Utilizing Animal Models to Monitor ECG in Post-Traumatic Epilepsy

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Introduction

Studies on Post-Traumatic Epilepsy (PTE) in large animal models are scarce. Neocortical PTE has been better understood thanks to recent developments in neocortical microscopy. Nevertheless, it is incredibly difficult to induce believable neocortical PTE in rodents. Hence, large animal models that expand neocortical PTE can also offer helpful insights that may yet be better suited to human patients. Long-term video EEG recording is necessary because to the lengthy latent periods of gyrencephalic species. Here, we provide documentation of a fully subcutaneous EEG implant in freely moving pigs for up to fourteen months during epileptogenesis following bilateral cortical effect accidents or phantom surgical procedures. The advantages of this device include the availability of an easily installed, commercially available device, a low failure rate following surgical EEG implantation, radiotelemetry that allows continuous tracking of freely moving animals, excellent video to EEG synchronisation, and a high signal to noise ratio. The accretion of cranium bone, which entirely encased a portion of cranial screws and EEG electrodes, and the inability to arrange the EEG electrode array are the hazards of this device on this species and age. These dangers can be reduced by employing implants for a larger meaningful montage and by using splicing a subdural electrode strip to the electrode leads so that cranium growth is less likely to interfere with long-term signal capture. Researchers studying epileptogenesis in PTE can benefit. from this commercially available gadget on this bilateral cortical effect swine version.

PTSD-Related Epilepsy

Traumatic Brain Injury (TBI) has a significant impact on patients' longevity, and those who survive the initial phases of TBI typically have an elevated risk of later-life disability and comorbidity. In this situation, Post-Traumatic Seizures (PTS) and Post-Traumatic Epilepsy (PTE), two disabling sequelae of traumatic brain injury, are frequent. PTS is categorised as "Early" Post-

Traumatic Epilepsy (EPTS) if it starts within 7 days of the event and "Late" Post-Traumatic Epilepsy (LPTS) if it starts more than 7 days later, depending on when it starts. It is categorised under. The distinctions between the underlying mechanisms and the risk of more seizures are reflected in this cutoff. A main damage mechanism linked to EPTS5, sometimes referred to as acute symptomatic seizures, temporarily reduces the seizure threshold. Instead, LPTS is defined by enduring neurobiological changes brought on by secondary injury, and the likelihood of further seizures is determined by a biochemical cascade of epilepsy development pathways.

The International League Against Epilepsy (ILAE), which recently redefined clinically what constitutes epilepsy, now considers LPTS to be epileptic if there is a risk of subsequent seizures following one unprovoked seizure that occurs more than seven days after a traumatic brain injury. For, it is high enough. Hence, PTE and LPTS are frequently used as synonyms. While PTE accounts for 10%–20% of symptomatic epilepsy in the general community and 5% of all epilepsy, the overall incidence of PTE among inpatients is only about 3%-5%. Acute seizures have a significant impact on the progression of new brain injury. Particularly, EPTS seems to raise the likelihood of PTE development and increase morbidity and mortality in the initial phases following TBI [1-3].

Taking into account all of these variables, clinical practise frequently employs early post-TBI seizure prevention, with various degrees of success. It has been a subject of study for decades as a result. Antiepileptic Medications (ASM) have been shown to be effective in preventing EPTS, but there is no evidence to support their superiority to LPTS and PTE. In fact, the use of preventative therapy is advised by the most recent Brain Traumatic Foundation Guidelines 20 to lower the risk of EPTS within 7 days of severe TBI. Before to its problems, phenytoin was the Medication of choice for prophylaxis; however, levetiracetam is now a more popular substitute [4-6].

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