

# **Desynchronization of rhythmic burst activity in a simulated neural network**

Wim van Drongelen and Kurt E. Hecox

The University of Chicago, Department of Pediatrics, 5841 South  
Maryland Avenue MC 3055, Chicago, IL 60637.

## **Summary**

The electroencephalogram (EEG) or the electrocorticogram (EcoG) is the sum of the contributions of extracellular currents generated by the individual neural elements [4]. Epileptiform activity is generally associated with increased synchronization of electric activity, and indeed, multiple studies show highly coherent activity in neighboring electrodes over the area where seizures occur, e.g. [6]. Non-linear metrics show that EEG and EcoG during, and possibly before, epileptic activity demonstrate a low Lyapunov exponent, a decreasing number of dimensions, and reduced Kolmogorov entropy, e.g. [2], [3]. Electrophysiology of slices of brain tissue are studied under the assumption that the behavior of small networks can provide insight into the activity of the intact brain. This approach may be useful, in spite of the non-linear relationships between activities, considering that many types of seizures are initiated in a small network before propagating to a wider area. With epilepsy control in mind, successful attempts were made to restrict the activity of neural networks by controlling bursts of neural activity

in a slice of brain tissue using an electrostimulator connected to an ‘in-line’ chaos control device [5]. In this study we examine different types of electrostimulation as a tool to stop discharges in a simulated neural network by desynchronization of the individual activities. We tested hyperpolarization, depolarization, and a current proportional to the computed field potential generated by model neurons in a network. The rhythmic bursts in the network are used as a model of epileptiform activity.

Neural elements. The unit of the model network is a Hodgkin-Huxley type membrane model [1]. Each element consists of membrane capacitance (C), ion currents (I) (sodium, potassium, and a leakage channel), and an external current ( $i_e$ ) to simulate direct electric stimulation.

Network structure. The network was represented as a chain of a small number of units ( $< 10$ ) coupled via excitatory synapses. A neuron represented by a membrane element (k) can be coupled to another unit (k+1) by injecting a depolarizing current ( $i_s$ ) in cell k+1 if an action potential (AP) occurs in k. Because the network is closely spaced in this model, the delays in transmission time were mainly determined by the synaptic activity. Additional activity from other areas in the brain was simulated by independent random excitatory and inhibitory activity ( $i_R$ ) on each neuron.

Field potential. We simplify the calculus of the extracellular potential by assuming that the recording electrode is situated in the middle of the neural network. In the case where the electrode position is equidistant to all the neurons, their contribution to the recorded field potential is proportional to the sum of their individual electric activities [4], [7], [8]. The field potential was used to provide a feed-back current ( $i_{FB}$ ) to the network elements. The equation describing the membrane potential ( $V$ ) of one network element in time ( $t$ ) is:

$$C.(dV/dt) + I + i_s + i_e + i_R + i_{FB} = 0$$

Implementation. The model is implemented as a MatLab (The MathWorks Inc, Natick, MA) script file and C programs created from these scripts. The accuracy of simulation is 1/25 ms.

The activity of the network could be synchronized by a depolarization of all cells at the start of a simulation. The cells remained synchronous for a period of time that was determined by the strength of the coupling in the network and the random activity simulating the input from other areas. In some of the cases synchronous network activity transitioned abruptly and

‘spontaneously’ to a desynchronized state. As expected, an overall depolarization of the neurons increased their activity level. This stimulus did not affect synchronization in the network. Overall hyperpolarization of the elements stopped the activity for the duration of the stimulus, however the network’s activity remained synchronized after the stimulus offset. Turning on a feed-back current proportional to the inverted field potential, inhibited and subsequently desynchronized the individual activities. After the offset of the feed-back, the units remained desynchronized. This study justifies a next step: further exploration of a feed-back device to control seizures in animal models of epilepsy.

### **Acknowledgment**

This work was supported in part by the Falk Grant.

### **References.**

- [1] A.L. Hodgkin and A.F. Huxley, A quantitative description of membrane current and its application to conduction and excitation in nerve. J. Physiol. (London) 117 (1952) 500-544.
  
- [2] L.D. Iasemidis, J.C. Sackellares, H.P. Zaveri and W.J. Williams. Phase space topography and the Lyapunov exponent of electrocorticograms in partial seizures. Brain Topography 2 (1990) 187-201.

- [3] K. Lehnertz and C.E. Elger. Can epileptic seizures be predicted? Evidence from nonlinear timeseries analysis of brain electric activity. *Phys. Rev. Lett* 80 (1998) 5019-5022.
- [4] P.L. Nunez. Electric fields of the brain. Oxford University Press (New York, 1981)
- [5] S.J. Schiff, K. Jerger, D.H. Huong, T. Chang, M.L. Spano and W.L. Ditto. Controlling chaos in the brain. *Nature* 370 (1994) 615-620.
- [6] V.L. Towle, R.K. Carder, L. Khorasani and D. Lindberg. Electrocorticographic coherence patterns. *J. Clin. Neurophysiol.* 16 (1999) 1-20.
- [7] R.D. Traub, J.G.R. Jefferys, R. Miles, M.A. Whittington and K Toth. A branching dendritic model of a rodent CA3 pyramidal neurone. *J. Physiol* (London) 481 (1994) 79-95.

[8] H.R. Wilson and J.M. Bower. Cortical oscillations and temporal interactions in a computer simulation of piriform cortex. *J. Neurophysiol.* 67 (1992) 981-995.