Abstract View

COMPUTATIONAL MODEL OF SEIZURES INDUCED BY CORTICAL DEAFFERENTATION.

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Partial cortical deafferentation in vivo induces an increase in intrinsic excitability that leads to seizures (Topolnik et al., 2001 SFN abstract). Immediately after the cortical undercut activity was reduced, but 2-3 hours later the intrinsic excitability of cortical neurons was enhanced, which contributed to the generation of paroxysmal discharges. In vitro evidence suggests that chronic deafferentation may induce changes in the intrinsic excitability of neurons and synaptic efficacy. Chronic blockade of activity in cultured cortical neurons results in increased Na^+ and decreased K^+ conductances (Desai et al., 1999) and increased efficiency of excitatory synapses between pyramidal cells (Turrigiano et al., 1998). We explored the consequences of increased excitability in computational models of neocortex including pyramidal cells (PY) and interneurons (IN). In the active state PY cells fired asynchronous spike trains. Abolition of excitatory inputs to a region of the network strongly reduced activity in that region. Following an increase in Na^+ conductance and/or decrease in K^+ conductance of the PY cells and enhancement of excitatory connections between PY cells, activity was reinitiated in the form of repetitive high-frequency burst discharges that were strongly synchronized in local subpopulations. The 1-3 Hz frequency of these paroxysmal-like discharges depended on the level of excitability, spontaneous activity and synaptic depression. The activity pattern either remained localized or traveled into the active network.

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