EPV1626

Exploring Heart Rate Variability Changes Following Respiratory Training in Individuals with Panic Disorder

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Introduction: Studies reported an association between panic disorder (PD) and diminished heart rate variability (HRV) (Zhang *et al.* J Affect Disord 2020; 15, 267 297-306), potentially heightening their susceptibility to cardiovascular mortality and diminished quality of life. Preliminary research has demonstrated that HRV-biofeedback training can enhance the regulation of autonomic function in both healthy individuals (Schumann *et al.* Front Neurosci 202;15 691988) and patients with anxiety (Herhaus *et al.* Psychosom Med 2022; 84, 2 199-209).

Objectives: We examined the impact of HRV-biofeedback training intervention on autonomic activity modulation among outpatients with PD with or without agoraphobia (AGO).

Methods: We conducted a retrospective observational pilot study including 10 outpatients (five females and five males; median age = 35.5 years) diagnosed with PD (9 with comorbid AGO and 1 without). The clinician-administered Panic Associated Symptoms Scale (PASS) was used to assess PD and AGO severity. All the included patients underwent a physiological assessment to determine HRV parameters and breathing rate during a 5-minute resting condition at baseline (T0) and follow-up (T1) assessment. Between T0 and T1 assessments, all patients underwent HRV-biofeedback training. Pre-post comparisons were conducted using the Wilcoxon Signed Ranks Test. To evaluate the impact of psychotropic medications, we used the Mann-Whitney U test to compare HRV parameters and disorder severity between patients with and without medications, at T0 and T1. Significance level was set at $p<0.05$.

Results: HRV-biofeedback training among individuals with PD with or without AGO fostered notable enhancements in RMSSD ($Z = -2.08$; $p < 0.01$), SDNN ($Z = -2.5$; $p = 0.01$), and LF ($Z = -2.09$; $p = 0.04$) parameters at T1 compared to T0. Furthermore, a significant decrease in heart rate ($Z = -2.19$; $p = 0.03$) was shown. These enhancements didn't seem related to psychopharmacological medications and disorder severity.

Conclusions: Despite limitations (e.g., small sample, no control group, lack of comorbidity and T1-PD severity assessment, missing inclusion/exclusion criteria, and unaccounted confounding variables affecting HRV), our study suggests that HRV biofeedback training may improve autonomic regulation in PD patients, aligning with existing literature (Herhaus *et al.* Psychosom Med 2022; 84, 2 199-209). Further research is needed to determine to what extent bio-feedback training can help to reduce panic symptoms and disorder severity.

Disclosure of Interest: None Declared

EPV1628

Analysis of interhemispheric EEG spectral variation at rest and after mental arithmetic task in schizophrenic patients compared to healthy controls

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Introduction: Electroencephalogram (EEG) has emerged to be valuable for understanding the neurophysiological mechanisms underlying cognitive dysfunctions in psychiatric disorders. EEG mental arithmetic enables to assesse cognitive abilities in schizophrenic patients, by inducing brain activity that can be observed through frequency band analysis

Objectives: This study aimed to assess the absolute spectral density (ASD) of various EEG frequency bands in schizophrenic patients and healthy controls, at rest and during a mental arithmetic task, in order to identify specific neural differences

Methods: We conducted a cross-sectional, descriptive, and analytical case-control study involving 15 schizophrenic patients and 15 healthy controls. The study was carried out at the outpatient unit of Psychiatry Department "C" at Hedi Chaker University Hospital in Sfax, Tunisia. Participants underwent a standard wakefulness EEG with eyes closed at the Functional Explorations Department of Habib Bourguiba Hospital in Sfax, Tunisia. Each participant also performed a mental calculation test during the EEG recording

Results: The ASD of the different EEGs was studied for each frequency band in the different cerebral lobes (Table 1). At rest, in schizophrenic patients, a significant difference was found between the mean ASD of the delta, theta, and beta1 frequency bands in the right and left occipital regions. Also, frequency band activity was more diminished in the left occipital regions than in the right. After mental calculation, the interhemispheric asymmetry disappeared in schizophrenic patients. In the control group, no significant differences were found between the mean ASD for any frequency band in the right and left frontal, temporal, and occipital regions, either at rest or after mental calculation (Table 1)

Table 1 : Comparison of EEG absolute spectral density between hemispheres in schizophrenics and controls at rest and after mental calculation task

	Schizophrenics		*p=*	*Controls*		*p=*
	Right	*Left*		*Right*	*Left*	
At rest						
DELTA Occipital	30,44± 26,37	22,74±19,34	**0,021**	5127,7±19031	5111,41±18968	0,373
THET Occipital	58,17±47,67	45,67±44,88	**0,023**	3856,8±12450	3863,76±12458	0,721
BETA 1 Occipital	7,22±4,25	6,24±3,77	**0,002**	497,9±1854	498,26±1860	0,854
After mental calculation task						
DELTA Occipital	22,62±17,8	24,84±33,76	0,725	4970,14±15006	4979,39±15068	0,762
THETA Occipital	46,35±41,48	62,62±96,01	0,442	1895,46±5301	1849,43±5245	0,766
BETA1 Occipital	5,76±3,45	5,43±3,17	0,466	934,81±3152	919,04±3093	0,328

Conclusions: These results underline the importance of considering cerebral lateralization in the diagnostic and therapeutic approach to schizophrenia. It is important to note that mental

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