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Febrile convulsions, rat brains, and scientific pessimism

Febrile convulsions are, by and large, benign. There is now a good deal of evidence to show that for the vast majority of affected children the prognosis is excellent. Why then does a commentary in *Nature Medicine* (1999;5:871–2) have the title “Febrile convulsions: a ‘benign’ condition?” It must be, surely, that some scientific advance has now shown that these children are at a considerable, previously unforeseen, risk. Not so. Not so at all.

What produced this commentary is an article from California (Kang Chen and colleagues. *Ibid*: 888–94) describing experiments on hyperthermia in young rats. These rats, when exposed at the age of 10 days to hot air, almost all developed mainly tonic seizures when their core temperature reached 40–41.5°C. After such seizures, which lasted for about 23 minutes, the rats were decapitated and electrophysiological studies were performed on brain slices. They showed that after hyperthermic seizures there was a presynaptic increase in inhibitory transmission in the hippocampus. The inhibition was reversed by a GABA inhibitor and did not occur in rats subjected to hyperthermia but given pentobarbital to prevent seizures. Subsequent experiments suggested that the changes were brought about by increased activity of cyclic AMP dependent protein kinase. The inhibitory changes lasted into the animals' maturity (10 weeks after the induced seizures).

Increased inhibition, of course, might be a good thing and explain why children who have had febrile convulsions are not particularly prone to epilepsy. Chen and colleagues, however, point to evidence that increased inhibitory activity may lead to neuronal synchronisation, which could have a paradoxical convulsant effect. There are, however, differences between clinical febrile convulsions and these experimental convulsions. For instance, febrile convulsions in children are rarely caused by externally induced hyperthermia; perhaps the experiments should be repeated with pyrogen induced fever. But the proof of the pudding . . . Which brings us back to my first two sentences.

Chen and colleagues conclude that their data “do not support the prevalent view of the ‘benign’ nature of early life febrile convulsions”. I confess to being flummoxed by that conclusion, unless they have redefined the word benign. (**Benign** *adj* (from Latin, *bene* well and *gignere* to produce): a word used by neuroscientists to mean not producing any detectable change in the rat brain.) Could it be that the writers of scientific papers are more motivated to pessimism than to optimism? Perhaps pessimism pays.

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