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Innovations in Neurophysiology and Their Use in Neuropsychiatry

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ABSTRACT

Many structural and functional tests are used to explore the nature of neurodevelopmental and neurodegenerative diseases. Cognitive involvement has become more and more remarkable in many neurological and psychiatric diseases. This condition evoked a paradigm shift, and today disorders are addressed from a neuroscientific perspective, including silent symptoms. The spatial resolution of structural studies is lacking and is combined with the unique temporal resolution of EEG methods. In our current clinical practice, EEG does not have definitive diagnostic value in psychiatric disorders, but it helps to make a correct diagnosis by excluding other neurological diseases. However, the use of EEG for research purposes is promising in both groups. In this review; there is up-to-date information on the use of electrophysiological examinations in neurological diseases, especially Alzheimer's disease, Parkinson's disease, Frontotemporal dementia, and psychiatric disorders such as schizophrenia, mood disorders, attention deficit and hyperactivity disorder, and obsessive-compulsive disorder, to define the point we have reached in our journey to understand these disorders.

Keywords: Electroencephalography, event-related oscillations, neurophysiology, neuropsychiatry

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INTRODUCTION

Electroencephalography (EEG) is a recording method performed on the skull to measure electrical signals, which are basically the natural frequency components of the brain. Complex oscillations consisting of the overlapping of different frequencies are obtained by polarizing the electric current resulting from the changes in the postsynaptic potentials of the pyramidal neurons in the cortex (1). For many years, the EEG studies pioneered by Hans Berger have been used mainly to evaluate epileptic activity with the expectation that the patient studied would show no function or activity.

In the eyes-closed resting-state EEG (rsEEG) of a healthy person, alpha band (8-15 Hz) is dominant in the posterior areas. In his experiments, Berger found that the amplitude of the alpha waves decreased in situations requiring mental effort and selective attention and that the alpha waves were replaced by beta waves, which have been discovered second to alpha waves. In the following years, this process was named as alpha-blocking. Over time, it was observed in the resting state that delta (0.5-3.5 Hz) and theta (4-7 Hz) oscillations were suppressed, whereas alpha (8-15 Hz) and beta (15-30 Hz) wave powers increased. Oscillations in event-related EEG recordings vary according to the characteristics of the cognitive paradigm. Event-related potential (ERP), on the other hand, is different from oscillations. ERP refers to the total activity that occurs as a result of time-locked superposition of event-related oscillations (ERO) in all wavebands corresponding to a stimulus (2–4). Determining the frequency band where the oscillatory responses contained in this wave, which appears like a single activity, are stronger and examining the changes that occur in these different frequencies by time, is as important as the measurement of the total activity, and various biophysical methods are used for this analysis.

Highlights

- **The importance of electrophysiology in the diagnosis and follow-up in neuropsychiatry is gradually increasing.**
- **Electroencephalography complements structural examinations with its spatial resolution.**
- **Dynamic electrophysiological examinations are important for early diagnosis during the prodrome period.**

Use of EEG in the context of neurodegenerative diseases that cause cognitive disorders both for understanding normal brain dynamics and for diagnostic purposes could only be possible in the last few decades. Most EEG studies conducted on neurodegenerative diseases focus on the two most common neurodegenerative diseases, that is, Alzheimer's disease (AD) followed by Parkinson's disease (PD). EEG's higher temporal resolution compared to structural neuroimaging methods helps understanding the brain dynamics. In addition, EEG is a non-invasive, cost-effective and easily accessible method. Therefore, data produced by EEG can be potentially used as a "cognitive biomarker".

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This review article, which features the evaluation of EEG use outside of the clinical practice, mainly focuses on two EEG recording methods: rsEEG and event-related potential/oscillation (ERP/O) applications (Table 1). Resting-state EEG, as the name suggests, is based on recording a person's activity in the resting state. It can be conducted either as eyes open or closed. Event-related applications, on the other hand, basically involve giving a stimulus and/or assigning a task to the person during the EEG recording. Electrophysiological activations that occur within the scope of these two methods have quite different dynamics and are thus analyzed using different methods. In this direction, up-to-date information about rsEEG and ERP/O studies in the context of neuropsychiatric diseases will be presented in the remainder of this article with a view to inspire future studies by exploring the dynamics of neuropsychiatric diseases from past to present and understanding the achievements in electrophysiology.

ELECTROPHYSIOLOGICAL ADVANCES IN THE CONTEXT OF NEUROLOGICAL DISEASES

Alzheimer's Disease Spectrum

Electrophysiological studies performed in healthy individuals and in patients with Alzheimer's disease-mild cognitive impairment (AD/MCD) revealed that there were differences between cognitive networks in the two groups. Such studies will facilitate understanding the nature of complex functional networks and thereby identifying possible biomarkers in the diagnosis and follow-up of AD/MCD (5).

The increasing use of pathological examinations and the ability to make clinicopathological correlations have expanded the knowledge on the underlying pathological mechanism of AD. It has been speculated that pathological peptides, which are responsible for the spread of neurodegeneration with a synergistic effect by accumulating predominantly in the entorhinal cortex, hippocampus, basal forebrain, and claustrum in the brain, affect the electrical nature of neurons (6). Amyloid and tau, which are the main proteins expressed in AD, synergistically trigger progressive neurodegeneration. Hence, the clinical picture in AD progresses in parallel with histopathological changes (7,8). Progressive neurodegeneration has for long been held responsible for the electrophysiological changes and the clinical picture associated with AD. However, with the increased number of attempts to change the progression of the disease, it has been recently hypothesized that the initiating factor causing pathological accumulation of peptides is the increase in excitability in the brain (9).

Magnetic resonence imaging (MRI) and positron emission tomography (PET) studies conducted for many years have demonstrated that occipital metabolism is preserved in patients with AD, however the activity decreased in the anterior regions (10,11). Impairment of the functioning of this default mode network, the activity of which increases at resting state in healthy individuals, in the event of AD, leads to hypersynchronization in EEG. As a matter of fact, amyloid deposition has been attributed to hypersynchronization in EEG (12). The appearance of silent epileptic seizures on EEG before dementia develops in relation to the diseases in the AD spectrum proves the relationship between the course of the disease and excitability (13). In this regard, it has also been hypothesized that the hippocampus and its surrounding structures feature a pacemaker activity on the neocortex. The recording of activities with different frequencies simultaneously entering the hippocampus in electrophysiological examinations are to support this hypothesis (10,14).

The disconnection between cortical structures and the thalamus resulting from the progressive pattern of neurodegeneration in AD causes isolated thalamic behavior. In line with this hypothesis, increased synaptic activity is observed in the resting wakefulness state (10). As a matter of fact, the cortical hyperexcitability in human and animal AD models has been attributed to disconnection between the cortical structures and the thalamus (5,15–18). The fast-wave hyperconnectivity observed in the EEG when a cognitive stimulus is given can be considered an early indicator of

Table 1. EEG/Event-related potentials in various neuropsychiatric diseases

EEG		AD/aMCI	DLB/PD	SC	BD	ADHD	OCD
resting state							
	alpha	↓	↓		↓	↓	
	beta	↓	↓		↑	↓	
	theta	↑	↑			↑	↑(frontal)
	delta	↑	↑			↑	↑(frontal)
Task-related							
	theta: power/connectivity	↓	↓	↓↑			
	delta	↓	↓				
	gamma: power/connectivity	↑					
	alpha	↔	↔	↑			
	ERN						↑
General							
	Vigilance	↑	↓				
	Hyperexcitability	↑					
	Epileptic Activity	↑					

AD: Alzheimer's Disease; ADHD: Attention Deficit Hyperactivity Disorder; aMCI: Amnestic Mild Cognitive Impairment; BD: Bipolar Disorder; DLB: Dementia with Lewy Bodies; ERN: Event-Related Negativity; OCD: Obsessive Compulsive Disorder; PD: Parkinson's Disease; SC: Schizophrenia.
↔: No difference between the groups.
↓↑: Variable results.

neurodegeneration-related cognitive deterioration (19). This hypothesis has also been supported by animal models (20).

The concept of hyperexcitability has gained importance given the incidence of epilepsy has been determined to be 7-8 times higher in clinical studies conducted with AD patients (6). In addition, an increase has been observed in comparable studies in hypersynchrony and fast frequencies in patients with AD compared to healthy individuals. These findings suggest that amyloid beta accumulation increases the expression of glutamate receptors, namely NMDA (N-methyl-D-aspartate) and AMPA (α - amino - 3 - hydroxyl - 5 - methyl - 4 - isoxazole - propionate) receptors, excitatory postsynaptic activity, and thereby the neuron hyperexcitability (5,21,22). It has been stated that hyperexcitability in AD develops with the onset of amyloid deposition approximately 20 years before the manifestation of relevant clinical symptoms, as demonstrated by the cerebrospinal fluid (CSF) amyloid levels (13,23).

It has been speculated that the change in the neurotransmitter level in the anatomical regions where neurodegeneration is initiated cause signal transfer from these areas to pyramidal neurons in the cortex through synchronization (24,26). Additionally, in AD, as in other dementias, an increase in slow wave (1-7 Hz) activity was reported and a decrease in fast wave activity at alpha and beta frequencies, in the eyes-closed resting state (26). In healthy individuals, on the other hand, event-related delta responses are expected to increase in the presence of cognitive stimuli. Therefore, the decrease in event-related delta responses, also seen in other cognitive disorders, is accepted as an electrophysiological indicator of cognitive impairment (5,11,37).

It has been speculated that the inconsistent increase in gamma coherence activity may be related to a different type of GABAergic (gamma-aminobutyric acid modifying) system dysfunction in the context of AD. Related experimental evidence suggests that the GABAergic system is relatively less affected compared to the glutamatergic and cholinergic systems. It is the compensatory subunit changes that are thought to allow the GABAergic system to remain roughly intact in the event of AD despite the changes in GABA type A (GABA-A) receptor subunits (27). This may be achieved by the increase in GABA-A receptor subunit synthesis by the surviving hippocampal neurons in order to maintain the inhibitory hippocampal circuit (5,28).

The relationship between memory and gamma response has been demonstrated in many studies (29). It is known that inhibitory GABAergic interneurons have a direct modulatory impact on gamma oscillations (30). Certain neurotransmitter combinations are implicated in simple cognitive responses. GABA, GABA/glutamate and dopamine have been defined as neurotransmitters that have an effect on gamma frequency (31,32).

ERP/O studies provide information about the excitable networks of the brain. While simple sensory stimuli do not cause a change in the preclinical/prodromal periods of amnestic processes, cognitive stimuli reduces delta power in the fronto-central regions of the brain (18). In addition, while emotional stimuli cause an increase in the P100 response in the occipital region, the simultaneous decrease in the N170 response in the fronto-central region indicates that there is a decrease in activity in the early stages of the disease in the emotional and cognitive networks called heteromodal association areas, long before the occurrence of the hyperexcitability in the primary visual area (11,12,14).

With the progression from mild cognitive impairment (MCI) to AD, prolongation of latency and decrease in amplitude have been shown in auditory P300 in parallel with a healthy cognitive profile (33-35). In a longitudinal study, it was reported that auditory P300 latency values were correlated with the severity of cognitive impairment and that this correlation could be utilized as a cost-effective method for monitoring neurodegenerative processes in AD (11).

Cognitive Disorders in Lewy Bodies/Parkinson's Disease

PD is a neurodegenerative disorder that targets neurons in the substantia nigra, the dopamine-producing center of the brain. The course of cognitive impairment and the manifestation of related symptoms may differ depending on the progression pattern of the pathogen peptide and neurodegeneration (10).

In PD, as in AD dementia, it was determined that the event-related delta and theta power spectrum and phase locking, which occur during the cognitive stimulus were lower. In addition, it has been found that event-related delta and theta coherences decrease as the severity of cognitive impairment increases in the event of both AD and PD. In contrast, the delta-theta response has reportedly increased with cognitive stimulus in healthy individuals (11,14,22,36).

In PD, eye-closed rsEEG characteristically features a slowdown in the form of a decrease in alpha and beta band strength and an increase in delta and theta band strength. In parallel, in a study on the relationship between cortical alpha slowing and clinical stage, significant correlations were determined in terms of memory, fluency, and executive functions (37,38). Although posterior alpha rhythm in AD dementia is much lower compared to PD dementia, delta rhythm disruption is observed to a lesser extent (11,39).

It is known that the P300 potential, which is one of the most frequently studied ERPs, decreases during the course of PD. In a study comparing cognitively normal PD (PD-CN) and mild cognitive impairment PD (PD-MCI) patients with healthy control subjects, it was shown that visual P300 amplitude values decreased in frontal areas in PD-CN patients and in frontal, central and parietal areas in PD-MCI patients (40).

Event-related potentials and oscillations are indicators of neurodegeneration as well. As a matter of fact, a relationship was found between ERP/O and MRI findings, another indicator of neurodegeneration. Frontal delta ERO power in AD was found to be directly proportional to the volume of the frontal region. On the other hand, the decrease in P300 amplitude has been associated with the decrease in putamen volume (40).

The comparison of PD-MCI and AD-MCI patients revealed that the theta ERO gradually decreased and the visual system was more affected than the auditory system in both groups (38,40). On the other hand, it has been reported that inter-trial phase-locking was preserved in AD-MCI patients, but decreased in PD-MCI patients. Additionally, the decrease in the occipital region was reportedly prominent in PD-MCI, whereas the increase in the occipital region was prominent in AD-MCI (43). As supported by the findings of other structural and functional studies, the decrease in occipital strength in PD-MCI, as opposed to the preservation of the occipital lobe in AD-MCI, may be attributed to the primary involvement of the occipital lobe in PD-MCI (5,11,42).

Frontotemporal Dementia

Frontotemporal dementia (FTD), which accounts for 20% of early-onset dementia cases, is a neurodegenerative disease characterized by changes in three different symptom groups, i.e., motor, language and behavioral variants, depending on the region featuring frontotemporal lobar degeneration. However, given that the symptoms can be seen simultaneously and that the behavioral-language symptoms can be insidious, it may be very difficult to determine the FTD variants and make a differential diagnosis (44).

In one of the early studies, different dementia groups and healthy individuals were distinguished by frequency spectrum analysis in quantitative EEG (QEEG) with a sensitivity and specificity of over 85% (45). In one of the more recent studies conducted with a total of 61 patients, including 21 patients who met the diagnostic criteria for behavioral variant of FTD (bvFTD) according to the Rascovsky criteria (46) and 40 patients with AD, PD, and dementia with Lewy bodies (DLB), the accuracy of the differential diagnosis made with QEEG was calculated as 93% for the differential diagnosis between DLB/PD-FTD and 88% for the differential diagnosis between AD-FTD (43).

The QEEG patterns of FTD patients differ markedly from those of AD patients. In a study conducted with 19 FTD, 19 AD, and demographically matched healthy control subjects, patients' QEEG analyses were examined and detailed neuropsychological tests were performed. The global power of QEEG is calculated specifically for six frequency bands; delta, theta, alpha, beta-1, beta-2 and beta-3. The fast frequency bands consisted of alpha, beta-1, beta-2 and beta-3, whereas the slow frequency bands consisted of delta and theta waves. The comparison of FTD patients with healthy control subjects revealed that there was no increase in slow wave QEEG activities and no decrease in fast wave activities. On the other hand, the comparison of AD patients with healthy control subjects revealed an increase in slow wave QEEG activities and a lesser decrease in fast wave activity.

The logistic regression analysis involves the delta and theta activities which have the highest predictive value in terms of FTD and AD. Visiospatial capacity and episodic memory are also quite effective methods in distinguishing between FTD and AD. The accuracy of the differential diagnosis utilizing the combination of both methods has been reported as 93.3% (5,47).

Given the misdiagnosis rate of up to 86% in pathological series, supplementary use of QEEG in the diagnosis of FTD patients has gained importance. Although most of the FTD patients are followed up with the diagnosis of AD due to confounding symptoms, they were found to be compatible with Pick's disease, a type of FTD, the pathological examinations (46). The clinical picture pertaining to degeneration is similar in the later stages of cognitive impairment, yet the anatomical structures involved especially in the early stages are clearly distinguished. For example, the hippocampal and posterior temporal and parietal neocortical involvement seen in AD are seen in the frontal and anterior temporal regions in FTD (48). It is generally accepted that the electrophysiological findings presumably support this pathology, however the focal and generalized slowing is more prominent in AD and the differentiation between FTD and AD is more problematic in FTD. Studies have shown that the most important QEEG variables in the differentiation of FTD and AD are beta-2 bands originating from the temporal region, and theta, alpha and beta-2 bands originating from the parietal region (49,50).

It is very difficult to recognize and correctly diagnose the primary progressive aphasia (PPA), the language variant of FTD. As a matter of fact, in a cross-sectional study conducted with 40 patients with PPA subtypes, i.e., non-fluent variant PPA (nfvPPA), semantic variant PPA (svPPA) and logopenic PPA (lvPPA), rsEEG successfully distinguished healthy control subjects from patients with PPA, but not the PPA patients with different variants. The use of artificial intelligence and machine learning in this type of EEG analyses renders the analysis of big data possible (51).

In another study investigating the effectiveness of EEG in differentiating FTD variants, it was observed that the brain dynamics highly vary between FTD variants. In rsEEG, it is known that decreased theta power supports the diagnosis of svFTD, while increased diffuse theta power supports the diagnosis of nfvPPA (52).

Especially in its early stages, the behavioral variant of FTD may present symptoms comparable to the behavioral symptoms of late-onset bipolar disorder. As a matter of fact, most FTD patients are reportedly diagnosed with bipolar disorder. The use of electrophysiological findings in combination with neuroimaging methods has been shown to supplement the differential diagnosis of FTD (53).

ELECTROPHYSIOLOGICAL ADVANCES IN THE CONTEXT OF PSYCHIATRIC DISORDERS

A better understanding of the normal electrophysiology of the brain and the desire to reveal the organic etiology in psychiatric diseases have further increased the number of scientific studies on this subject. The clinical features of psychiatric diseases that render their differential diagnosis difficult have accelerated the search for electrophysiological biomarkers. In parallel, differences that exist between healthy individuals and patients with certain psychiatric diseases in terms of electrophysiological features pertaining to the brain are being investigated recently (5).

Schizophrenia

Numerous studies have been conducted on the mechanisms common to both neurodevelopmental and neurodegenerative diseases and the role of abnormal brain oscillations in the relevant pathophysiologies (54). Based on the results of these studies, the view that argues that the temporal relationship between neural oscillations and perception, memory and neural responses should be examined has gained importance (55). Schizophrenia, a disease characterized by psychotic symptoms and behavioral problems, has been redefined as a disease accompanied by cognitive problems in the light of cognitive studies conducted in recent years. Cognitive impairment is observed in 80% of the patients with schizophrenia independent of the clinical factors, treatment regimen, and not having received any treatment at all (56).

In a clinical study investigating theta and gamma oscillations using n-back, a working memory paradigm, in patients with schizophrenia and healthy control subjects, it was noted that the positive relationship expected to be seen between gamma amplitude and the task difficulty-based cognitive load was not observed in patients with schizophrenia (57). Additionally, theta amplitude increased significantly in the frontocentral region during task activation in healthy control subjects, contrary to patients with schizophrenia. Given its importance in terms of working memory in healthy individuals, frontal region activity determines the intensity of theta oscillations. The hypofrontality hypothesis put forward in the context of schizophrenia explains concepts including the frontal dysfunction, the decrease in theta amplitude and impaired working memory (54).

Many studies targeted high frequency oscillations in the gamma band revealed gamma band dysfunction in schizophrenia. Additionally, working memory studies conducted with the n-back paradigm revealed that gamma amplitude increased as the task became more difficult (58). On the other hand, in patients with schizophrenia, the gamma amplitude remained the same regardless of the difficulty of the task. This finding suggests that patients with schizophrenia use complex cognitive processes even in the easiest cognitive task, without choosing the strategy to be used in accordance with the task difficulty, contrary to healthy individuals. Hence, selective attention decreases due to the use of complex cognitive processes for every task.

It has also been shown that gamma amplitude has increased even during positive symptoms such as hallucinations in patients with schizophrenia (30). Some studies reported that the gamma phase-synchronization decreased in the early gamma range (0-100 ms), whereas other studies reported that the gamma amplitude decreased in the late gamma range

(300-500 ms). These findings suggest a possible fronto-thalamo-cortical circuit dysfunction (11).

Multiple frequency band abnormalities dominate the rsEEG in schizophrenia. This situation specifically affects verbal learning skills negatively (58). The power of the rsEEG gamma band has become very important in understanding the nature of cognitive dysfunction and planning appropriate interventions. The theta and gamma oscillations that occur in the visual field differ in patients with schizophrenia when they are shown transforming (reversible) shapes and the deterioration in perceptual-cognitive integration processes may be attributed to these differences. The difficulty experienced by patients with schizophrenia in producing coherent perception negatively affects multiple frequency bands and time windows (54).

The analysis of auditory evoked potentials in healthy individuals revealed that early alpha response causes a high amplitude oscillation in the common fronto-central region (59). On the other hand, it is very striking that the alpha response to auditory stimuli is detected in the parietal and occipital regions with a three-times higher amplitude in patients with schizophrenia. It is very rare for a response to an auditory stimulus to have such a high amplitude in the visual field. Many researchers have explained this situation with impaired connectivity (disconnectivity). It is for the said reason that the functional integration of perception and attention is impaired in patients with schizophrenia and that there is a disruption in the long-term inhibition of task-related cortical areas (5,54).

One of the main issues addressed in studies in the context of schizophrenia is whether high or low frequency oscillations are associated with schizophrenia. Relevant data revealed that there is a coordination disorder between different functional units of the brain, indicating the importance of the whole-brain network theory in understanding schizophrenia.

Bipolar Disorder

Bipolar disorder (BD) is a mental disorder that progresses with unpredictable relapses and remissions due to manic or depressive episodes (60). The presence of cognitive impairment in euthymic periods suggested that cognitive involvement is a fundamental component of BD. The clinical course of BD, which is still being explored, and the resemblance of its symptoms with other neurodegenerative diseases, have made BD the most remarkable disease among mood disorders in electrophysiological studies (61).

Resting-state EEG may reveal the neurophysiological activity of the normal-state network in BD. As a matter of fact, significant elevations in delta, theta and alpha activity were demonstrated in BD patients in resting state. In the low-resolution electromagnetic tomography (LORETA) study (high-resolution EEG source localization), it was reported that the theta band was significantly low in patients with BD in resting state, regardless of whether they were in a manic or depressive episode and that the beta power increased in the prefrontal cortex, parietal cortex, and temporal pole (61). In general terms, it is known that alpha activity is greatly reduced in most BD patients, whereas the beta activity is much increased (5).

Another remarkable finding is the decrease observed in the gamma band power with the ASSR (auditory steady state response) task during euthymic and manic periods (62). ASSR and mismatch negativity (MMN) tasks provide assessment of auditory cortex and related structures. These changes seen in BD and schizophrenia may indicate an auditory cortex pathology common to both diseases. Psychotic symptoms in BD have been accepted as one of the most determining factors on EEG (63). Almost all of the psychotropic drugs have effects that can change electrophysiology. However, there are discrepancies between relevant studies conducted with patients receiving psychotropic medications which are mostly attributed to the disease stage (62,64,65).

Compared to healthy control subjects, it has been shown that P300 values of BD patients may be lower or the same in classical ERP analysis (1,63, 66). On the other hand, P200 and N200 values of BD patients were found to be lower compared to patients with schizophrenia and healthy control subjects (67,68).

Power spectral density (PSD) is an electrophysiological parameter measured by resting-state magnetoencephalography. It is noteworthy that PSD is low in low frequency bands throughout the default mode network (bilateral medial prefrontal cortex, bilateral precuneus, left inferior parietal lobe, right temporal cortex-alpha band) and salience network in patients with BD. The fact that no change is observed in low beta or high beta activity suggests that PSD is unrelated to age and other clinical scales in BD patients. The variability of PSD in the delta, theta, and alpha bands of the default mode network and the salience network in BD patients supports the hypothesis that the switching mechanism between two networks is impaired and that there is vulnerability in terms of self-referential cognitive activity (69).

In a clinical study investigating the resting-state EEGs of 35 BD and 39 major depressive disorder (MDD) patients and 42 healthy control subjects, it was suggested that EEG may be a determinant in revealing cortical functional network involvement in these diseases. There was a significant difference between the groups in the high beta band. The study findings indicated that BD and MDD patients can be easily distinguished from healthy control subjects based on EEG, but the similar EEG features of BD and MDD patients make it difficult to differentiate between them (70).

In a study investigating the strength of connectivity of EEG functional networks in patients with BD and schizophrenia who have not yet started to receive any treatment, a P300 task was assigned to patients and the prestimulus/event-related connectivity strength (CS) was calculated in the theta band and globally. The low CS modulation in the theta band was noteworthy in both patients with BD and schizophrenia. It has been demonstrated that antipsychotic, lithium, benzodiazepine and anticonvulsant treatment modalities have no effect on the CS value. The study findings revealed increased connectivity in the whole-brain network in patients with schizophrenia, unlike in patients with BD; suggesting the presence of a general hypersynchronous basal state that impairs cognition in these patients (71,73).

In terms of brain functional networks, the etiopathogenetic mechanism in BD is associated with damage to the corticolimbic and fronto-temporal networks (74). These networks perform cognitive and mood-regulating functions (75). Therefore, it is argued that the theta oscillations related to the cognitive event may be affected by this pathogenesis and that the variable theta responses are a neurophysiological indicator of cognitive involvement in BD (63,72).

Attention Deficit Hyperactivity Disorder (ADHD)

Attention Deficit Hyperactivity Disorder (ADHD) is a highly inherited neurodevelopmental disorder and is associated with a wide range of cognitive and neurophysiological disorders (77). Functional MRI (fMRI) studies revealed abnormalities in networks that affect executive functions (hypoactivation in the fronto-parietal network) and in networks other than executive functions (hyperactivation in the default mode network and ventral salience network). The strong spatial resolution of fMRI enabled drawing the boundaries of these regions (77–79,84).

Resting-state EEG studies conducted with ADHD patients revealed an increase in power in delta and theta low frequency bands, whereas a

decrease in power in alpha and beta fast frequency bands was observed (76,80). These findings support the presence of a hypo-aroused brain state and low vigilance in ADHD patients. Further research revealed that very low frequency (<0.2 Hz) activity, which is an indicator of the default mode network, also decreases in children and adults with ADHD (77,81).

It was observed that the theta/beta ratio increased in patients with ADHD compared to healthy control subjects. The increase in theta/beta ratio can be considered an indicator of the imbalance between slow and fast EEG rhythms in patients with ADHD and may be used in diagnostic evaluations (82).

The most striking findings are low P3 and Cue-P3, which are thought to be the neurophysiological reflection of attentional orientation and task-related attention deficits that are evident in the clinical picture (83).

As initialevidence from studies using more detailed cognitive and neurophysiological approaches emerged, also emerged the thinking that the underlying mechanisms in ADHD are impairments in stimulus processing (phase consistency of theta event), selection secondary to attention (decreased alpha event-related desynchronization (ERD) following the target), and motor preparation (low target-related alpha and beta OID) (84). In one of the recent review articles published on this subject, the importance of electrophysiological examinations in ADHD was emphasized (81).

Obsessive Compulsive Disorder

Obsessive compulsive disorder (OCD) is a neuropsychiatric disorder characterized by obsessions (troubling repetitive thoughts) and accompanying compulsions (repetitive behaviors/mental rituals) (85). There is still no physical biomarker for the definitive diagnosis of OCD, which has a very high prevalence, and imaging methods also fall short in diagnosing OCD (86,87). It is self-evident that detailed description of the brain activity in the context of OCD will be guiding for neuromodulatory interventions to be developed for the diagnosis and treatment of OCD.

Many spontaneous EEG studies have been performed in individuals with OCD so far. It is thought that the underlying pathology is orbitofrontal-striatal-pallido-thalamic circuit dysfunction and that the electrophysiological changes observed in relation to OCD are a reflection of this dysfunction. This circuit consists of the dorsolateral prefrontal cortex, orbitofrontal cortex, anterior cingulate cortex, supplementary motor area and the basal ganglia (88).

A consistent elevation has been shown in frontal delta and theta power in patients with OCD (89). In addition, a decrease in alpha power was observed in patients with OCD compared to the healthy control subjects. Further analysis of decrease in alpha power in patients with OCD revealed that this decrease was observed in the fronto-temporal areas in OCD patients with uncertainty obsession and in the parieto-occipital areas in OCD patients with checking compulsion (90).

One of the most common abnormalities in the EEG of patients with OCD is the increase in event-related negativity (ERN) (89). This component is observed in anterior areas when patients give an incorrect answer in a trial. The ERN represents the probability that the outcome will be worse than expected. Principal component analysis of single ERN trials and simultaneous fMRI recordings revealed that the generator of this component in the brain is the anterior cingulate cortex (ASC) (91). As a matter of fact, abnormal spontaneous theta activity has been observed in the ASC region of patients with OCD, indicating that patterns of dysfunctional activity in OCD can be observed in different experimental paradigms.

There are contradictory results in the ERP literature during oddball tasks in the context of OCD. While some studies reported a shorter P300 latency in OCD patients than in healthy control subjects (92,94), other studies did not find a difference between the groups (70,86,94). The difference in latency between the groups seems to emerge when the task difficulty increases. However, this difference disappeared in studies with larger samples (94,95,96). Similarly, there are studies that reported an increase in P300 amplitude in OCD patients than in healthy control subjects (97,98), in addition to studies that reported a decrease (70,97) and the studies which did not find a difference between the groups (86,98). Based on all these, it has been concluded that ERPs are not suitable candidates for the determination of OCD-specific activity patterns. As a matter of fact, ERO replaced ERP in obtaining important findings that cannot be obtained with traditional ERP (5,19).

CONCLUSION

Since the beginning of the 1990s, medicine is attempting to comprehend the nature of cognitive processes based on electrophysiological properties measured in physiological and pathological situations using different methodologies. Electrophysiological assessment in the context of neuropsychiatric diseases is becoming increasingly important in terms of its potential as a diagnostic biomarker as well as a mediator for interventions that can change the course of the disease. The fact that it is easily accessible and that its temporal resolution renders dynamic examination of the brain possible makes electrophysiological evaluation indispensable for deciphering neurodegeneration.

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