Chapter 84
Adaptable Intermittency and Autonomous Transitions in Epilepsy and Cognition

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Abstract In this paper we investigate the mechanisms underlying transitions in epilepsy and cognition by exploring intermittency in simple recurrent neural network models. We demonstrate that neural activity can change dynamical phases without requiring plasticity or reliance on external cues. However, we also demonstrate that the characteristic of the resultant phases can undergo modification with localized parametric alteration. By modifying a single connection in the network we can change the trajectory of the dynamics and thus illustrate that intermittency mechanisms are compatible with more traditional models of ictal transitions that depend on alteration in structural parameters. Finally, we argue that intermittency-based neural transitions coupled with weight adaptation mechanisms can apply beyond epilepsy and might form a foundation for autonomous neurodynamics in biological systems. As such, the implications of these intermittent models may be extended to sleep, emotion, attention and other transitions in cognition seen in autonomous embodied agents.

Keywords Attention · autonomous neurodynamics · computer model · epilepsy · intermittency · neural network · plasticity · seizure

Introduction

How does the brain transition between epilepsy and non-epileptic dynamics? Are these transitions dependent on the external environment? Do these transitions depend on changes to network structure or to constituent unit properties? What about transitions in attention and other cognitive states? Rapid transitions in activity are a fundamental feature of neurodynamics. Epilepsy is a striking example for which the mechanisms of transitions remain largely unknown. Theories regarding transitions in and out of seizures have most often focused on two possible mechanisms: (1) parametric alteration and (2) multi-stable systems. In the first case, the brain

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properties are assumed to change such that a new seizure state – or attractor – is either formed or made more prominent. The parametric change can (a) lead to a bifurcation that directly brings about a seizure or (b) modify the system such that a stimulus that in the past may not have had any discernable effect will now fall within the enlarged basin of attraction and trigger a seizure. Kindling might be an example of such a process. According to the second, related, hypothesis (2) a transition is envisioned as a jump between two or more pre-existing attractors. In this multi-stability scenario, the attractor for a seizure already exists and the ictal phenomenon is brought about when the brain is pushed into the seizure state as the result of either an external or internal perturbation. The perturbation may be a sensory stimulus as in the case of certain reflex epilepsies or it may be random noise in which case the onset of the seizure becomes unpredictable. These scenarios are related in that they posit attractors that correspond to the seizure state. For a review of these attractor-based theories see: [1, 2, 3]. More recently, our group has suggested an alternate possibility in which intermittent transitions are an inherent feature of a network's dynamics [4, 5]. In such networks the activity autonomously switches between two (or more) phases without perturbation from either the environment or another brain structure. We have demonstrated that such dynamics can be sustained in a network even if the structure is static. The implications of such models are that transitions to seizure – and indeed, transitions between any cognitive conditions – may take place even in the absence of environmental input, stochastic fluctuations (noise) or plasticity. Here we expand on this model by showing that although autonomous transitions do not require external input or network alterations, intermittent systems are also compatible with structural change mechanisms. Specifically, we demonstrate that intermittent activity can be modified in a continuous fashion by local synaptic change and illustrate how these structural changes may alter the distribution and trajectory of the intermittent neural activity.

Methods

To explore these intermittent dynamics we used computational models of recurrent networks with random connections and initial conditions. The networks were fully connected including both inhibitory and excitatory weights as well as self-feedback ($w_0..w_j$). The total input (E_i) for a unit (i) was the weighted sum of the activations of the input units (S_j) such that:

$$E_i = \sum_j w_{ij} S_j \tag{84.1}$$

Units had radial basis activation functions (RBF):

$$S(E_i) = e^{(-E_i)^2} \tag{84.2}$$

Simulations included networks of up to 100 units with 10,000 connections and various plasticity algorithms. However, to illustrate that mechanisms require neither complex structural nor plasticity assumptions, we present results for static networks with only 5 units and 25 connections. Network dynamics were categorized using variants of close return and Lyapunov exponent algorithms. For details regarding the analytic approach see [4].

Results

Intermittency in Recurrent Networks

We began by generating random networks and then excluded networks that exhibited simple fix-point and limit-cycle oscillations. Some networks exhibited multistability activity patterns depending on the initial conditions. For a discussion of the distribution of dynamical categories in random networks see [6]. For the purposes of this study we selected networks that exhibited intermittent activity. Figure 84.1a–c illustrates intermittent behaviour in which the activity autonomously switched from laminar to turbulent epochs. Although weight changes were not required for obtaining ictal transitions in any of these simulations, we found that changes in connectivity *could* influence the properties of the intermittent activity in a generally continuous manner. Graphs 1(a), (b) and (c) show a progressive decrease in the duration of laminar periods corresponding to increasing the strength of a single recurrent connection.

The properties of the activity were also modifiable by external input but the intermittency could persist under a wide range of perturbations including ongoing

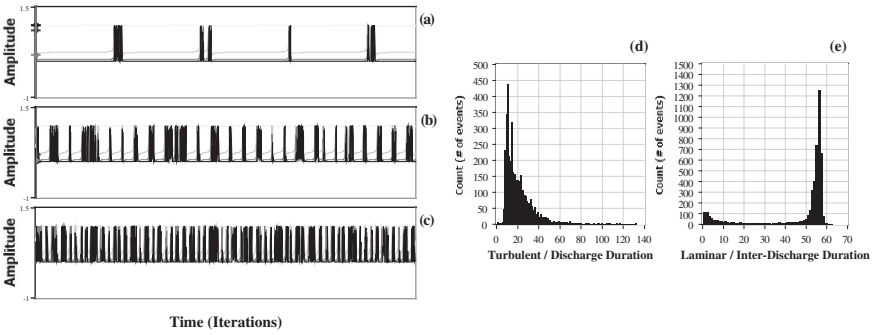

Fig. 84.1 *A*ctivity Traces and Statistics of Intermittent Networks: (a), (b), (c) are the activity traces for three networks exhibiting intermittent activity. Each graph shows the superimposed activity traces of 5 units over 2000 iterations. These graphs illustrate the large change in laminar duration as a result of the modification of a single weight. Histogram (d) is the distribution of the turbulent epochs for the middle network (b). Histogram (e) is the distribution of the laminar durations for the same network

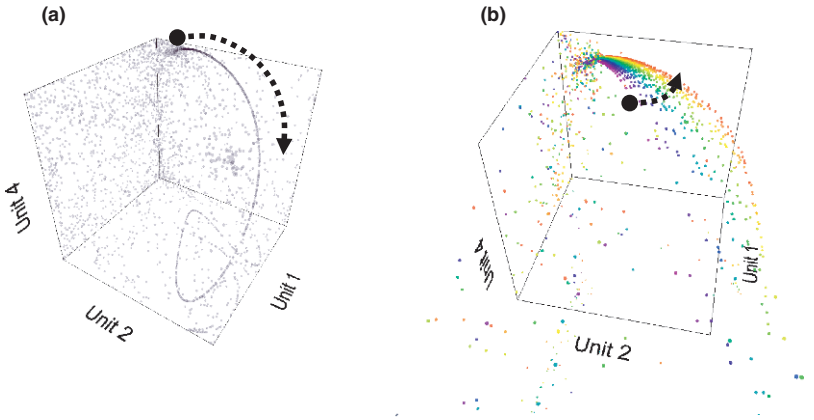

Fig. 84.2 Intermittent neural network activities and alteration of the laminar trajectory with weight change: (a) Illustrates the states of three units over 10,000 iterations in 3-dimensional activity space (or phase space) [4]. (b) Changes in the trajectory of the intermittent activity as a single connection in the network is incremented in steps of 0.1. The output associated with each weight change is represented by a unique color. The arrow highlights the manner in which the laminar trajectory rotates in space as the weight is changed

noise injection and varied initial conditions (not shown). Figure 84.1d illustrates the distribution of the turbulent epochs and 1(e) is the distribution of the laminar durations for the middle trace (b). These event duration histograms show a statistical profile characteristic of Type I intermittency. The turbulent epoch distribution (d) had a sparsely populated long tail. The laminar events distribution (e) had two peaks, a minor one at the short durations and a peak at long intervals marking the maximal length of a laminar phase.

Activity Space and Trajectory Modification with Weight Changes

In order to better illustrate the nature of the changes to the dynamics we plotted the activity in phase space (Fig. 84.2). The cloud of points in Fig. 84.2a corresponds to the turbulent activity and the arrow indicates the trajectory of the laminar activity. Figure 84.2b illustrates changes to the laminar activity trajectory in phase space as a result of changes to connectivity.

Discussion

The network model illustrates that dynamical transitions do not require: (i) a triggering input (including noisy processes) (ii) changes in the intrinsic cell properties nor (iii) ongoing changes to the network structure (plasticity). The model thus raises the possibility that intermittent dynamics need not be a consequence of changes

to synaptic connectivity nor external perturbation but rather could be a recurring event entirely due to intrinsic network properties. The fact that the properties of these transitions are modifiable through synaptic changes demonstrates that these intermittency mechanisms are compatible with a parametric change scenario.

Implications to Epilepsy

The model forwards a novel way to understand autonomous seizure transitions. Once network connectivity is set to exhibit intermittency, ictal events can occur intermittently without further changes or extrinsic triggering. There is no requirement for synaptic or gap junction modification nor changes in individual neuronal properties. However, these intermittency-based autonomous transitions do not preclude the involvement of other mechanisms such as parameter-based bifurcations [1, 2] or multi-stability [3]. Indeed, here we show that parametric weight modulation can directly alter the duration and distribution of turbulent and laminar phases. That is, even small changes in the weights of a network might directly affect the response characteristics of the system. These findings suggest that the appearance of a seizure can be independent of network changes but subtle changes in connectivity could alter seizure duration, interictal duration, and other seizure-related signal features. The model may thus help us understand the factors governing the genesis and abolition of seizure susceptibly. The statistical signatures may suggest new ways of identifying such mechanisms in clinical studies, diagnosis and therapy of epilepsy. For example, we note that if epileptic seizures are induced by random parameter fluctuations or noise-driven transitions between states in multi-stable systems, the transitions may be unpredictable. However, if the transitions are intermittency-based, our model offers the possibility of early warning for the turbulent phase. Using a variant of Lyapunov exponent analysis, we have shown that the spectrum changes when entering the "point of no return" to a turbulent phase [4]. As such, the method indicates that the onset or duration of certain intermittent seizures may be predictable depending on whether the turbulent phase corresponds to the ictal or interictal period.

Implications to Cognition

Although it might be simplest to conceive of the laminar epoch as a quiescent interictal state and the turbulent epoch as representing a seizure, it is the turbulent activity that may correspond best to healthy forms of complex neurodynamics. The distinction between the epochs and the relation of dynamic categories to cognitive states ultimately requires consideration of functional embodied behaviour [6]. Nonetheless, the fact that a simple random network without more complex connectivity assumptions such as small world connectivity can exhibit intermittency illustrates that this is a viable mechanism to implement rapid transition in neurodynamics. The

simulations highlight the possibility that certain transitions in cognition may be best characterized as a continuous trajectory in a system with heterogeneous dynamics rather than alternations between distinct attractors (or states). Although these networks can respond to external stimuli, the ability to implement transition independently of the environment suggests that intermittency could play an important role in autonomous cognitive transitions including changes to attention, alertness and emotions. The model helps show that such transitions do not require plasticity. However, the present study also demonstrates that structural changes, including single synapse and unit modification, can affect intermittency dynamics. The fact that the change is local opens the possibility for biologically plausible learning algorithms. Moreover, the autonomous nature of the transitions coupled with the potential for adaptivity make this a candidate mechanism in embodied biological systems that depend on quick transitions, autonomy and the ability to learn.

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