Hippocampal seizure

The association between epilepsy and the hippocampus is well known and important. Mesial temporal lobe epilepsy with hippocampal sclerosis is a syndromic diagnostic entity and indeed a quite common one. There are different theories on the pathophysiological pathways, as the hippocampus is often involved in seizures, even if they are not generated there. Whether hippocampal sclerosis is a cause or the effect of seizures is a subject of ongoing debate, but the predominant opinion is that seizures probably do not cause relevant hippocampal volume loss in the mature brain. A diagnosis of epilepsy with hippocampal sclerosis is made based on typical semiological signs and symptoms, interictal and ictal EEG findings, cerebral imaging, and neuropsychological testing. Antiepileptic medication is indicated as a first-line treatment. Should the epilepsy prove to be medically intractable, which is commonly the case in these patients, an early evaluation regarding epilepsy surgery must be performed. Different epilepsy surgery techniques are available, from minimal ones like the selective amygdalohippocampectomy to more extensive ones like additional temporal lobe resection. Postoperative results concerning seizures and neuropsychological outcomes are very encouraging and depend on various predictive factors. Alternative procedures like stereotactic radiofrequency amygdalohippocampectomy and hippocampal stimulation are currently being assessed, partly with very promising results [1].

Repeated seizure activity can lead to long-term changes in seizure dynamics and behavior. However, resulting changes in brain-wide dynamics remain poorly understood. This is due partly to technical challenges in precise seizure control and in vivo whole-brain mapping of circuit dynamics.

Choy et al. developed an optogenetic kindling model through repeated stimulation of ventral hippocampal CaMKII neurons in adult rats. They then combined fMRI with electrophysiology to track brain-wide circuit dynamics resulting from non-afterdischarge (AD)-generating stimulations and individual convulsive seizures. Kindling induced widespread increases in non-AD-generating stimulation response and ipsilateral functional connectivity and elevated anxiety. Individual seizures in kindled animals showed more significant increases in brain-wide activity and bilateral functional connectivity. Onset time quantification provided evidence for kindled seizure propagation from the ipsilateral to the contralateral hemisphere. Furthermore, a core of slow-migrating hippocampal activity was identified in both non-kindled and kindled seizures, revealing a novel mechanism of seizure sustainment and propagation [2].

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2)
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