19. Seizure-Induced Neuronal Injury: Vulnerability to Febrile Seizures in an Immature Rat Model

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Febrile seizures are the most common type of seizure in young children. Whether they induce death of hippocampal and amygdala neurons and consequent limbic (temporal lobe) epilepsy has remained controversial, with conflicting data from prospective and retrospective studies. Using an ap-propriately aged rat model of febrile seizures, the acute and chronic effects of hyperthermic seizures on neuronal integrity and survival in the hippocampus and amygdala were investi-gated via molecular and neuroanatomical methods. Hyper-thermic seizures—but not hyperthermia alone—resulted in numerous silver-stained neurons in discrete regions of the limbic system. Within 24 hours of the seizures, a significant

proportion of neurons in the central nucleus of the amygdala and in the hippocampal CA3 and CA1 pyramidal cell layer were affected. These physicochemical alterations of hip-pocampal and amygdala neurons persisted for at least 2 weeks but were not accompanied by significant DNA frag-mentation, a marker of apoptotic cell death, as determined by in situ end labeling. By 4 weeks following the seizures, no significant neuronal dropout in these regions was evident. In conclusion, in the immature rat model, hyperthermic sei-zures lead to profound yet primarily transient alterations in neuronal structure. Whether these neuronal alterations have long-term electrophysiological sequelae is under current in-vestigation.

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