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Reviewing the effect of deep brain stimulation on brain rhythms in epilepsy

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Authors

Shadi Choupankareh[1],
Mohammad Reza Raoufy[2,]
[2], Javad Mirnajafi-Zadeh[3,*]

1.1Department of Physiology, Faculty of Medical Sciences, Tarbiat Modares University, Tehran, Iran
2.Faculty of Medical Sciences, Institute for Brain Sciences and Cognition, Tarbiat Modares University, Tehran, Iran
3. Faculty of Medical Sciences, Institute for Brain Sciences and Cognition, Tarbiat Modares University, Tehran, Iran

***Corresponding authors**:
Javad Mirnajafi-Zadeh, Department of Physiology, Faculty of Medical Sciences, Tarbiat Modares University,

mirnajaf@modares.ac.ir

ABSTRACT

In epilepsy as a chronic neurological disease, there are significant alterations in the brain network which results in abnormal brain activity. Understanding the exact changes in brain rhythms may help the investigators to find the brain networks activity in health and disease more precisely. In this article, at first we reviewed the findings from recent animal and clinical studies showing that brain rhythms are affected in the epileptic brain. Then, some documents demonstrating the compensative effect of DBS on these oscillations will be discussed. In this article we reviewed the studies in field of epilepsy and brain rhythms. For this purpose we searched the duciments by scientific serach engeens including PubMed, ScienceDirect and Google Scholar. The electrophysiological studies have indicated significant changes in delta (1–4 Hz), theta (4-7 Hz), alpha (8-12 Hz), beta (13–30 Hz), and gamma (30–80 Hz) oscilations in the epileptic brain. Recently, deep brain stimulation (DBS) has been suggested as a potential and efficient treatment for pharmacoresistant epileptic seizures. The exact mechanism of DBS action is unclear, but some studies demonstrate that one of its probable mechanisms is modulating neural network activity. It seems that the probable compensative alteration in brain rhythms may be considered as a mechanism of DBS anticonvulsant action.

Keywords: Epilepsy; Seizure; Deep brain stimulation; Brain rhythms; Neural networks

Introduction

Epilepsy is a chronic brain disease resulting from abnormal neuronal activity due to an imbalance between excitation and inhibition in the neural networks. Different factors may cause epilepsy, such as trauma, infection, hypoxia, fever, and genetic factors. Although many antiepileptic drugs are helpful, about 30% of epilepsy patients have uncontrolled seizures. One alternative treatment for this kind of refractory epilepsy is deep brain stimulation (DBS). Mesial temporal lobe epilepsy is the most common form of drug-resistant epilepsy in adults. In mesial temporal lobe epilepsy, abnormal neural activities may observe in the brain's limbic areas such as the hippocampus, rhinal cortices, and amygdala.

These abnormal activities may represent themselves as changes in brain rhythms and neural oscillations.

Brain oscillations result from the population activity of neurons and can be evaluated through local field potential recordings. Multiple studies demonstrated that these rhythms contribute to the cognitive process; for example, theta rhythms have an essential role in memory, particularly episodic memory, and gamma oscillations have a role in processes such as learning, memory, and other complex cognitive operations.

The electrophysiological studies have indicated significant changes in these oscillations in the epileptic brain. Cognitive impairments are common comorbidity of chronic epilepsy. The disruption of these oscillations' activity results in some cognitive impairment. In this review, we discuss new findings that reveal DBS as a treatment for refractory epilepsy may affect the characteristics of brain waves.

Delta rhythms

High amplitude delta oscillations, generally defined as 1–4 Hz rhythms, are observed in states of the absence of consciousness, including slow wave sleep, anesthesia, generalized epileptic seizures, and disorders of consciousness (1). Delta oscillations are involved in cognitive processes (2, 3) and contribute to learning, motivational processes, and the brain reward system (4). Delta oscillations, as inhibitory oscillations, may modulate the activity of those networks that should be inactive to perform the task (5). Both awake and slow-wave sleep delta sources are in the medial prefrontal, orbitofrontal, and anterior cingulate cortices (6).

There are many reports about the changes in these rhythms during epileptic seizures. Delta rhythms have a role in modulating high-frequency oscillations during seizure episodes. This phenomenon decreases neuronal firing (7, 8). Additionally, it is suggested that in non-convulsive status epilepticus, spikes are phase-locked to the delta oscillations (9). Epileptic animals in the pilocarpine model of temporal lobe epilepsy exhibited significantly higher delta power in both hippocampal and two cortical layers (Ctx1 and Ctx2) compared to control animals (10). In a clinical study, phase-amplitude coupling between delta band (0.3–4) and high-frequency oscillations was significantly elevated in epileptic channels recorded from scalp EEG of epileptic patients and was significantly stronger in the seizure onset zone compared to normal regions (11–13).

Theta rhythms

Theta rhythms are oscillations with 4-12 Hz frequency and large amplitude, which are dominant during exploration and rapid eye movement (REM) sleep, and occur in many cortical and subcortical regions. In the hippocampus, theta rhythms have a crucial role in memory formation, particularly spatial and episodic memory. Theta oscillations occur in all hippocampal subregions (14, 15). Some GABAergic interneurons of the medial septum-diagonal band of Broca are theta pacemakers (16). The septal interneurons target interneurons in hippocampal subregions (dentate gyrus, CA1and CA3), thereby disinhibiting the hippocampal pyramidal cells, creating theta rhythms in their firing (15).

Many studies have demonstrated substantial changes in hippocampal theta activity in epilepsy. However, there are contradictory studies. In several rodent models of temporal lobe epilepsy, a decrease in hippocampal theta power and phase-locking, such as theta-gamma coupling, has been demonstrated. This alteration is associated with memory deficit (17–19).

Using the pilocarpine model of mesial temporal lobe epilepsy in rats, the absolute power and median frequency of theta rhythm has been reported to decrease soon after pilocarpine-induced status epilepticus, which was correlated with spatial memory deficit (10, 20). This effect was persistent and time-dependent during epileptogenesis in the pilocarpine model in rats (21). In addition, another study has shown that sporadic spikes reduced theta power both transiently and persistently during epileptogenesis, and this negative effect was acuter in the early stage than later stage as in the latent stage power of theta was strong (22). The decrement of theta power was significantly higher in the stratum oriens and lacunosum-molecular layer of the hippocampal CA1 region (10).

Moreover, in the lithium-pilocarpine model, both theta frequency and power were lower in temporal lobe epilepsy animals (23). Colom et al. study in the pilocarpine model of chronic epilepsy also demonstrated a significant reduction in the theta power spectrum associated with an increase in mean theta frequencies in epileptic animals (24).

In contrast to the above mentioned reports, Bae et al. have reported that there was a significant peak in EEG power in the range of theta band frequency (between 3.4–5.8 Hz and 7.8–8.7 Hz) in epileptic mice (25).

In kainite model of medial temporal lobe epilepsy, the theta frequency was decreased all over the medial entorhinal cortex and dentate gyrus, which was recorded in epileptic mice during rest, exploration, and running (26). In another study, there was a reduction of hippocampal theta power and coherence along the CA1–dentate axis due to discoordination of hippocampal inputs from layers III and II of the entorhinal cortex and the contralateral hippocampus that may cause episodic-like memory impairment (26–28).

Consistent with these findings, an alteration of theta coherence between the entorhinal cortex and dentate gyrus in kainate-injected epileptic mice has been shown during the interictal phase. They suggested that hippocampal cell loss can cause the discoordination of this microcircuit (29). Additionally, there is a disruption of theta-gamma coordination in the dorsal hippocampus of epileptic rats, which is related to episodic-like memory deficits in interictal activities. It is suggested that dysfunction of parvalbumin basket cells, an interneuronal population, may be involved in this hippocampal rhythmopathy (30). In the kainite model of *status epilepticus*, an interesting finding is that theta power decreases during convulsive seizures in mice, however, the theta power increases in rats (31).

In a study that used perforant path stimulation to induce temporal lobe epilepsy, it was found that the occurrence of spontaneous recurrent seizures decreased theta oscillations in the hippocampus; however, the synchrony between the hippocampus and mPFC increased in the pri-ictal phase. This event is correlated with a strong coupling between hippocampal theta and mPFC gamma oscillations (32). However, in the kindling stimulation of the perforant path in rabbits, an abrupt increase in the theta range in both hippocampal EEG and medial septal–diagonal band complex before the generation of the epileptic activity was observed (33).

In addition to animal studies, there are some reports about the changes in theta rhythms in epilepsy patients. For instance, Fu and coworkers in a clinical study of 4 patients with refractory temporal lobe epilepsy showed that interictal spikes which are a biomarker of epilepsy and occur between seizures may affect the theta rhythm. Indeed right after spikes, the power of theta rhythm is reduced and it is sustained between spikes periods (34). It has been shown that the sporadic spikes in patients with temporal lobe epilepsy reduced theta power and it may be a biomarker to evaluate the damage of epilepsy to cognitive rhythms (35). Investigation of attention dysfunction in patients with temporal lobe epilepsy showed an impairment of power spectral density in the theta band and coherence and correlation of theta oscillation in the frontal area (36).

However, unlike these findings, an increase in cortical theta-band activity has been also observed in epileptic patients compared to healthy controls (37).

Altogether, according to the experimental and clinical studies, the alteration in theta activity is contradictory and maybe depends on the model used to induce epilepsy and the stage of epileptogenesis, or the kind of animal used in experiments.

Alpha rhythms

Alpha oscillations (8-12 Hz) are usually recorded in posterior regions with eyes closed during wakeful relaxation. Alpha-band activity is predominant in layer V of occipital regions and participates in inhibitory processes which have a role in various cognitive operations such as attention and memory (38–40). It has been reported that alpha frequency decreases in patients with epilepsy (41). In a clinical study significant decrease in an alpha subband has been shown in focal epileptic patiants by fourier analysis of the interictal scalp EEG (Pyrzowski et al., 2015). Moreover it is suggested that absence

of “normal posterior alpha rhythm” is a state marker of seizure disorder (42).

Furthermore, in patient with epilepsy when seizures spreading across the cortex, one of the patterns is theta-alpha activity (TAA); which characterized by sustained oscillations in the theta-alpha range with gradually increasing amplitude. This pattern is useful to distinguish between local onset and propagated seizures since, it was commonly associated with the regions of seizure spread (43).

Beta rhythms

Beta oscillations (13–30 Hz) are predominant in the parietal cortex and link to motor function (44). Several studies of perception in humans have indicated a specific association of beta band activity with endogenously triggered perceptual changes (45). In epilepsy the beta activity changes. In patients with focal epilepsy there is an increase in the voltage of the beta activity on the side of epileptic discharge (46).

In a case report study it is demonstrated the various patterns of focal beta activity on EEG during status epilepticus in a patient with complex partial seizures. For example in early days of treatment with antiepileptic drugs there was a spindle-like beta activity in temporal left area, and, the later days temporal beta activity developing into alpha activity, followed by rhythmic sharp waves. This is a rare seizure pattern in patient with refractory epilepsy (48).

In the absence seizures, there is a positive correlation between spike-wave discharge and pre-ictal 20–40 Hz (beta) spectral power and negatively correlates with 4–7 Hz (theta) power (47).

Gamma rhythms

Gamma rhythms are oscillations with high frequency (30–80 Hz) and low amplitude associated with attentiveness, focused arousal, sensory perception, movement, and prediction (49, 50). In the neocortex, gamma oscillations were dominant in the frontal and parietal areas (51). Gamma rhythms (25–100 Hz) in the CA1 hippocampus are modulated by sensory inputs, memory consolidation, and attention. Hippocampal gamma rhythms encompass two types of frequency ranges, including slow gamma (~25– 55 Hz) that is driven by inputs of CA3 and fast gamma (~60–100 Hz) that is entrained by inputs from the medial entorhinal cortex. There is some evidence that inhibitory interneurons are crucial and sufficient for gamma generation (15).

A pathologically increased gamma-band activity was observed both close to seizure time and during interictal periods in a rat model of temporal lobe epilepsy and also in a clinical study (52, 53). In kainate-treated animals, there was an impairment of theta-gamma coupling in the hippocampus, mPFC, and medial entorhinal cortex. Since the coupling between the phase of theta (slow oscillations) and the amplitude of gamma (fast oscillations) may be involved in information processing (54), it is suggested that this disturbance in the cortical areas may play a role in episodic memory dysfunction following seizure development. It may result from poor oscillatory firing of parvalbumin basket cells (30, 55).

In a human study of hippocampal activity recordings during the free recall task, a standard episodic memory test, epileptogenic hippocampi exhibited a significant decrease in gamma band power during successful item encoding. In contrast, the non-epileptogenic group exhibited a normal gamma-band effect (56).

Furthermore, in the absence seizures gamma oscillatory activity increases simultaneously with an increase in lower EEG frequencies activity. The rise in gamma oscillations is short-lasting and decreased before lower EEG frequency ranges (57).

Other frequencies

Pathological high-frequency oscillations (HFOs) in the epileptic brain of animals and patients differ from normal HFOs in the hippocampus. Pathological HFOs result from the hypersynchronization of action potentials in a population of neurons. They are a biomarker of the epileptic region, essential in determining the seizure onset zone. In animal models of chronic limbic epilepsy, the frequency range of HFOs is 250-600 Hz and occurs in the dentate gyrus, CA1, and CA3 areas of the hippocampus, subiculum, and entorhinal cortex (58).

Effect of DBS on brain rhythms in epilepsy

As we briefly reviewed in the previous sections, there are significant alterations in the epileptic brain network which results in abnormal brain activity. Recently, deep brain stimulation has been suggested as a potential treatment way to compensate for these changes and return brain activity to normal situations.

The exact mechanism of DBS action as a treatment of refractory epilepsy is unclear, but some studies demonstrate that one of its probable mechanisms is modulating neuronal network activity. Here we discuss some studies about the DBS effects on brain oscillation characteristics.

In pilocarpine-induced epileptic mice applying DBS (130 Hz) in the anterior nucleus of the thalamus decreased theta power and increased gamma power in the motorcortex area (25). Low-frequency DBS (5 and 20 Hz) in the medial septum in the kainate-induced acute seizure model significantly increased the power of theta oscillation. Indeed DBS in the medial septum alleviated the severity of seizure activities. So it is suggested that increased theta power could involve in the antiseizure effect of DBS (59).

In the intra-hippocampal kainate mouse model study, the effects of low-frequency stimulation (LFS) and high-frequency stimulation (HFS) were contradictory. LFS (1 Hz, 100 μs, 300 μA) in the bilateral anterior nucleus of the thalamus decreased the delta power while increased the gamma power in the EEG of the hippocampus after treatment for one week. By contrast, after HFS (100Hz, 100 μs, 30 μA), the total power of all brain waves decreased. It is indicated that LFS also decreased the frequency of high-frequency oscillations (HFO) and interictal spikes which are the important indices of seizure severity, however, HFS increased the HFO frequency. Furthermore, LFS in chronic epileptic mice improved their performance in the object-location task, novel object recognition, and freezing test, but HFS did not affect these behaviors. So it is concluded that LFS in the anterior nucleus of the thalamus caused cognition improvement by modulation of hippocampal EEG rhythm (60).

According to the important role of theta oscillations of septohippocampal networks in excitability, seizure generation, and cognitive processing, in the pilocarpine model of epilepsy, theta frequency stimulation (7.7 Hz) of the medial septal nucleus improved spatial learning in a behavioral task like the Barnes maze spatial navigation task (61).

Sohal and coworkers applied responsive cortical stimulation via a cranially programmable implant to describe the effects of neurostimulation on rhythmic activity in the neocortex and hippocampus in patients with refractory epilepsy. They found that the effects of stimulation on phase locking seem to occur throughout the gamma-frequency range (35–100 Hz) but not at lower frequencies (10–30 Hz), such as alpha and beta bands.

High-frequency stimulation (100 and 333 Hz) suppresses phase locking between gamma-frequency rhythmic activities recorded at different locations. Therefore, their results suggest a specific mechanism that responsive stimulation could suppress epileptiform activity, disturb the network between the stimulated region and downstream targets, and possibly have therapeutic effects in epilepsy (62).

Conclusion

Investigating the modulation of brain rhythms can be considered as an appropriate supplementary method for examining the efficacy of DBS in epilepsy patients. Altogether, it seems that DBS modulates alternations of brain rhythm that accrue in the epileptic brain, thereby improving network function in the cognitive process, particularly impaired learning and memory in epilepsy. Therefore, DBS could be an effective treatment for drug-resistant epilepsy.

Conflict of interest

The authors declare that they have no conflicts of interest.

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