Pediatric Neurology Part I: Chapter 46. Epileptogenesis in the developing brain (Handbook of Clinical Neurology)

Claude G. Wasterlain, David S. Gloss, Jerome Niquet, Amy S. Wasterlain

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The neonatal brain has poorly developed GABAergic circuits, and in many of them GABA is excitatory, favoring ictogenicity. Frequently repeated experimental seizures impair brain development in an age-dependent manner. At critical ages, they delay developmental milestones, permanently lower seizure thresholds, and can cause very specific cognitive and learning deficits, such as the permanent impairment of neuronal spatial maps. Some types of experimental status epilepticus cause neuronal necrosis and apoptosis, and are followed by chronic epilepsy with spontaneous recurrent seizures, others appear relatively benign, so that seizure-induced neuronal injury and epileptogenesis are highly age-, seizure model-, and species-dependent. Experimental febrile seizures can be epileptogenic, and hyperthermia aggravates both neuronal injury and epileptogenicity. Antiepileptic drugs, the mainstay of treatment, have major risks of their own, and can, at therapeutic or near-therapeutic doses, trigger neuronal apoptosis, which is also age-, drug-, cell type-, and species-dependent. The relevance of these experimental results to human disease is still uncertain, but while their brains are quite different, the basic biology of neurons in rodents and humans is strikingly similar. Further research is needed to elucidate the molecular mechanisms of epileptogenesis and of seizure- or drug-induced neuronal injury, in order to prevent their long-term consequences.

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