

019. EEG ACTIVITY IN DEEP SLEEP AND ANESTHETIC COMA. WHAT IS DIFFERENT?

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The necessity of understanding basic mechanisms of sleep, both at the cellular and molecular level, has imposed the necessity of a suitable model. Anesthesia emerged as the most prominent one, due to a series of common features such as: loss of consciousness, diminished reactivity to noxious stimulation, recuperative properties, and, above all, the ability to produce electroencephalographic (EEG) patterns similar to the ones recorded during sleep. The use of anesthesia for the study of sleep elicited important breakthroughs in exposing the cellular mechanisms of cerebral networks during sleep. However, the above-mentioned model has notable limitations that will be discussed during the talk. First, sleep is a natural process occurring either actively or passively as a function of a given neuromodulating set point. Anesthesia acts through activation/blockage of various receptor/transporter sites on neurons and glial cells. There are different classes of anesthetics having distinct effects on different receptor categories (GABA, glutamate, etc.). Each of them tends to produce a given pattern of activity at the global level of the EEG. This results from the intimate action of the anesthetic on various cerebral structures (cortex, thalamus, brainstem, spinal cord). The reactivity of the brain is determined by the blood concentration of the used anesthetic, leading to a given degree of deafferentation. At difference, natural sleep is a self-centered process meant to release/recuperate a homeostatic pressure built up during the preceding wakefulness. Finally, the end of sleep occurs either at the end of this regulatory process or at any significant sensory stimulation, while waking from anesthesia is only allowed when blood levels of the agent drop below a threshold blood concentration.

020. BEHAVIOURAL PATTERN AND HIPPOCAMPAL REORGANIZATION IN RATS WITH TEMPORAL LOBE EPILEPSY

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Epilepsy is one of the most common neurological diseases and it is characterized by the reoccurrence of seizures with variable severity and frequency. One of its most frequent form is temporal lobe epilepsy (TLE) during which the reorganization of the neuronal network, changes in the number and function of individual neurons and the consecutive modification of the electrical properties of both the cells and the network leads to the synchronized firing of a large number of neurons and as a result to reoccurring seizures. The burden of epilepsy is actually more than having seizures as the disease is frequently associated with comorbid cognitive and behavioral problems.

In order to investigate the myriad of facets of this disease we induced TLE in juvenile Wistar rats by injection of pilocarpine. The animals were continuously monitored using a video system in order to establish seizure pattern and behavior, then the animals underwent cognitive testing using Orton's 8 arm radial-maze. Later the animals were sacrificed and Nissl staining as well as triple-immuno-labeling was performed in an attempt to identify changes regarding pyramidal cells as well as the major types of interneurons of the hippocampus. Spontaneous recurrent seizures developed in animals in average after 14 days. The epileptic animals' working memory was affected without significant reduction in the reference memory. The pyramidal cells of CA1 area almost completely disappeared, nevertheless the seizure pattern and the hippocampal sclerosis could not be correlated. Basal inhibition, created by perisomatic and dendritic interneurons was maintained, however the reduction in the number of the O-LM cells and Ivy cells may be responsible for the hyperexcitability of the network.

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