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

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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