Does Hippocampus Control Thalamo-Cortical Oscillations? An EEG Study in Genetically Epileptic Rats.

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Sensory information, passing from peripheral receptors to the primary sensory neocortical regions, relay in the specific thalamic nuclei. The information flow is blocked when thalamic relay neurons switch to the bursting mode firing. In the EEG this state is characterized by the presence of light slow wave sleep and sleep spindles, generated in the thalamo-cortical circuitry. The bursting mode of thalamic neurons is modulated by inputs from the reticular formation in the brainstem, cortical and forebrain projections. The limbic system including the entorhinal cortex and hippocampus is considered not to be involved in the control of this arousal-sleep related system. However, we have recently found that WAG/Rij rats, which have a low threshold for self-induced occurrence of thalamo-cortical oscillations (sleep spindles and spike-wave discharges) [1], exhibit a low threshold for the spread of electrically induced afterdischarges into the limbic system [2]. Besides, an inverse correlation between the individual threshold of limbic afterdischarges and the occurrence of spike-wave discharges was found.

The present study was aimed to investigate whether acute local intrahippocampal administration of progesterone might affect the occurrence of SWDs.

Male WAG/Rij rats (n=20) were equipped with permanent EEG electrodes and bilateral cannulas in the CA3 region of the dorsal hippocampus. One week later rats received either progesterone (2 μl; 5 mg/ml) or 45% γ-cyclodextrin (CD; 2 μl), or a sham injection. The data showed that both progesterone injection and CD administration resulted in a reduction of spontaneous SWDs during 60 min (for CD) and 120 min (for progesterone) after administration. There was no decrease in the occurrence of SWDs after a sham injection. These data give the first evidence to suggest that the hippocampus might be involved in the regulation of oscillations generated in the cortico-thalamo-cortical network and imply an interaction between the limbic and the arousal-sleep related system.

References:

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