Abstract View

THE EFFECTS OF LESIONS ON DYNAMICS IN NETWORK MODELS OF EPILEPSY

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Acute trauma and pathogen-related cell death can result in large scale changes in neural activity. Changes to surviving cells are often postulated as the pathophysiological mechanisms. Using network models we demonstrate that alterations in connectivity may be sufficient to cause system-wide changes in dynamics even if intrinsic properties of surviving cells remain constant. The models consisted of single or two-layer networks of up to 10,000 elements. For a range of parameters, isolated pairs of excitatory and inhibitory cells created oscillatory units with bursting-like behavior; the addition of full lateral local connectivity within excitatory and inhibitory layers resulted in a quiescent state. Under these conditions large-amplitude local stimulations and small-amplitude spatially distributed activations generated short-term bursting that quickly dissipated. However, when a small lesion was introduced the local dampening effects were lost. Removal of excitatory units effectively reduced input to inhibitory cells which led to disinhibition and bursting. Once these oscillations were initiated at the lesion boundary they progressively increased in amplitude spreading to adjacent non-boundary cells and eventually triggered a system-wide hyper-excited oscillatory state. The introduction of the lesion thus caused a change in the bifurcation point effectively reducing discharge threshold. These observations suggest that network connectivity factors may be sufficient to cause post-traumatic epilepsy and may also underlie inherited forms of epilepsy. These principles may similarly account for changes in activity patterns in neurodegenerative diseases as a consequence of cell death and the creation of micro-lesions.

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