MOOD DISORDERS: PROTECTION OF THE HYPERACTIVE BRAIN OR A RISK FACTOR?

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Depression and anxiety are the most frequent psychiatric disorders identified in patients with epilepsy. Despite the greater prevalence of anxiety and depression in patients with epilepsy, the causes, nature and mechanisms of the relationship between these mood disorders and epilepsy remains poorly understood, partly due to the lack of proper animal models.
The main objectives of the present study were threefold: first, to explore whether emotional behavior, elicited by stimulation of the hypothalamus, can influence seizure development during kindling epileptogenesis; second, to determine whether such stimulation can alter the expression of generalized convulsions when the fully epileptic syndrome has been established beforehand; third, Variation of seizure activity in nondepressive and endogenically depressive animals following injection of antidepressants.
Experiments were conducted in Wistar (n=24) and audiogenic seizure prone (n=18) rats. Temporal lobe epilepsy animal model (kindling) was used.
The present study shows that the emotional behaviors (anxiety) elicited by stimulation of the dorsomedial hypothalamus can suppress the development of generalized motor limbic convulsions during epileptogenesis, as well as dampen seizure expression in already established limbic epilepsy. It appeared also that after depressants administration a significant enhancement of both EEG and behavioral seizure reactions occurred in nondepressive and endogenically depressive rats.
We assume that the emotional disturbances can be considered as the emergence of instinctive behavior with an adaptive significance of defense and as a by product of the inhibitory processes that build up to protect against the future occurrence of seizures.
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