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Dependence of absence seizure dynamics on physiological parameter evolution

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ABSTRACT

A neural field model of the corticothalamic system is applied to investigate the temporal and spectral characteristics of absence seizures in the presence of a temporally varying connection strength between the cerebral cortex and thalamus. Increasing connection strength drives the system into an absence seizure-like state once a threshold is passed and a supercritical Hopf bifurcation occurs. The dynamics and spectral characteristics of the resulting model seizures are explored as functions of maximum connection strength, time above threshold, and the rate at which the connection strength increases (ramp rate). Our results enable spectral and temporal characteristics of seizures to be related to changes in the underlying physiological evolution of connections via nonlinear dynamics and neural field theory. Spectral analysis reveals that the power of the harmonics and the duration of the oscillations increase as the maximum connection strength and the time above threshold increase. It is also found that the time to reach the stable limit-cycle seizure oscillation from the instability threshold decreases with the square root of the ramp rate.

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1. Introduction

Epileptic seizures involve intense, abnormal, synchronous neural firing in the corticothalamic system (Browne and Holmes, 2000; Coombes and Terry, 2012; Engel and Pedley, 1997; Hall and Kuhlmann, 2013; Jirsa et al., 2017; Kim et al., 2009; Kramer et al., 2012; Larter et al., 1999; Liley and Bojak, 2005; Lytton, 2008; Lytton et al., 2005; Penfield, 1933; Robinson et al., 2002; Salek-Haddadi et al., 2006; Schiff, 2012; Velazquez et al., 2006; Wendling et al., 2005). Absence seizures are generalized epileptic seizures, most commonly observed in children (Crunelli and Leresche, 2002; Engel and Pedley, 1997; Proix et al., 2014; Suffczynski et al., 2004; 2005; Vercueil et al., 1998), which involve a sudden loss of consciousness, motionless stare, and cessation of ongoing activities (Browne and Holmes, 2000; Engel and Pedley, 1997; Panayiotopoulos, 1999). The majority of absence seizures last for 20 seconds or less (Panayiotopoulos, 1999).

The characteristic hallmark of absence seizure is bilaterally synchronous “spike and wave” discharges (SWDs) with a frequency of 3–4 Hz (Breakspear et al., 2006; Crunelli and Leresche, 2002; Jirsa et al., 2014; Marten et al., 2009; Panayiotopoulos, 1999; Proix et al., 2014; Lopes da Silva et al., 1997; Suffczynski et al., 2004; 2005; Vercueil et al., 1998; Zhao and Robinson, 2015). A number of authors have investigated the mechanism behind SWD generation (Breakspear et al., 2006; Crunelli and Leresche, 2002; Jirsa et al., 2014; Marten et al., 2009; Panayiotopoulos, 1999; Proix et al., 2014; Lopes da Silva et al., 1997; Suffczynski et al., 2004; 2005; Vercueil et al., 1998; Zhao and Robinson, 2015) by using both neural network (Crunelli and Leresche, 2002; Panayiotopoulos, 1999; Proix et al., 2014; Suffczynski et al., 2004; 2005; Vercueil et al., 1998) and neural field approaches (Breakspear et al., 2006; Marten et al., 2009; Zhao and Robinson, 2015). It is widely considered that the transitions from healthy states to spike-wave discharges occur upon changing corticothalamic connectivity strengths, e.g., increasing excitatory connections between the cortex and the thalamus (Breakspear, 2017; Breakspear et al., 2006; Chen et al., 2014; Destexhe, 1999; Dhamala et al., 2004; Guye et al., 2006; Kim and Robinson, 2007; Luo et al., 2012; Marten et al., 2009; Meeren et al., 2002; Roberts and Robinson, 2008; Robinson et al., 2002; Rodrigues et al., 2009; Tan et al., 2007; Ullah et al., 2015; Wallace et al., 2001; Zhao and Robinson, 2015). Thalamic involvement in the SW generation is also supported by in vivo studies (Andrew, 1991; Prevett et al., 1995; Seidenbecher et al., 1998; Steriade et al., 1998; Steriade and Contreras, 1998; Vergnes and Marescaux, 1992; Voss et al., 2009; Williams, 1953).

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Many modeling studies have been done to find the underlying corticothalamic mechanisms of absence seizures (Breakspear et al., 2006; Chen et al., 2014; Marten et al., 2009; Roberts and Robinson, 2008; Robinson et al., 2002; Rodrigues et al., 2009; Yang and Robinson, 2017; Zhao and Robinson, 2015), as well as oscillatory activity during the pre-ictal to ictal transition in the cortex and the thalamus (Berényi et al., 2012; Godlevsky et al., 2006; Liley et al., 1999; van Luijtelaar et al., 2016; Wendling, 2008). However, the impact of underlying parameter changes on the onset and dynamics has not been studied in detail. The possible physiological reasons behind the variation of the amplitudes of the oscillations with time (Breakspear et al., 2006), which are seen in the clinical EEGs like Breakspear et al., are also needed to be explored.

In this study, we apply a widely used neural field model of the corticothalamic system (Breakspear et al., 2006; Kim and Robinson, 2007; Marten et al., 2009; Roberts and Robinson, 2008; Robinson, 2006; Robinson et al., 2002; 2004; Zhao and Robinson, 2015). Neural field theory (NFT) is a continuum approach that predicts the dynamics of large numbers of neurons (Deco et al., 2008; Pinotsis et al., 2012). This model (Freeman, 1975; Jirsa and Haken, 1996; Nunez, 1974; Robinson et al., 1997) has reproduced and unified many observed features of brain activity, including alpha rhythm (O'Connor and Robinson, 2004; Robinson et al., 2003), age-related changes in the physiology of the brain (van Albada et al., 2010), evoked response potentials (Rennie et al., 2002), arousal state (Abeysuriya et al., 2015) dynamics, and many other phenomena as well as enabling estimation of underlying brain parameters by fitting model prediction to data (Abeysuriya et al., 2014a; 2014b; Breakspear et al., 2006; 2003; Chiang et al., 2011; Kim and Robinson, 2007; Marten et al., 2009; Roberts and Robinson, 2008; Robinson, 2006; Robinson et al., 2002; 2004; 2005; 2001; Rodrigues et al., 2006; Zhao and Robinson, 2015). This model has also been used in seizure studies (Breakspear et al., 2006; Kim and Robinson, 2007; Marten et al., 2009; Roberts and Robinson, 2008; Yang and Robinson, 2017; Zhao and Robinson, 2015).

Several studies have demonstrated that a gradual increase of the connection strength between the cortex and thalamus can provoke seizure like dynamics in a NFT model of the corticothalamic system as a result of a supercritical Hopf bifurcation (Breakspear et al., 2006; 2003; Marten et al., 2009; Zhao and Robinson, 2015). However, the dependence of the seizure on the time course of the connection strength has not been studied in detail. In this study, we implement a smooth flat-topped temporal variation function to vary level, duration, and ramp rate to explore the dependence of the seizure dynamics on these parameters. The aims are to understand the effects of physiological parameters, such as, the maximum connection strength, the rate of change of the connection strength, and the time course of the connection strength on the temporal and spectral characteristics of model seizure dynamics, and how the observed dynamics constrain the underlying physiological parameters. We also intend to see the dependence of the amplitude of the model seizure oscillations on these parameters which possibly could lead us to relate the underlying physiological changes to the variation of the amplitude of the oscillations with time seen in clinical EEG (Breakspear et al., 2006).

The outline of this study is as follows: In Section 2 we present the theory of corticothalamic neural field model, in Section 3 we explain the numerical method, in Section 4 we explore the dependence of seizure dynamics on the temporal variation of connection strength, and in Section 5 we give a summary.

2. Theory

In this section we present a brief description of the corticothalamic neural field model, along with the form of temporal variation of corticothalamic coupling strength.

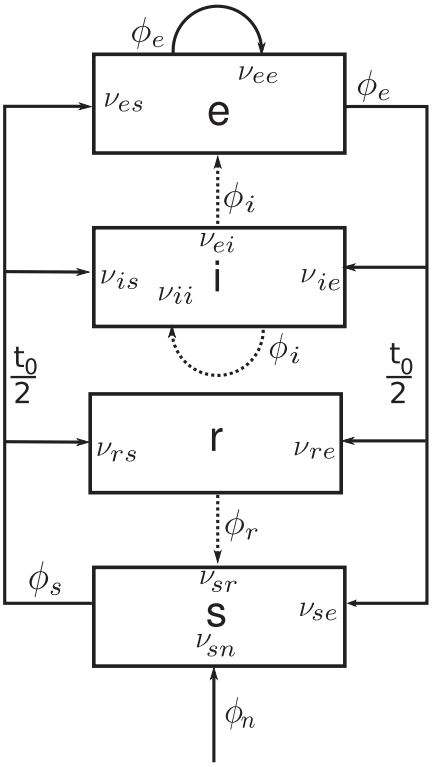

Fig. 1. Schematic diagram of the corticothalamic model system. The neural populations shown are cortical excitatory (e), inhibitory (i), thalamic reticular (r), thalamic relay (s), and n = external inputs. The parameter ν_{ab} quantifies the connection to population a from population b. Inhibitory connections are shown with dashed lines.

2.1. Corticothalamic field model

In order to investigate the dynamics of absence seizure, we use the neural field model of the corticothalamic system seen in Fig. 1. The neural populations are denoted as: e = excitatory cortical; i = inhibitory cortical; s = thalamic relay neurons; r = thalamic reticular nucleus; and n = external inputs. The dynamical variables within each neural population a are the local mean cell-body potential V_a, the mean rate of firing at the cell-body Q_a, and the propagating axonal fields ϕ_a. The firing rates Q_a are related to the potentials V_a by the response function

$$Q_a(\mathbf{r},t) = S[V_a(\mathbf{r},t)], \tag{1}$$

where S is a smooth sigmoidal function that increases from 0 to $Q_{\max}$ as V_a increases from $-\infty$ to ∞, with

$$S(V_a) = \frac{Q_{\max}}{1+\exp[-\pi(V_a-\theta)/\sigma\sqrt{3}]}, \tag{2}$$

where θ is the mean neural firing threshold, σ is the standard deviation of this threshold, and $Q_{\max}$ is the maximum firing rate.

In each neural population, firing rates Q_a generate propagating axonal fields ϕ_a that approximately obey the damped wave equation

$$D_a\phi_a(\mathbf{r},t) = Q_a(\mathbf{r},t), \tag{3}$$

where the spatiotemporal differential operator D_a is

$$D_a = \frac{1}{\gamma_a^2}\frac{\partial^2}{\partial t^2} + \frac{2}{\gamma_a}\frac{\partial}{\partial t} + 1 - r_a^2\nabla^2, \tag{4}$$

where $\gamma_a = v_a/r_a$, r_a and v_a are the characteristic range and conduction velocity of axons of type a, and ∇^2 is the Laplacian operator.

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