)|e^{j\phi(f)}$. If the source signal and location are known, $h(t)$ is the 'true' impulse response function between source and receiver. In array processing with unknown source, $h_{n,n+1}(t)$ describes the dispersion between receivers n and $n + 1$. If it can be assumed spatially stationary, $h_{n,n+1}(t) = h(t)$ and $\phi_{n,n+1}(f) = \phi(f)$. In the brain, particularly in long-range propagation, this assumption may not be valid but, for simplicity in this preliminary analysis, we assume spatial stationarity of dispersion. Phase velocity can then be estimated directly from the phase of the transfer function:

$$v_{\phi}(f) = \frac{2\pi f d_{n,n+1}}{\phi(f)} \quad (1)$$

where $d_{n,n+1}$ is the distance between receivers n and $n + 1$. Frequency-wavenumber methods typically used in this estimation are based on the assumption of the validity of the plane wave model [10][11], which may not be meaningful in the context of brain oscillations. In conventional frequency wavenumber (CVFK) estimation, for each frequency ω we compute the cross-spectral matrix $R(\omega) = E(Y(\omega)Y^H(\omega))$, i.e by averaging the signals at all receivers at a particular frequency. Then, the estimator may be written as

$$P(\omega, \vec{k}) = S(\omega, \vec{k}) R(\omega) S(\omega, \vec{k})^H \quad (2)$$

where $S(\omega, \vec{k}) = [e^{j\vec{k}\vec{r}_1} \dots e^{j\vec{k}\vec{r}_M}]^T$ are the steering vectors for wavenumber $\vec{k}$ and frequency ω . There are also variations of this technique, which optimize the estimation and address potential problems associated with energy leakage along lines of constant wavenumber [10].

Spatial autocorrelation methods [12] assume that the wavefield is stationary in both time and space. Given spatio-temporal correlations between spectral densities, the azimuthally averaged spatial autocorrelation $\rho(d, \omega)$ is given by:

$$\langle \rho(d, \omega) \rangle = J_0\left(\frac{\omega d}{v_\phi(\omega)}\right) \quad (3)$$

where $\langle \rho(d, \omega) \rangle$ is the averaged spatial autocorrelation, for a pair of receivers at distance d from each other, and J_0 is the zeroth order Bessel function of the first kind. Thus, we can use the above expression to determine phase velocity $v_\phi(\omega)$. More recently, complex-coherence functions have been used instead of the spatial autocorrelation, to directly estimate $J_0(\omega d)$. Although EEG signals are non-stationary, we may assume that they are 'piecewise' stationary and use a sliding window to sequentially process data segments which can be assumed stationary, as in the short-time Fourier Transform. In this study we computed the complex coherence functions (CCF) of the EEG channel array. Phase velocity is inversely proportional to the derivative of the cross-coherence phase with respect to frequency. We assumed that CCFs can be expressed as the superposition of the Bessel function $J_0(\cdot)$ and an angle-dependent error term [13]. Averaging over angle between the reference receiver and the rest of the array reduced the error term significantly. Thus, the averaged coherence functions were used as the estimate for $J_0(\cdot)$. Examples of cross-coherence functions are shown in Figure 4. We assumed a standard head model and thus our estimates of phase velocity from the intracranial EEG data should be more accurate than those for the scalp data. The intracranial grid covers a smaller area of the head and thus the error in distance between sensors when neglecting the patient-specific head geometry will be smaller than for the scalp data. We did not calculate the error explicitly. In future analyses patient-specific head geometry and measurements may be easily included.

Finally, we also used another phase velocity estimation method typically applied to seismic signals, which involves filtering the data in narrow frequency bands, processing the filtered signals assuming they are non-dispersive and then using semblance velocity analysis to estimate phase velocities in multiple frequency bands [14]. Semblance analysis compares two datasets on the basis of their phase, as a function of frequency. We modified this approach by using dominant EEG modes instead of arbitrarily chosen narrow-band signals. There are several methods for mode extraction, some of which require *a priori* knowledge of the propagating medium. Empirical mode decomposition (EMD) is a technique that sequentially extracts dominant signal components, based on a residual variance criterion for convergence, without assuming linearity or non-stationarity of the signal [15]. The technique has been used for different purposes in the field of epilepsy, e.g. for seizure detection and discrimination of ictal and inter-ictal EEG features [16][17][18]. The original technique sometimes results in mode shapes with amplitude instabilities at the endpoints, and which depend on the choice of the processing window. Using a modified empirical mode decomposition to optimize mode shape and eliminate these potential instabilities, we extracted a subset of nearly orthogonal modes in the time domain [19]. We then estimated the dispersion curves for each of the dominant modes in the EEG spectrum. Although dispersion was clearly evident only in a few EEG channels, we used the entire array in our estimation, to ensure adequate spatial frequency resolution, which is inversely proportional to the number of receivers. Given the higher channel density of the intracranial grid, the spatial resolution of these data were significantly higher.

3. Materials

Scalp EEG recordings used in this analysis were recorded in the Epilepsy Unit at Beth Israel Deaconess Medical Center, Boston MA. Scalp electrodes were arranged according to the 10-20 International Placement System and data were sampled at 200 Hz. The 60 Hz noise and its harmonics, typically seen in the EEG signal, were filtered out using a 2nd order elliptical stop-band filter with 1.5 Hz bandwidth. The data were filtered in both directions to eliminate potential phase shifts associated with the non-zero phase of the filter. A standard model for the head was used to calculate inter-electrode distances. Intracranial EEG data were recorded at Children's Hospital Boston, MA. The 64-channel (8 by 8) grid was placed over the posterior temporal lobe of the left hemisphere, and inter-electrode distance was 5 mm. The data were sampled at 500 Hz and stop-band filtered to remove the 60 Hz noise and its harmonics, using the same filter as for the scalp EEG data.

4. Dispersion During Seizure Propagation

Signal dispersion in background brain oscillations is not always evident in the EEG spectrum and thus cannot be easily estimated [2]. However, during seizure evolution dispersion is often clearly seen in the EEG spectrum, either at seizure onset or later during the ictal interval. Even during seizures, dispersion is identifiable only for a subset of modes, those with large amplitude contributions (dominant) to the EEG signal. Thus, it is more meaningful to conduct this analysis focusing only on these modes. For this purpose, we examined both scalp and intracranial EEG signals during seizures. Figures 1 and 2 show examples of respective recordings from two different patients. Clear dispersion effects in the EEG spectrum could only be identified in a subset of channels, not necessarily due to selective increase in spectral power in those channels during seizures. In addition, signal dispersion was only seen at frequencies above ~ 10 Hz and below ~ 60 Hz.

The 13 s window shown in Figure 1 is a segment of the ictal interval. Yet, the high power dispersion curves could be seen only for ~ 6 s in 3 channels and for a longer interval in the T5-01 channel. Evidently, this depends also on the location of the channel relative to the seizure focus and its primary propagation path. Similarly, in the intracranial recordings, dispersion was evident only in a subset of channels of the subdural grid. Related regions in the spectrogram were narrower than corresponding regions in the scalp EEG spectrogram, but wider in frequency range, spanning below 10 Hz to 60-70 Hz. The 100 s window shown in Figure 2 corresponds to approximately the entire duration of the seizure. Seizure onset may be seen clearly in the spectrum and dispersive effects may be seen shortly after onset (~ 5 s). The dispersive signal duration was significantly longer (~ 30 s). Therefore, there may be several scales of dispersion in the propagating medium, or seizure-dependent dispersive effects which may vary with propagating characteristics of individual seizures. Also, additional attenuation mechanisms may affect the observed larger-scale dispersion in the scalp EEG.

Dominant modes were extracted from EEG signals as described in Section 2. A mode was considered dominant if its amplitude contribution to the broadband signal, i.e., its energy, was significant. An example of a basis of intrinsic mode functions (IMFs) for one channel, from the ictal intracranial recording shown in Figure 2, is shown in Figure 3.

To estimate phase velocity and consequently compute dispersion curves for the EEG data, for both the spatial correlation method and semblance analysis we estimated channel cross-coherence functions, assuming the first channel as the reference. Phase velocity was estimated from the phase of the channel cross-coherence, for the extracted dominant modes of signal pairs.

Channel cross-coherence varied with distance between receivers, time and frequency. In this particular example, cross-coherence between F7-T3 and T4-T6, which are channels diagonally symmetrical in opposite hemispheres, was significantly higher (confidence limits:[0.7,0.82] versus [0.31, 0.47]) than cross-coherence between channels in the same hemisphere and in close proximity to each other, particularly at frequencies below 40 Hz. Thus, this parameter also depends on the particular propagation characteristics of the seizure, including its spatial propagation, constructive versus destructive interference between channels with phase lags, etc. We computed dispersion curves based on our estimates of phase velocity, in the order of 7-12 ms⁻¹, in agreement with previously reported estimates of phase velocity in the cortex [2]. Examples of the fit between estimated curves and data are shown in Figure 5, for selected dominant modes in the data.

5. Discussion

We have presented preliminary results from the estimation of dispersion during seizure propagation, using multidimensional signal analysis of scalp and intracranial EEG recordings. Specifically, we estimated cross-coherence functions, and phase velocity from the cross-coherence phase, between pairs of recording EEG electrodes, for frequencies corresponding to the dominant modes of the EEG signals. We also estimated phase velocities and computed dispersion curves corresponding to these modes using semblance analysis. Instead of arbitrarily selecting a narrowband decomposition of the signals of interest, we extracted their dominant modes and subsequently estimated mode-specific dispersion parameters. We showed that despite assumptions of spatial stationarity and a standard rather than patient-specific head geometry, and consequently potentially inaccurate distances between receivers, inter-modal dispersion was evident in both scalp and intracranial EEG signals during seizures, and computed dispersion curves fitted the data well. The analysis of both types of EEG recordings showed dispersion at potentially different scales in the brain, a more local scale, as identified in the intracranial EEG signal and a global scale, as shown in the scalp signal. This is expected given that local propagation is affected by the local morphology of the brain, whereas global propagation is affected by large-scale structures. In addition, scalp recordings are also further attenuated due to the presence of the skull. In the context of the localization of the epileptic focus, estimation of dispersion is an important first step. If it is not estimated and accounted for prior to inversion, inaccurate estimates of the focus may be obtained. Thus, simulation studies with synthetic data and *a priori knowledge* of the seizure focus can be very useful to estimate errors in localization associated with not accounting for dispersion in the propagating brain.

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Fig. 1.
EEG signal dispersion as seen in scalp recordings in 4 channels during seizure evolution.

Fig. 2. EEG signal dispersion as seen in intracranial recordings in 4 channels during seizure evolution.

Fig. 3.
Mode decomposition of an intracranial EEG signal.

Fig. 4. EEG Channel cross-coherence from semblance analysis, between channel F7-T3 and the other 3 channels shown in Figure 1. Lightest areas correspond to time-frequency regions of highest cross-coherence.

Fig. 5. Estimated dispersion curves superimposed to scalp and intracranial EEG data, for a few selected modes, identified as dominant modes via signal decomposition.