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Short Communication

The Derailment of Sleep Homeostatic Plasticity may Lead to System Epilepsies

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DESCRIPTION

The article provides an overview on the epileptic network concept and its manifestations in system epilepsies. The common concept of epileptogenesis is realized by the upregulation of large physiological brain functions (networks), based either on aquired brain damages or genetic abnormalities.

We demonstrate here these theoretical lines validity through description the transformation of several major brain functions to epileptic working mode, pointing out the essential role of NREM sleep functions and their homeostatic regulation in epileptogenesis.

This approach is an extension of the work of Beenhakker et al., demonstrated that normal brain circuits provide a template that epileptic circuits use to "generate seizures", or in other words: "Normal and epileptic brain circuits share common features" [1]. In their emblematic work these authors demonstrated the epileptic transformation on two epileptic syndrome: Medial temporal lobe epilepsy and absence epilepsy.

Through the recognition of functional seizure triggers like putting into motion certain brain systems as for example falling to sleep or arousing from sleep, the group of 'reflex-epilepsy' could be broadened by apparently 'spontaneous' epilepsies and seizures triggered by normal functioning of the affected system. In other words, an epileptically facilitated network triggers network-specific seizures. The key element in any such case is not the trigger, but rather the epileptic upregulation of a certain function. This approach challenges the classic definition of epilepsy requiring spontaneous seizures.

The epileptic syndromes the present review is dealing with, are the follows:

- The members of the Self-Limiting Childhood Focal Epilepsy that spectrum (SeLFE) (several language related epilepsies may progress to epileptic encephalopathies- (developmental encephalopathis with spike-wave activation in sleep; DE-SWAS; ESES and LKS) consituting the system epilepsies of the the perisylvian human communication network (PN).
- Absence epilepsy and Sleep related Hypermotor Epilepsy (SHE), the twin conditions of the antagonistic systems of falling asleep and arousing from NREM sleep.
- Juvenile myoclonic epilepsy is hosted by the thalamocortical system and there are widespread additional cortical epileptogenic fields causing myoclony,photisensitivity, and other reflex features.
- Posttraumatic epilepsy, modelling epileptogenesis in any damaged brain region experiencing an epileptic derailment of homeostatic plasticity.

Features of epileptogenesis

The diverse presentations of different epilepsies do not contradict to the existence of a common way of epileptogenesis. Epileptogenesis prefers those networks involved in plasticity, it favors developmental periods, and may resolve. It carries a risk of escalation in both localization and severity e.g. to epileptic encephalopathies (ILAE consensus) [2]. NREM sleep enhances all types of spiking and pathological HFO [3-5]. It promotes epileptogenesis. Sleep oscillations become templates for Interictal Epileptiform Discharges (IEDs) [6]. The clearest example of such transformation is that from the hippocampal SpW-R to its epileptic counterpart spike-pathological ripple Buzsáki, Gulyás Freund [7,8]. Epileptogenesis and epileptic progression are both underlined by an increase and spreading of excitation, shown by the shared presence of pathological HFOs. IEDs play important roles in epileptogenesis. The relationship between IEDs and the up- and down-states of slow sleep oscillations has been investigated in detail [9]. In contrast to spindles that link to the 'up' states of slow waves, HFO predominantly occur in the transition-zones from the 'up' to the 'down' state. In our pre-surgical epilepsy investigations, IEDs prefer the "down" state of the frontal scalp slow waves [10]. These studies highlight the so-called "bistability" (or alternating polarity) of sleep slow waves as a potential factor driving epileptogenesis in sleep.

CONCLUSION

This article provides an overview of the system epilepsy concept

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through examples of major clinical epilepsy syndromes and also enlights the homeostatic derailment hypothesis of common epileptogenesis. It emphasizes how alterations in neural network dynamics and sleep regulation may contribute to the onset and progression of epilepsy. This integrates evidence from clinical observations and experimental models to support this hypothesis. Furthermore, it highlights the interplay between sleep disturbances and epileptic activity. This article offers potential avenues for targeted therapeutic interventions in epilepsy management.

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