arithmetic involves complex cognitive processing, essentially working memory. Hence the importance of adopting a therapeutic approach incorporating not only pharmacological treatments, but also cognitive remediation therapies

Disclosure of Interest: None Declared

EPV1629

Slow EEG potentials as predictors of cognitive impairment in patients with clinical high risk for schizophrenia

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Introduction: Cognitive deficits in schizophrenia are associated with impaired predictive processes, however, the neural mechanisms of these impairments at the early stages of the disease are poorly understood. A modified memory-guided saccade task can be informative for studies in this field. The contingent negative variation (CNV) slow negative potentials (SNP1, 2, 3 waves) in 1000-ms interval before a memory-guided response are considered to be neural correlates of attention, memory, motor, and inhibitory predictive processes.
Objectives: We aimed to assess the CNV-type slow negative event-related potentials (ERP) during the latent period before the signal to perform remembered saccades in patients with clinical high risk (CHR) for schizophrenia.
Methods: An electroencephalogram (EEG) from 24 electrodes and electrooculogram of horizontal eye movements were recorded in 16 patients with CHR and 18 healthy controls. The participants had to remember the location of a peripheral stimulus (PS, 150ms) and perform a saccade or antisaccade (50% probability) when the central fixation stimulus (CFS) was turned off after a delay period of 2800–3000 ms. The CFS shape (cross or circle) defined a motor response type: saccade or antisaccade.
Results: The task performance (assessed based on response latency and errors) was worse in CHR patients compared to controls. In the antisaccade condition, SNP1 was faster in CHR patients compared to controls possibly reflecting attention deficits in CHR patients. The SNP1 amplitude peaks were equally distributed across the EEG leads in CHR patients but were located predominantly in frontal and central leads in controls. Diffuse representation of the amplitude peaks may reflect a compensatory involvement of posterior temporal and parietal-occipital cognitive control networks at the early stages of schizophrenia. At the last 300 ms of the delay period, the late SNP3 wave was shorter before memory-guided antisaccades compared to saccades only in patients. This may reflect the violation of predictive attention processes as well as proactive inhibition deficits, that are well-known in schizophrenia, in CHR patients.
Conclusions: Based on our data we consider the SNP1 and SNP3 components in the memory-guided saccade task to be potentially significant neurobiological markers of cognitive control at the early stages of schizophrenia.
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EPV1632

Neuropsychiatric Circuitry and Receptor Dysregulation in the Pathogenesis of Bruxism

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Introduction: Bruxism, characterized by the grinding and clenching of teeth, is often associated with psychiatric disorders such as anxiety and stress. Bruxism not only results in significant dental pathology but can also contribute to underlying neurophysiological disturbances.
Objectives: To elucidate the relationship between bruxism and psychiatric medication by focusing on the neurophysiological mechanisms involved and the resultant dental pathologies.
Methods: A comprehensive literature review was conducted using databases such as PubMed, PsycINFO, and Google Scholar, focusing on studies from the last decade that investigate the association between bruxism, psychiatric medications, and neurophysiological factors. The review included clinical studies, neuroimaging research, and behavioral analyses.
Results: The findings indicate a strong association between bruxism and the use of psychiatric medications, particularly antidepressants and antipsychotics. Neurophysiological studies reveal dysregulation in neurotransmitter systems, notably dopamine and serotonin, which play critical roles in both bruxism and the effects of psychiatric medications. This dysregulation affects motor control circuits and stress response pathways in the central nervous system, leading to involuntary teeth grinding and clenching.
Table 1: Neurophysiological Mechanisms

Mechanism	Description
Dopamine Dysregulation	Inhibition of dopaminergic neurons leads to dysregulation of motor control and contributes to spontaneous movement of jaw muscles.
Serotonin Imbalance	Excess serotonin enhances excitatory neurotransmission and disrupts dopaminergic pathways, contributing to increased anxiety and masseter muscle hyperactivity.
Autonomic Nervous System	Hyperactivity in the sympathetic branch, driven by chronic stress, leads to increased arousal and muscle tone causing bruxism.

Table 2: Dental Pathologies Resulting from Bruxism

Pathology	Description
Tooth Wear	Enamel erosion due to repetitive grinding, leading to dentin exposure.
Fractures	Microfractures in teeth from constant pressure, progressing to severe cracks.
TMJ Disorders (TMJD)	Chronic bruxism contributes to TMJD, characterized by pain and joint dysfunction.
Periodontal Damage	Excessive force on teeth exacerbates periodontal issues, leading to gum recession.

Conclusions: Bruxism is both a symptom and a potential side effect of various psychiatric medications, rooted in neurophysiological disturbances. The interplay between dysregulated neurotransmitter

systems, psychiatric medications, and resultant dental pathologies highlights the need for integrated dental and psychiatric care. Effective management of bruxism through targeted dental interventions and tailored psychiatric treatments can significantly improve both dental health and psychiatric well-being.

Disclosure of Interest: None Declared

EPV1633

Exploring the Links Between Cognitive Deficits and EEG Spectral Density in Schizophrenia

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Introduction: The research revealed significant correlations between cognitive performance, assessed by psychometric scales, and variations in frequency bands in the electroencephalography (EEG), illustrating the link between electroencephalographic activity and cognitive functions in schizophrenic patients
Objectives: Our study aimed to explore the relationship between the electroencephalographic spectral power of slow frequency bands (delta and theta) and cognitive functions in patients with schizophrenia by comparing them to healthy subjects.
Methods: We conducted a cross-sectional, descriptive, and analytical study involving 15 schizophrenic patients and 15 healthy controls. The study was performed at the Psychiatry Department "C" outpatient unit at Hedi Chaker University Hospital in Sfax in Tunisia. We used the Arabic literary version of the Screen for Cognitive Impairment in Psychiatry (SCIP) scale to assess cognitive functions. Participants underwent a standard wakefulness EEG with eyes closed at the Functional Explorations Department of Habib Bourguiba Hospital in Sfax in Tunisia. Linear regression analysis was used to examine correlations between the total SCIP score and the absolute spectral density (ASD) values of EEG oscillations.
Results: Linear regression analysis revealed a negative correlation between the total SCIP score and the delta wave ASD at T5 (left temporal) ($r = -0.37$; $p = 0.025$) and theta wave ASD at Fp2 (right prefrontal) ($r = -0.131$; $p = 0.006$). A positive correlation was found between theta wave ASD at F3 (left frontal) ($r = 0.125$; $p = 0.02$) and the total SCIP score. It revealed a negative correlation between the total SCIP score and the age of onset of schizophrenia ($r = -0.647$; $p = 0.001$).
Conclusions: These results suggest that theta and delta power at rest, as measured by EEG, may serve as potential biomarkers for cognitive deficits in patients with schizophrenia. These findings could contribute to a better understanding of the neurophysiological basis of cognitive alterations associated with this condition.

Disclosure of Interest: None Declared

EPV1634

Distinguishing Quantitative Electroencephalogram Findings between Panic Disorder and Generalized Anxiety Disorder

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Introduction: It is important to have early diagnosis and early intervention for generalized anxiety disorder(GAD) and panic disorder(PD). However, it is difficult to distinguish GAD from PD. Neurobehavioral markers that differentiate GAD and PD would be helpful to refine classification schemes based on neurobiological measures.
Objectives: The aim of this study is to determine the distinguishing neurophysiological characteristics between generalized anxiety disorder and panic disorder using quantitative EEG.
Methods: The study included 36 patients with GAD and 25 patients with PD. Resting vigilance controlled EEG recordings were assessed at 64 electrode sites according to the international 10/20 system. QEEG were compared between GAD and PD groups by frequency bands (delta 1-3 Hz, theta 4-7 Hz, alpha 8-12 Hz, beta 12-25 Hz, high beta 25-30 Hz, gamma 30-40 Hz and total 1-40 Hz) made by spectral analysis.
Results: The absolute powers of theta and alpha bands at the frontal area differed between GAD and PD group. The absolute power of the theta activity was decreased in FP1 and FP2 ($p < 0.05$) and the absolute power of the alpha activity was decreased in F3 ($p < 0.05$) in cases with GAD compared to PD.
Conclusions: The differences in QEEG power suggest that underlying pathophysiologic mechanisms may be different between GAD and PD. The findings that the decreased absolute powers of the theta and alpha activity at the frontal area in GAD may be the main neurophysiological characteristics of the GAD and help to have early differential diagnosis between GAD and PD.

Disclosure of Interest: None Declared

Psychosurgery and Stimulation Methods (ECT, TMS, VNS, DBS)

EPV1635

Two-Year Overview of Theta-Burst Stimulation for Treatment-resistant Depression: Assessing Efficacy and Outcomes

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Introduction: Major depressive disorder (MDD) is a very common and debilitating disorder. MDD accounts for 4.3% of the global burden of disease, is among the largest single causes of disability worldwide, and is an important cause of premature death. Depression expands its negative influence in all aspects of life, being estimate that 12 billion productive workdays are lost every year to depression and anxiety.
On top of that, non-response to first line pharmacological and psychotherapeutic treatments are substantial, with treatment-resistant depression (TRD) affecting approximately one third of these patients. These patients are thus candidates for non-invasive neuromodulation procedures such as repetitive transcranial magnetic stimulation (TMS), included in all major treatment guidelines.
Objectives: With this work we intend to present a descriptive analysis of the efficacy of the intermittent theta burst TMS (iTBS)