

Epidural Electrode Implant-induced Glial Response and Neuronal Modulation in the Pilocarpine Mouse Model of Epilepsy

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Introduction

Epilepsy is a neurological disorder that affects approximately 50 million people worldwide, characterized by recurrent and unpredictable seizures resulting from abnormal electrical activity in the brain. The pathophysiology of epilepsy involves a complex interaction between neurons, glial cells, and the extracellular matrix. These interactions often lead to long-term changes in brain structure and function. Understanding these processes in detail is crucial for developing new therapeutic strategies for managing epilepsy. One promising avenue of research is the use of epidural electrodes, which are implanted on the surface of the brain to both monitor and modulate neuronal activity. Epidural electrodes have become increasingly important in neurophysiological studies, particularly in animal models of neurological diseases like epilepsy. These electrodes allow for the non-invasive recording of brain activity and can be used to deliver electrical stimulation, a method that has shown promise in controlling epileptic seizures [1].

Description

The pilocarpine model of epilepsy is one of the most widely used methods for inducing seizures in rodents. Pilocarpine, a muscarinic acetylcholine receptor agonist, is administered to animals to induce Status Epilepticus (SE), a prolonged seizure that often results in neuronal injury and subsequent epilepsy. SE in the pilocarpine model typically leads to the development of Spontaneous Recurrent Seizures (SRS) after a latent period of several weeks. This model closely mimics human temporal lobe epilepsy, which is characterized by hippocampal sclerosis, neuronal loss, and the development of abnormal excitability in the brain. The pilocarpine model of epilepsy involves several stages: initial induction of status epilepticus by pilocarpine injection, the latent period during which neuronal and glial changes occur, and the chronic phase marked by recurrent spontaneous seizures. One of the hallmark features of the pilocarpine model is the hippocampal damage that occurs, particularly in the CA1 and CA3 regions. This neuronal injury contributes to epileptogenesis, the process by which a normal brain becomes prone to developing seizures [2].

Epidural electrode implantation is a technique commonly used in both animal models and clinical settings for monitoring and modulating brain activity. These electrodes are typically placed on the surface of the cortex, where they can record Local Field Potentials (LFPs) or provide electrical stimulation to specific brain regions. In research settings, epidural electrodes are often used to examine the effects of electrical stimulation on seizure control and to study the neurophysiological changes associated with epilepsy.

The implantation of epidural electrodes involves the placement of metal or conductive materials on the surface of the brain, usually through small surgical incisions. This procedure can induce both mechanical and inflammatory responses in the brain, with glial cells being particularly sensitive to injury. The glial response to electrode implantation has been well-documented in a variety of studies, showing that glial activation can lead to changes in the local microenvironment, including alterations in ion homeostasis, inflammation, and extracellular matrix remodelling. These changes may, in turn, affect neuronal activity and the overall dynamics of seizure generation and propagation [3].

Glial cells play a critical role in maintaining the integrity of the CNS, responding to injury, and modulating neuronal activity. When epidural electrodes are implanted, they can disrupt the normal functioning of glial cells in several ways. The mechanical disruption caused by the electrode insertion can lead to immediate activation of astrocytes and microglia. These glial cells, which normally maintain homeostasis and support neuronal function, become reactive in response to injury. Astrocytes, the most abundant type of glial cell, are involved in regulating the blood-brain barrier, ion balance, neurotransmitter uptake, and synaptic function. Upon activation, astrocytes can release pro-inflammatory cytokines and chemokines, which can exacerbate neuronal damage and contribute to epileptogenesis. Microglia, the resident immune cells of the CNS, plays a similar role by sensing and responding to injury. Their activation can result in the release of pro-inflammatory mediators that further influence neuronal excitability and promote the development of epilepsy [4].

Neuronal modulation refers to the alteration of neuronal activity through various mechanisms, including electrical stimulation. Epidural electrode implantation allows for the direct modulation of cortical circuits, which can influence seizure activity. The ability to control or suppress seizure activity through electrical stimulation is a key component of neuromodulation therapies for epilepsy, such as Deep Brain Stimulation (DBS) and Responsive Neurostimulation (RNS). Electrical stimulation through

epidural electrodes can have both excitatory and inhibitory effects on neuronal circuits, depending on the parameters used (e.g., frequency, intensity, and duration of stimulation). In epilepsy, the goal of neuronal modulation is to restore the balance between excitatory and inhibitory signaling, which is often disrupted in epileptic brain regions. By using epidural electrodes to apply targeted stimulation to specific brain regions, it is possible to influence the local excitability of neurons and potentially reduce the occurrence of seizures [5].

Conclusion

The implantation of epidural electrodes in the pilocarpine mouse model of epilepsy provides a valuable platform for studying the complex interactions between neurons and glial cells in the context of seizure generation and modulation. This research highlights the important role of glial cells in the brain's response to injury, including the mechanical disruption caused by electrode placement. The activation of astrocytes, microglia, and oligodendrocytes following electrode implantation can lead to significant changes in the local environment, which may impact neuronal excitability and the overall dynamics of epilepsy. Furthermore, the ability to modulate neuronal activity through electrical stimulation opens new avenues for therapeutic interventions aimed at controlling seizures. However, the effectiveness of electrical stimulation may be influenced by the glial response to electrode

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implantation, which can either enhance or inhibit the desired effects on neuronal circuits. Understanding these interactions is crucial for optimizing the use of neuromodulation techniques in the treatment of epilepsy.

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Conflict of Interest

There are no conflicts of interest by author.

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