25 Years Ago in the Canadian Journal of Neurological Sciences

KINDLING
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SECONDARY EPILEPTOGENESIS IN FROG FOREBRAIN: EFFECT OF INHIBITION OF PROTEIN SYNTHESIS

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Summary: Secondary epileptogenesis was induced in the hippocampal cortex of the paralyzed bullfrog by means of localized, unilateral, intermittent electrical stimulation (kindling). Stimuli were designed to yield a brief but definite after-discharge. In control animals a progressive increase in after-discharge duration occurred at the 1^0 (stimulated) site and then at the 2^0 site (contralateral hippocampus). Spontaneous epileptiform potentials (SEPs) occurred between stimuli, eventually independently on both sides. Cycloheximide (50mg/kg) caused 88-99% reduction in protein synthesis, measured by ^{14}C-leucine incorporation into brain protein. Cycloheximide-treated animals revealed no evidence of progressive prolongation of after-discharge duration when subjected to the kindling procedure ($p=0.1205x10^{-7}$). SEPs were reduced in the cycloheximide-treated animals, and confined to 1^0 hemisphere ($p=0.6x10^{-10}$).

Since cycloheximide did not disturb normal electrogenesis or disrupt the after-discharges, this experiment distinguishes processes dependent upon electrical events from those requiring macromolecular synthesis. Protein synthesis seems critical to the emergence of spontaneous and autonomous epileptiform behavior of neural aggregates.

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PRIMARY AND "TRANSFER" SEIZURE DEVELOPMENT IN THE KINDLED RAT

W. McIntyre Burnham

Summary: An investigation was made of both primary and "transfer" kindling as they occur in ipsilateral limbic sites. Primary kindling was found to involve progressive growth of after-discharge (AD), propagation and convulsive behavior. It was noted that AD growth did not take place gradually but occurred in sudden, large increments. "Transfer" (a significant acceleration of secondary kindling) was found at every secondary limbic site. It was associated with the early appearance of full-blown ADs, super-normal propagation, and well-developed seizures. The post-transfer interference of primary site function previously reported by Goddard et al was also found, but it occurred in significant amounts only after transfer kindling of the amygdala. It is believed that the data offer some support for both of the hypothetical mechanisms of transfer which have been proposed.

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Split-brain Rat: Transfer and Interference of Kindled Amygdala Convulsions

Dan C. McIntyre

Summary: Rats were subjected to varying degrees of commissurotomy, followed by implantation of a bipolar electrode into each amygdala. After the kindling of six convulsions at one electrode (primary site), the procedure was applied to the contralateral amygdala (secondary site). Convulsions were observed to develop more rapidly, independent of the degree or kind of transection. After six secondary site convulsions, the primary site was retested and convulsion-triggering was blocked, except in animals with transection of the rostral portion of the corpus callosum (CC).

Collectively, the data indicate: (i) amygdala kindling develops a lasting trace which operates through the midbrain or brainstem; (ii) kindling from a second site utilizes this trace; (iii) a series of six convulsions produces negative after-effect which manifests itself at the convulsion level via the anterior CC; (iv) the anterior CC is important in determining the laterality of the forelimb clonus; and (v) the inter-amygdala propagation of after-discharge is blocked by the combined sectioning of the anterior CC and the anterior commissure.

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For brief prescribing information see page A-37

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