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Prolonged febrile seizures: neuroanatomical and functional consequences

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Rationale and Objectives
Febrile seizures are common, affecting 2–5% of infants and young children worldwide (1–3). The relationship of childhood febrile seizures to adult temporal lobe epilepsy (TLE) has remained a focus of intense controversy (see 4–7 for brief recent reviews): Whereas prospective epidemiological studies have not shown a progression of febrile seizures to TLE, retrospective analyses of adults with TLE have demonstrated a high prevalence (30–60%) of a history of prolonged (longer than 10–15 minutes) febrile seizures during early childhood, suggesting an etiological role for these seizures in the development of TLE. Specifically, neuronal damage induced by febrile seizures has been suggested as a mechanism for the development of mesial temporal sclerosis, the pathological hallmark of TLE. However, this high correlation should not be taken to indicate a causal relationship, and alternative mechanisms may exist for the correlation of prolonged febrile seizures and TLE. These involve pre-existing, genetic or acquired, functional or structural neuronal changes, that may underlie both the prolonged febrile seizures and the subsequent TLE (see diagram):

Alternative I:
Normal brain → Febrile seizures → neuronal damage → TLE

Alternative II:
Pre-existing injury/lesion → fever-triggered seizure = first sign of TLE
exchange the hippocampal circuit to promote the hippocampal expression of LTP

The hippocampal expression of LTP

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Further investigations that may provide a better understanding of the molecular and cellular mechanisms underlying these seizure types are necessary. The possibility of surgical loss of hippocampal tissue in the presence of over-active CA3 CA1 circuits, as well as the impact of excitation-inhibition imbalance on hippocampal circuitry, warrants further investigation.

References


