



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

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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 documents by scientific search engines 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) oscillations 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

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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, CA1 and 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 pre-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 patients 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 μ s, 300 μ A) 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 μ s, 30 μ A), 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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References

- [1] Frohlich J, Toker D, Monti MM. Consciousness among delta waves: a paradox? *Brain* 2021; 144(8):2257–77.

- [2] Knyazev GG, Slobodskoj-Plusnin JY, Bocharov AV. Event-related delta and theta synchronization during explicit and implicit emotion processing. *Neuroscience* 2009; 164(4):1588–600.
- [3] Başar E, Başar-Eroglu C, Karakaş S, Schürmann M. Gamma, alpha, delta, and theta oscillations govern cognitive processes. *International Journal of Psychophysiology* 2001; 39(2-3):241–8.
- [4] Knyazev GG. Motivation, emotion, and their inhibitory control mirrored in brain oscillations. *Neuroscience & Biobehavioral Reviews* 2007; 31(3):377–95.
- [5] Buzsaki G. *Rhythms of the Brain*: Oxford university press; 2006.
- [6] Dang-Vu TT, Schabus M, Desseilles M, Albouy G, Boly M, Darsaud A et al. Spontaneous neural activity during human slow wave sleep. *Proceedings of the National Academy of Sciences* 2008; 105(39):15160–5.
- [7] Guirgis M, Chinvarun Y, Carlen PL, Bardakjian BL. The role of delta-modulated high frequency oscillations in seizure state classification. *Annu Int Conf IEEE Eng Med Biol Soc* 2013; 2013:6595–8.
- [8] Ibrahim GM, Wong SM, Anderson RA, Singh-Cadieux G, Akiyama T, Ochi A et al. Dynamic modulation of epileptic high frequency oscillations by the phase of slower cortical rhythms. *Experimental neurology* 2014; 251:30–8.
- [9] Hindriks R, Meijer HGE, van Gils SA, van Putten MJ. Phase-locking of epileptic spikes to ongoing delta oscillations in non-convulsive status epilepticus. *Frontiers in systems neuroscience* 2013; 7:111.
- [10] Pasquetti MV, Meier L, Marafija JR, Caus LB, Tort ABL, Calcagnotto ME. Hippocampal CA1 and cortical interictal oscillations in the pilocarpine model of epilepsy. *Brain Research* 2019; 1722:146351.
- [11] Amiri M, Frauscher B, Gotman J. Phase-amplitude coupling is elevated in deep sleep and in the onset zone of focal epileptic seizures. *Frontiers in human neuroscience* 2016; 10:387.
- [12] Yu H-T, Cai L-H, Wu X-Y, Wang J, Liu J, Zhang H. Cross-frequency network analysis of functional brain connectivity in temporal lobe epilepsy. *Chinese Physics B* 2019; 28(4):48702.
- [13] Weiss S, Sheybani L, Seenarine N, Fried I, Sharan A, Wu C et al. Delta Oscillation Coupled Propagating Fast Ripples Precede Epileptiform Discharges in Patients with Focal Epilepsy 2022.
- [14] Buzsáki G, Vanderwolf CH. Cellular bases of hippocampal EEG in the behaving rat. *Brain Research Reviews* 1983; 6(2):139–71.
- [15] Colgin LL. Rhythms of the hippocampal network. *Nature Reviews Neuroscience* 2016; 17(4):239–49.
- [16] Green JD, Arduini AA. Hippocampal electrical activity in arousal. *Journal of neurophysiology* 1954; 17(6):533–57.
- [17] Suthana N, Fried I. Deep brain stimulation for enhancement of learning and memory. *Neuroimage* 2014; 85:996–1002.
- [18] Shuman T, Amendolara B, Golshani P. Theta rhythmopathy as a cause of cognitive disability in TLE. *Epilepsy currents* 2017; 17(2):107–11.
- [19] Moxon KA, Shahlaie K, Girgis F, Saez I, Kennedy J, Gurkoff GG. From adagio to allegretto: the changing tempo of theta frequencies in epilepsy and its relation to interneuron function. *Neurobiology of disease* 2019; 129:169–81.
- [20] Chauviere L, Raftafi N, Thinus-Blanc C, Bartolomei F, Esclapez M, Bernard C. Early deficits in spatial memory and theta rhythm in experimental temporal lobe epilepsy. *Journal of Neuroscience* 2009; 29(17):5402–10.
- [21] Fu X, Wang Y, Belkacem AN, Cao Y, Cheng H, Zhao X et al. Interictal Spike and Loss of Hippocampal Theta Rhythm Recorded by Deep Brain Electrodes during Epileptogenesis. *Sensors* 2022; 22(3):1114.
- [22] Ge M, Wang D, Dong G, Guo B, Gao R, Sun W et al. Transient impact of spike on theta rhythm in temporal lobe epilepsy. *Experimental neurology* 2013; 250:136–42.
- [23] Richard GR, Titiz A, Tyler A, Holmes GL, Scott RC, Lenck-Santini P. Speed modulation of hippocampal theta frequency correlates with spatial memory performance. *Hippocampus* 2013; 23(12):1269–79.
- [24] Colom LV, García-Hernández A, Castañeda MT, Perez-Cordova MG, Garrido-Sanabria ER. Septo-hippocampal networks in chronically epileptic rats: potential antiepileptic effects of theta rhythm generation. *Journal of neurophysiology* 2006; 95(6):3645–53.
- [25] Bae S, Lim H-K, Jeong Y, Kim S-G, Park S-M, Shon Y-M et al. Deep brain stimulation of the anterior nuclei of the thalamus can alleviate seizure severity and induce hippocampal GABAergic neuronal changes in a pilocarpine-induced epileptic mouse brain. *Cerebral Cortex* 2022.
- [26] Kiliyas A, Häussler U, Heining K, Frieppel UP, Haas CA, Egert U. Theta frequency decreases

- throughout the hippocampal formation in a focal epilepsy model. *Hippocampus* 2018; 28(6):375–91.
- [27] Inostroza M, Brotons-Mas JR, Laurent F, Cid E, La Prida LM de. Specific impairment of “what-where-when” episodic-like memory in experimental models of temporal lobe epilepsy. *Journal of Neuroscience* 2013; 33(45):17749–62.
- [28] Laurent F, Brotons-Mas JR, Cid E, Lopez-Pigozzi D, Valero M, Gal B et al. Proximodistal structure of theta coordination in the dorsal hippocampus of epileptic rats. *Journal of Neuroscience* 2015; 35(11):4760–75.
- [29] Froriep UP, Kumar A, Cosandier-Rimélé D, Häussler U, Kiliyas A, Haas CA et al. Altered theta coupling between medial entorhinal cortex and dentate gyrus in temporal lobe epilepsy. *Epilepsia* 2012; 53(11):1937–47.
- [30] Lopez-Pigozzi D, Laurent F, Brotons-Mas JR, Valderrama M, Valero M, Fernandez-Lamo I et al. Altered oscillatory dynamics of CA1 parvalbumin basket cells during theta-gamma rhythmopathies of temporal lobe epilepsy. *neuro* 2016; 3(6).
- [31] Sharma S, Puttachary S, Thippeswamy A, Kanthasamy AG, Thippeswamy T. Status epilepticus: behavioral and electroencephalography seizure correlates in kainate experimental models. *Frontiers in neurology* 2018; 9:7.
- [32] Broggin ACS, Esteves IM, Romcy-Pereira RN, Leite JP, Leao RN. Pre-ictal increase in theta synchrony between the hippocampus and prefrontal cortex in a rat model of temporal lobe epilepsy. *Experimental neurology* 2016; 279:232–42.
- [33] Kitchigina VF, Butuzova MV. Theta activity of septal neurons during different epileptic phases: the same frequency but different significance? *Experimental neurology* 2009; 216(2):449–58.
- [34] Fu X, Wang Y, Ge M, Wang D, Gao R, Wang L et al. Negative effects of interictal spikes on theta rhythm in human temporal lobe epilepsy. *Epilepsy & Behavior* 2018; 87:207–12.
- [35] . Manling G, Jundan G, Yangyang X, Zhiguo F, Weide L, Xinxin M et al. Transient reduction in theta power caused by interictal spikes in human temporal lobe epilepsy. *Annu Int Conf IEEE Eng Med Biol Soc* 2017; 2017:4256–9.
- [36] Li X, Hou Y, Ren Y, Tian X, Song Y. Alterations of theta oscillation in executive control in temporal lobe epilepsy patients. *Epilepsy Research* 2018; 140:148–54.
- [37] Quraan MA, McCormick C, Cohn M, Valiante TA, McAndrews MP. Altered resting state brain dynamics in temporal lobe epilepsy can be observed in spectral power, functional connectivity and graph theory metrics. *PloS one* 2013; 8(7):e68609.
- [38] Roohi-Azizi M, Azimi L, Heysieattalab S, Aamidfar M. Changes of the brain’s bioelectrical activity in cognition, consciousness, and some mental disorders. *Medical journal of the Islamic Republic of Iran* 2017; 31:53.
- [39] Knyazev GG. Motivation, emotion, and their inhibitory control mirrored in brain oscillations. *Neuroscience & Biobehavioral Reviews* 2007; 31(3):377–95.
- [40] . Rosanova M, Casali A, Bellina V, Resta F, Mariotti M, Massimini M. Natural frequencies of human corticothalamic circuits. *Journal of Neuroscience* 2009; 29(24):7679–85.
- [41] . Larsson PG, Kostov H. Lower frequency variability in the alpha activity in EEG among patients with epilepsy. *Clinical Neurophysiology* 2005; 116(11):2701–6.
- [42] Aich TK. Absent posterior alpha rhythm: An indirect indicator of seizure disorder? *Indian J Psychiatry* 2014; 56(1):61–6.
- [43] Viktor Sip, Julia Scholly, Maxime Guye, Fabrice Bartolomei, Viktor Jirsa. Evidence for spreading seizure as a cause of theta-alpha activity electrographic pattern in stereo-EEG seizure recordings.
- [44] . Chakarov V, Naranjo JR, Schulte-Mönting J, Omlor W, Huethe F, Kristeva R. Beta-range EEG-EMG coherence with isometric compensation for increasing modulated low-level forces. *Journal of neurophysiology* 2009; 102(2):1115–20.
- [45] Engel AK, Fries P. Beta-band oscillations—signalling the status quo? *Current opinion in neurobiology* 2010; 20(2):156–65.
- [46] . Green RL, Wilson WP. Asymmetries of beta activity in epilepsy, brain tumor, and cerebrovascular disease. *Electroencephalography and Clinical Neurophysiology* 1961; 13(1):75–8.
- [47] . Sorokin JM, Paz JT, Huguenard JR. Absence seizure susceptibility correlates with pre-ictal β oscillations. *J Physiol Paris* 2016; 110(4 Pt A):372–81.
- [48] . Bonati LH, Naegelin Y, Wieser H, Fuhr P, Ruegg S. Beta activity in status epilepticus. *Epilepsia* 2006; 47(1):207–10.
- [49] Timofeev I, Bazhenov M, Seigneur J, Sejnowski T. Neuronal synchronization and thalamocortical rhythms in sleep, wake and epilepsy. *Jasper’s Basic Mechanisms of the Epilepsies* [Internet]. 4th edition 2012.

- [50] Jia X, Kohn A. Gamma rhythms in the brain. *PLoS biology* 2011; 9(4):e1001045.
- [51] Benchenane K, Peyrache A, Khamassi M, Tierney PL, Gioanni Y, Battaglia FP et al. Coherent theta oscillations and reorganization of spike timing in the hippocampal-prefrontal network upon learning. *Neuron* 2010; 66(6):921–36.
- [52] Medvedev AV. Epileptiform spikes desynchronize and diminish fast (gamma) activity of the brain: an “anti-binding” mechanism? *Brain research bulletin* 2002; 58(1):115–28.
- [53] Medvedev AV, Murro AM, Meador KJ. Abnormal interictal gamma activity may manifest a seizure onset zone in temporal lobe epilepsy. *International journal of neural systems* 2011; 21(02):103–14.
- [54] Tort ABL, Komorowski RW, Manns JR, Kopell NJ, Eichenbaum H. Theta–gamma coupling increases during the learning of item–context associations. *Proceedings of the National Academy of Sciences* 2009; 106(49):20942–7.
- [55] Malkov A, Shevkova L, Latyshkova A, Kitchigina V. Theta and gamma hippocampal–neocortical oscillations during the episodic-like memory test: impairment in epileptogenic rats. *Experimental neurology* 2022; 354:114110.
- [56] Lega B, Dionisio S, Bingaman W, Najm I, Gonzalez-Martinez J. The gamma band effect for episodic memory encoding is absent in epileptogenic hippocampi. *Clinical Neurophysiology* 2015; 126(5):866–72.
- [57] Benedek K, Berényi A, Gombkötő P, Piilgaard H, Lauritzen M. Neocortical gamma oscillations in idiopathic generalized epilepsy. *Epilepsia* 2016; 57(5):796–804.
- [58] Bragin A, Engel Jr J, Staba RJ. High-frequency oscillations in epileptic brain. *Current opinion in neurology* 2010; 23(2):151.
- [59] Wang Y, Shen Y, Cai X, Yu J, Chen C, Tan B et al. Deep brain stimulation in the medial septum attenuates temporal lobe epilepsy via entrainment of hippocampal theta rhythm. *CNS neuroscience & therapeutics* 2021; 27(5):577–86.
- [60] Wang YI, Liang J, Xu C, Wang Y, Kuang Y, Xu Z et al. Low-frequency stimulation in anterior nucleus of thalamus alleviates kainate-induced chronic epilepsy and modulates the hippocampal EEG rhythm. *Experimental neurology* 2016; 276:22–30.
- [61] Izadi A, Ondek K, Schedlbauer A, Keselman I, Shahlaie K, Gurkoff G. Clinically indicated electrical stimulation strategies to treat patients with medically refractory epilepsy. *Epilepsia Open* 2018; 3:198–209.
- [62] Sohal VS, Sun FT. Responsive neurostimulation suppresses synchronized cortical rhythms in patients with epilepsy. *Neurosurg Clin N Am* 2011; 22(4):481–8, vi.