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Abstracts in alphabetical order delivered by the lecturers

EXPERTS SESSION

Michał Górecki, Piotr Czarnecki

Advanced reconstructive strategies in high-energy brachial plexus and peripheral nerve injuries - interdisciplinary collaboration between a neurophysiologist and a hand or nerve surgeon.

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High-energy injuries of the brachial plexus or high-level lesions of peripheral nerves are associated with prolonged regeneration times of effector structures, particularly muscles, which can lead to irreversible atrophy and permanent functional deficits. Advanced microsurgical reconstructive techniques—such as **end-to-side (ETS) nerve transfer**, **reverse end-to-side (RETS) reconstruction** (the so called *supercharge* or *babysitting* procedure), and **direct nerve transfers**—are designed to shorten the interval between injury and reinnervation. These procedures either accelerate functional recovery or maintain continuous stimulation of the target muscles prior to the arrival of regenerating native axons. The choice of surgical technique and type of nerve transfer depends on the level and nature of the peripheral nerve or brachial plexus injury.

Nerve transfers typically yield predictable and functionally meaningful outcomes within a relatively short time frame and are particularly beneficial in complex cases such as **spinal nerve root avulsions**. However, these procedures are technically demanding and require meticulous stepwise planning. The maximal functional recovery is generally observed within **1–2 years postoperatively**.

Preoperative assessment of **nerve conduction** and the **degree of effector muscle denervation** is crucial for selecting the optimal surgical approach. **Intraoperative neuromonitoring**, employing direct nerve stimulation and **electromyographic (EMG) activity analysis**, provides valuable real-time feedback during surgical decision-making. Additionally, **non-invasive techniques** such as **transcranial or transvertebral magnetic stimulation** with **motor evoked potential (MEP)** assessment enable evaluation of neural conduction and regeneration efficacy in the postoperative period.

An **interdisciplinary collaboration** between **neurophysiologists** and **hand or peripheral nerve surgeons** is essential to ensure optimal patient outcomes. This partnership facilitates comprehensive preoperative planning, selection of the most appropriate reconstructive strategy, and objective assessment of therapeutic and regenerative effects.

Juliusz Huber

Which spinal cord neural efferent pathways can be responsible for the mechanism of action of the reflex locomotion? Neurophysiological analysis based on the results of the motor evoked potential recordings in healthy subjects.

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Neurorehabilitation based on the activation of reflex locomotion is considered to be transmitted by supraspinal mechanisms to the spinal locomotor centres following stimulation of the Ia, Ib, II, cutaneous, interosseal and joint afferent fibres. Most likely, the afferent tracts to the thalamus and motor cortex, as well as pontomedullary reticular formation, are considered the substrates involved in transmitting the neuronal excitation or inhibition to the spinal motor centers in a recurrent way.
There is, however, another possibility of the direct activation of the spinal central pattern generators from the afferent sources in a di- or oligosynaptic way, and the leading role in neural crossed and uncrossed transmission may play the fibres of long propriospinal pathways interconnecting functionally cervical and lumbar enlargements.
Motor evoked potential recordings parameters of amplitudes and latencies from the upper and lower extremities muscles bilaterally under normal conditions, following magnetic stimulation at acromion level and transvertebrally at C3-C5 spinal levels for comparison, may reveal crossed and uncrossed mainstream patterns in neural transmission. Some results suggest engagement of a long axonal system responsible for the mainly crossed reflex control, having crossed projections at thoraco-lumbar levels.
Keywords: motor evoked potentials, neurorehabilitation, propriospinal neurons, mechanism of action

Małgorzata Reysner

Neurophysiological Basis of Pain Modulation through Regional Anesthesia: Insights from Current Literature

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Background: Pain is a dynamic neurophysiological phenomenon resulting from complex interactions between peripheral and central nervous system structures. Regional anesthesia, once considered a purely peripheral technique, is now recognized as a modulator of central pain processing. By interrupting nociceptive transmission, it influences spinal and supraspinal mechanisms responsible for sensitization, hyperalgesia, and long-term pain plasticity.
Objective: This narrative review aims to summarize current evidence on how regional anesthesia affects pain pathways at the neurophysiological level, with particular attention to peripheral receptor activity, dorsal horn modulation, and cortical reorganization.
Methods: A literature search was conducted in PubMed, Scopus, and Web of Science databases for studies published between 2010 and 2025 using keywords such as regional anesthesia, pain modulation, central sensitization, nociceptive pathways, and neuroplasticity. Both experimental and clinical studies were included to provide a comprehensive overview of current knowledge.
Results: Evidence indicates that regional anesthesia not only inhibits peripheral nociceptor activation but also attenuates dorsal horn hyperexcitability, decreases glial activation, and modulates the release of neurotransmitters such as glutamate and substance P. Moreover, electrophysiological and neuroimaging studies suggest that prolonged nociceptive blockade can induce cortical reorganization and transient alterations in somatosensory evoked potentials (SSEPs), reflecting adaptive plasticity of the pain matrix.
Conclusions: Regional anesthesia exerts profound neurophysiological effects that extend beyond local analgesia, influencing both spinal and cortical pain processing. Understanding these mechanisms provides a neurobiological foundation for its role in preventing central sensitization and chronic postsurgical pain. Further interdisciplinary

studies combining neurophysiology and clinical anesthesia are essential to optimize individualized pain therapy.
Keywords: Regional anesthesia, pain modulation, neurophysiology, central sensitization, neuroplasticity, nociceptive pathways

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Anesthetic Management in Scoliosis Surgery with Intraoperative Neuromonitoring: From Total Intravenous Anesthesia to Regional Blocks
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Scoliosis correction surgery presents unique anesthetic challenges due to extensive tissue dissection, risk of spinal cord injury, and the necessity of preserving intraoperative neuromonitoring (IONM). The choice of anesthetic regimen profoundly influences the quality of motor-evoked potentials (MEPs) and patient safety. This narrative review summarizes current evidence on anesthetic techniques used in scoliosis surgery with neuromonitoring, with particular emphasis on the Erector Spinae Plane Block (ESPB) and perineural adjuvants such as dexamethasone and dexmedetomidine. Total intravenous anesthesia (TIVA) with propofol and remifentanil remains the gold standard for reliable MEP and somatosensory-evoked potential (SSEP) recordings. Volatile anesthetics, in contrast, reduce MEP amplitude and prolong latency, limiting their use. Recent randomized trials have demonstrated that ESPB provides adequate postoperative analgesia, attenuates the inflammatory and stress response (measured by NLR and PLR), and does not interfere with MEP integrity.
Furthermore, perineural dexamethasone enhances MEP amplitude and prolongs analgesia, whereas dexmedetomidine may suppress MEPs and induce hemodynamic instability. Integration of regional anesthesia into multimodal analgesic strategies allows optimal pain control, reduced opioid consumption, and improved recovery without compromising neuromonitoring. Future research should focus on the molecular pathways underlying the interaction between anesthetic agents, neural conduction, and oxidative stress in spinal surgery.
Keywords: scoliosis surgery, intraoperative neuromonitoring, erector spinae plane block, motor-evoked potentials, dexamethasone, dexmedetomidine, TIVA, regional anesthesia, oxidative stress

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Thoracic outlet syndrome (TOS) or C8 cervical root injury: a neurophysiological differential diagnosis.
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Thoracic outlet syndrome presents in several variants and is characterized by the compression of the brachial plexus and subclavian vessels. A distinction is made between vascular TOS and neurogenic TOS. In vascular TOS, compression affects the subclavian artery or vein and is primarily manifested during rotation and abduction of the arm. Neurogenic TOS involves compression of the brachial plexus in its inferior trunk. Causes of compression include abnormal anatomical relationships within the thoracic outlet. These include the presence of an accessory cervical rib and an elongated transverse process of the C7 vertebra. TOS can occur when the nerve and vascular bundle are compressed by the scalene muscles, as well as in costoclavicular syndrome, where the

abnormal anatomical relationship between the clavicle and the rib, known as the costoclavicular isthmus, causes compression of the neuromuscular bundle. In cases of hyperabduction syndrome (Wright's syndrome), the neurovascular bundle is irritated by the pectoralis minor tendon or the coracoid process of the scapula. Given the complexity of the clinical symptoms, with dominant neuropathic pain in the projection of the lower trunk of the brachial plexus, it should also be differentiated from damage to the C8-Th1 spinal root. In neurophysiological studies, the primary examination in cases of neurogenic TOS involves assessing both sensory and motor fibers of the median nerve, ulnar nerve, and medial cutaneous nerve of the arm. Abnormalities in electroneurography confirm the diagnosis of neurogenic TOS. In the differential diagnosis between neurogenic TOS and damage to the C8-T1 spinal roots, the conduction parameters in the sensory fibers of the aforementioned nerves play a central role, as well as supplementing the electroneurographic examination with an electromyography evaluation of the muscles innervated by the C8-T1 root domain.
Keywords: neurogenic TOS, cervical roots, ENG, EMG

STUDENTS SESSION

Abstracts in order delivered by the lecturers

REVIEW ARTICLE

Can Heading a Ball Cause Dementia? The Correlation of TBI and Neurodegenerative Diseases.

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Introduction. The link between contact sports and neurodegenerative diseases has become an increasingly prominent topic of scientific and public interest. Repetitive head impacts (RHIs) and traumatic brain injuries (TBIs), common in contact sports, are associated with long-term neurological consequences, such as chronic traumatic encephalopathy (CTE) and Alzheimer's disease (AD). This has raised concerns among athletes, sports organizations, and medical professionals about the safety of contact sports and the need for preventive measures.
Aim. This review provides an overview of recent advances in understanding the prevalence of neurodegenerative diseases in contact sports, related morphological changes, diagnostic methods and biomarkers, risk factors, and current therapeutic strategies.
Material and Methods. A PubMed search was conducted for articles published within the last five years using the following keywords: football, soccer, heading, Alzheimer, TBI, and RHI.
Results. Contact sports are linked to an increased risk of neurodegenerative diseases. Among former soccer players, the risk is increased 3 times. Each additional year of rugby play raises CTE risk by ~14%. In American football, CTE can develop in players younger than 30. The outcomes of TBI depend on prior medical history and non-modifiable factors. CTE and AD are the most frequent neurodegenerative outcomes, while other complications include depression and insomnia. Diagnostic methods involve biochemical analyses of serum and cerebrospinal fluid (CSF), histopathology, mRNA profiling, and neuroimaging techniques such as PET and MRI. Currently, no disease-modifying treatment for CTE exists. Neuroprotective agents aim to reduce neuronal damage, while

novel techniques such as low-intensity pulsed ultrasound (LIPUS) and photobiomodulation show promise. Early rehabilitation remains essential.
Conclusions. Repetitive TBI in athletes is associated with neurodegenerative processes. Key challenges include insufficient diagnostic standardization and the lack of effective neuroprotective therapies, highlighting the need for further research.
Keywords: soccer, football, heading, Alzheimer, TBI, RHI, neurodegenerative disease
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Glymphatic System Disorders in Alzheimer's Disease - A Literature Review
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Introduction. Dysfunction of the glymphatic system (GS) impairs the removal of metabolites from the central nervous system, promoting neurotoxic change and the progression of Alzheimer's disease (AD). AD is characterized by the abnormal accumulation of neurotoxic amyloid-β and tau proteins within the brain.
Aim. In this review, we aim to summarize current knowledge on GS dysregulation and its association with AD development and progression.
Material and Methods. A systematic literature search was carried out in PubMed, Scopus, EBSCOhost, Web of Science and Embase to identify English-language original studies on GS alterations in AD in humans published within the last five years. Search terms included "glymphatic system," "glymphatic system disorders," "Alzheimer," and "Alzheimer's disease," combined with AND; open-access filters were applied when possible. After screening and eligibility assessment, 42 original articles were included. One review article was used as a background reference.
Results. According to our research AD appears to be associated with GS impairment. The most frequently reported indicators of GS dysfunction were the Diffusion Tensor Image Analysis ALong the Perivascular Space (DTI-ALPS) index, choroid plexus (CP) enlargement, and enlarged perivascular spaces (EPVS). A low DTI-ALPS index is used as an indicator of GS dysfunction. Patients with AD show significantly reduced DTI-ALPS index and an increased number of EPVS than controls. CP plays a crucial role in amyloid-β clearance, and CP enlargement is considered a manifestation of its dysfunction, as observed in patients with AD. Studies suggest that impaired CSF drainage may promote amyloid-β accumulation in the brain. GS dysfunction may also be indicated by genetic factors, perivascular inclusions, and physiological modulations.
Conclusions. The study demonstrated that dysfunction of the GS disrupts the drainage of cerebrospinal and interstitial fluid, leading to impaired removal of metabolites, accumulation of amyloid-β, and progressive neurodegeneration in AD.
Keywords: glymphatic system, neurodegeneration, Alzheimer's disease
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Bicycle Sign – Will Differentiating Parkinson's from Parkinsonism Be Easier in the Netherlands Than in Poland?

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Introduction. In extrapyramidal disorders, disturbances of alternating movements and postural reflexes are common, leading to impairments in both everyday mobility and cycling. The speed at which these impairments develop is important in differentiating Parkinson's disease (PD) from atypical parkinsonian syndromes (AP). Consequently, the loss of the ability to ride a bicycle called Bicycle Sign may serve as a useful clinical indicator in distinguishing PD from AP. However, its diagnostic value may differ between countries with a well-developed cycling culture, such as the Netherlands, and Poland, where cycling is mainly recreational.

Aim. The aim of the study was to assess whether differences in cycling culture between the Netherlands and Poland affect the diagnostic usefulness of the Bicycle Sign in distinguishing Parkinson's disease from atypical parkinsonism.

Material and Methods. The study was based on a review of literature available in the PubMed from 2011 to 2025 concerning Parkinson's disease, atypical parkinsonism and cycling culture in Poland and the Netherlands

Results. Patients with PD often retain the ability to ride a bicycle, whereas individuals with AP tend to lose this ability early in the disease course. In countries with a developed cycling culture like the Netherlands where cycling is the primary mode of transportation for many people, the loss of cycling ability is more quickly noticed and recognized by patients and clinicians, which may increase the sensitivity of bicycle sign in differentiating PD from AP.

Conclusions. The Bicycle Sign may represent a simple and useful indicator that can facilitate diagnosis and aid in distinguishing PD from AP. In Poland, compared with the Netherlands, its sensitivity is likely lower due to a less popular cycling culture. In these circumstances, it may be necessary to identify alternative markers that could similarly support in the diagnostic process.

Keywords: Bicycle Sign, Parkinson's disease, atypical parkinsonism, cycling culture, Netherlands, Poland.

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Earwax, saliva and skin - (a little gross) new era of diagnosis of neurological diseases?

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Introduction: The diagnosis of neurodegenerative diseases such as Parkinson's disease (PD) and Alzheimer's disease (AD) still relies on methods used at advanced stages, including clinical evaluation, neuroimaging, and cerebrospinal fluid analysis. These techniques are often invasive, expensive, and applied only after irreversible neuronal loss has occurred. Therefore, there is an urgent need for accessible, cost-effective, and non-invasive biomarkers enabling earlier diagnosis. Recent studies focus on unconventional biological materials such as saliva, skin, and earwax, which may reflect central nervous system pathology and offer new perspectives for early detection.

Aim: To review and summarize current evidence on the diagnostic potential of biomarkers identified in saliva, skin, and earwax for Alzheimer's and Parkinson's diseases.

Material and Methods: A literature review was conducted using the PubMed database with the following keywords: "biomarkers", "Parkinson's disease", "Alzheimer's disease", "saliva", "skin", "earwax". Only human observational studies and case reports published between 2014 and 2025 were included.

Results: In Alzheimer's disease, salivary studies demonstrated increased concentrations of amyloid-β42, tau protein, lactoferrin, and selected metabolites. In Parkinson's disease, phosphorylated α-synuclein (PASH) deposits in cutaneous nerve fibers showed high sensitivity and specificity, distinguishing PD patients from controls and persisting over time. Their presence in idiopathic REM sleep behavior disorder supports the use of skin biopsy for early diagnosis. Moreover, lipidomic analysis of earwax revealed specific lipid signatures differentiating PD patients from healthy individuals, with diagnostic accuracy exceeding 90%.

Conclusions: Saliva, earwax, and skin constitute promising, non-invasive sources of biomarkers that reflect neurodegenerative pathology. Their accessibility, stability, and correlation with brain changes may revolutionize early diagnosis and monitoring of Alzheimer's and Parkinson's diseases.

Keywords: biomarkers, Parkinson's disease, Alzheimer's disease, saliva, skin, earwax, α-synuclein, amyloid β42, tau protein

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Genetic Landscapes of Paediatric Drug-Resistant Epilepsy: Implications for Vagus Nerve Stimulation Outcomes

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Background: Genetic etiologies represent an increasingly recognised cause of paediatric drug-resistant epilepsy (DRE). Vagus nerve stimulation (VNS) constitutes an established neuromodulatory therapy, offering seizure reduction in children not amenable to resective surgery. However, evidence regarding its efficacy and antiseizure medication (ASM) modulation in genetically determined epilepsies remains limited and inconclusive. Objective: To compare the clinical effectiveness of VNS in paediatric DRE with genetic versus non-genetic etiologies, evaluating seizure reduction and changes in ASM pharmacotherapy burden.
Methods: The study was designed as a retrospective, single-center cohort study of 18 children (4–18 years) who underwent VNS implantation. Eight patients had genetically determined epilepsy, while ten presented with non-genetic causes. Seizure frequency reduction, ASM count, and exposure by mechanistic class were assessed pre- and post-implantation. Statistical comparisons were performed using non-parametric tests, with significance set at $p<0.1$.
Results: At a median follow-up of 15 months, the overall ≥50% responder rate was 61%. The non-genetic cohort demonstrated greater median seizure reduction (60.0%) compared with the genetic group (20.0%) ($p=0.09$). Reduction in ASM burden was observed in 33% of patients overall, predominantly in non-genetic cases (50% vs 13%) ($p<0.1$). Post-implantation calcium-channel modulator use differed between groups ($p=0.036$), with increased ion-channel ($p=0.002$) and decreased calcium-channel modulator (p=0,041) exposure observed within the cohort.
Conclusion: VNS is a safe and effective neuromodulation therapy for paediatric DRE irrespective of etiology. Despite modest response rates in genetic epilepsies, VNS may improve seizure control and neurodevelopmental outcomes in carefully selected patients, underscoring the importance of integrating genetic testing into neuromodulatory treatment pathways.

Keywords: Vagus Nerve Stimulation; Neuromodulation; Pediatric Drug-Resistant Epilepsy

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The use of AI in epilepsy diagnosis - an overview of contemporary tools

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Introduction. Epilepsy is a chronic neurological disorder characterised by recurrent seizures, caused by abnormal neuronal activity. Diagnosis of epilepsy remains a major

challenge due to heterogeneity of seizure types, different clinical manifestations and overlapping symptoms with other disorders. Recent advances in artificial intelligence (AI), particularly in convolutional neural networks (CNN) and support vector machines (SVM), have introduced new opportunities, which can help improve diagnostic accuracy.
Aim. The aim of this literature review is to summarize and critically evaluate the current applications of AI-based diagnostic methods of epilepsy. We focused on performance, clinical relevance and potential for integration into routine neurological practice.
Materials and Methods. A comprehensive literature search was conducted using PubMed for studies published between 2015 and 2025. Keywords such as ''convolutional neural network'', "support vector machine", "epilepsy" and "diagnosis" were used. A total of 60 studies were selected as sources for this discussion. The inclusion criteria comprised original research articles and literature reviews describing AI applications in electroencephalography (EEG) analysis, neuroimaging interpretation and seizure prediction.
Results. The review identified a growing number of AI tools used in technologies. Reported diagnostic accuracies differ between methods, but are particularly high in combination of traditional EEG data with hybrid models (CNN and SVM) giving over 90% in accuracy, sensitivity and specificity in selected studies.
Conclusions. AI technologies demonstrate significant potential to enhance the diagnostic process of epilepsy by improving analysis of neurophysiological data. Performance of AI in epilepsy diagnosis and management is promising, but there is still more to know, thus further validation and standardization are essential.
Keywords: epilepsy, machine learning, deep learning, convolutional neural network, support vector machine
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Brain-computer interfaces usage in neurorehabilitation of patients after stroke – systematic review

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Introduction. Stroke is one of the leading causes of disability worldwide, requiring long-term rehabilitation. Nowadays, neurofeedback (NF) and brain-computer

interface (BCI) based on electroencephalography (EEG) give opportunities for effective and accurate controlled neurorehabilitation tools.
Aim. To assess the therapeutic effect of EEG-based BCI usage in the process of neurorehabilitation of patients after stroke. The influence of neuroplasticity on motor functions after stroke will be evaluated.
Material and Methods. The systematic review was conducted in accordance with the PRISMA protocol guidelines. The comprehensive literature search was performed across 5 databases. Ultimately, 37 articles were included in the systematic review.
Results. The participant cohorts included both healthy individuals and patient groups, specifically those with chronic stroke. The review focused on methods described in the included studies with an emphasis on the most recent research. The interventions primarily utilized therapeutic sessions employing EEG-BCI, often incorporating selected techniques such as Virtual Reality (VR), Functional Electrical Stimulation (FES), Motor Imagery (MI), and Action Observation (AO). Most studies referred to the motor rehabilitation process with analysis of upper or lower limbs functionality. To access the therapeutic effects, different clinical scales such as Fugl-Meyer Assessment Upper Extremity (FMA-UE) or Lower Extremity (FMA-LE) and a range of additional tests were performed. The usage of EEG provided monitoring of neurophysiological changes assessed differently throughout studies, often utilized parameters like event-related desynchronization (ERD) and synchronization (ERS), the sensorimotor rhythm (SMR) or the laterality index (LI). Most articles revealed that EEG-BCI neurorehabilitation ensure improvement in brain function and clinical outcome, frequently better than convectional rehabilitation methods.
Conclusions. There are indications confirming that usage of BCI-related neurorehabilitation methods improves outcomes of patients after stroke. Positive neurological impact depends on the used BCI method type. Multiplicity of analyzed EEG parameters impedes direct comparison of different rehabilitation trials' effects.
Keywords: Stroke, EEG, neurorehabilitation, biofeedback, neurofeedback
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Turmeric as a Potential Dietary Factor Influencing the Development of Parkinson's Disease: A review.

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Introduction: Parkinson's disease (PD) is a progressive neurodegenerative disorder. The number of affected people is increasing, still there is no curative agent. Dopamine replacement therapies such as L-dopa remain the cornerstone of Parkinson's disease treatment, but prolonged use leads to dyskinesia and does not halt neurodegeneration. Epidemiologic data suggesting the lower incidence of PD among the Southeastern Asian population in comparison to Western populations, warrants detailed analysis of dietary tendencies and allopathic treatments that may partially contribute to such discrepancy. A product that is widely used within Southeast Asia is turmeric, containing curcumin - an extensively researched phytochemical. Curcumin has been proposed to alleviate PD symptoms and possibly slow disease progression through its multifaceted biological activities.

Aim: This review examines the mechanisms by which curcumin may modulate the progression of Parkinson's disease, with emphasis on recently elucidated pathogenic pathways. Additionally, it summarizes the findings of clinical trials involving both animal and human subjects.

Material and Methods: Relevant literature - three human clinical studies and several animal investigations - was identified via PubMed and Google Scholar.

Results: Curcumin was observed to attenuate dopaminergic denervation and motor dysfunctions in mice while also improving the state of gastrointestinal barrier and possibly inhabiting ferroptosis in dopaminergic neurons of affected rodents. Moreover, curcumin mitigated pathological changes in the rat cerebellum, a structure recently associated with PD. Two human studies demonstrated improvements in non-motor symptoms, whereas the third one reported no significant alleviation of motor dysfunction by curcumin therapy.

Conclusions: Data from animal studies indicate that curcumin has a mitigating effect on Parkinson's disease pathomechanisms such as oxidative stress, inflammation and ferroptosis while also restoring gastrointestinal barrier. However, evidence from human studies is limited and inconclusive. Further investigation is required.

Keywords: Parkinson's disease, turmeric, curcumin, neurodegeneration, oxidative stress, ferroptosis, gut-brain axis.

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Exosomes as Carriers of Biomarkers in Mood Disorders: Searching for Exosomal Signatures Differentiating Major Depressive Disorder and Bipolar Disorder

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Introduction. Major depressive disorder (MDD; also known as clinical depression), and bipolar disorder (BD) share similar, overlapping clinical symptoms, making differential diagnosis challenging and, consequently, adequate therapy. Exosomes, ie. small extracellular vesicles carring that carry molecular cargo (miRNAs, proteins, metabolites) reflect the physiological state of their parent cell1. They are able to cross the blood-brain barrier and are stable in biofluids which makes them promising non-invasive biomarkers for neuropsychiatric disorders2.

Aim. The aim of this study is to summarize results of research for exosomal biomarkers in MDD and BD and to evaluate their differentiating potential.

Material and Methods. A structured literature (original papers published in 2020-2025) search was conducted in PubMed/MEDLINE, Google Scholar, Scopus, Embase, and Web of Science databases using the following search terms: exosomes, major depressive disorder, bipolar disorder, extracellular vesicles, differentiating MDD and BD.

Results. Serum-derived exosomes from patients with BD display distinct metabolomic profiles with 26 differentially expressed metabolites, predominantly affecting sugar and amino-sugar metabolic pathways6. A 15-metabolite random-forest panel derived from these data achieved high internal classification performance (training accuracy 0.838; test accuracy 0.971) and showed discriminative ability versus other psychiatric groups in the same cohort6. Co-expression network analysis of exosomal metabolites identified modules correlated with clinical variables, implying coordinated metabolic dysregulation in BD6. Plasma exosomal miRNA profiling studies have reported sets of dysregulated miRNAs in BD versus healthy controls (including several consistently reported down- and up-regulated miRNAs across cohorts), indicating altered neuroinflammatory and synaptic pathways8. In depressive disorders, reviews of EV-miRNA studies report altered EV cargo linked to HPA-axis regulation, neuroinflammation and synaptic plasticity, but direct head-to-head exosome studies discriminating BD from MDD remain scarce1,2. Overall, promising exosome-derived metabolite and miRNA signals exist, yet most findings are preliminary, derived from small cohorts, and lack independent external validation 1,2,6.

Discussion. Exosomal cargo reflects key pathophysiological mechanisms, ie. neuroinflammation, HPA-axis dysregulation and altered synaptic plasticity. There are some promising findings, however, there is still room for larger studies. Standardization of EV isolation and data normalization is essential for further progress2. Future research should adopt multi-omics integration - miRNA+protein+metabolite) and machine-learning models to improve diagnostic specificity.

Conclusion. Exosomal biomarkers provide a promising, minimally invasive approach to understanding and differentiating mental disorders like MDD or BD. There is numerous evidence that exosomal biomarkers could become a diagnostic tool in the future. Future research should include standardized methods on large cohorts to develop new recommendations in diagnosing and treating both Major Depressive Disorder and Bipolar Disorder.

Keywords: exosomes, major depressive disorder, bipolar disorder, extracellular vesicles

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Neurophysiological Mechanisms of Trigeminal Neuralgia - From Demyelination Pathology to Central Sensitization

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Introduction. Trigeminal neuralgia (TN) is a severe neuropathic pain disorder characterized by brief, electric shock–like attacks within the trigeminal distribution. Although classically linked to neurovascular compression, recent evidence indicates that its pathophysiology extends beyond peripheral mechanisms to include central sensitization and neuroinflammatory processes.
Aim. The aim of this review was to analyze the neurophysiological mechanisms of TN, emphasizing the interplay between demyelination, altered ion channel expression, and central plasticity, as well as to present modern diagnostic and therapeutic approaches targeting these mechanisms.
Material and Methods. The study is a narrative review based on current, evidence-based literature concerning the peripheral and central mechanisms of TN, including electrophysiological, neuroimaging, and experimental model data.
Results. Peripheral demyelination at the root entry zone promotes ectopic discharges and ephaptic transmission between adjacent fibers, while upregulation of sodium channels (Nav1.6, Nav1.7) enhances neuronal hyperexcitability. Central sensitization involves activation of microglia and astrocytes releasing proinflammatory cytokines (IL-1β, TNF-α, IL-6) and neurotrophins (BDNF), contributing to persistent pain. Neuroimaging demonstrates cortical and subcortical reorganization in pain-processing regions. Therapeutic advances include selective sodium and calcium channel blockers, microvascular decompression, and neuromodulatory techniques such as rTMS and tDCS.
Conclusions. TN arises from an interplay between peripheral demyelination and central neuroplasticity. Understanding these interconnected mechanisms provides a foundation for more targeted and effective therapeutic strategies that address both neuronal hyperexcitability and neuroinflammation.
Keywords: trigeminal neuralgia, demyelination, ion channels, central sensitization, neuroinflammation, neuropathic pain.
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Excercise-induced Central fatigue: Mechanisms and Recovery

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In recent years, the topic of central fatigue and the recovery of the nervous system following physical exertion has been gaining increasing attention. Depending on the nature of the exercise, its intensity, and various external factors, different levels of fatigue may be induced. The aim of our presentation is to discuss the mechanisms underlying the development of central fatigue after physical activity, as well as the subsequent recovery processes within the nervous system. This narrative review is based on an analysis of publications from the last 10 years found in PubMed. The review included studies exploring the physiological and neurological aspects of exercise-induced central fatigue, as well as recovery strategies. The reviewed literature indicates that exercise-induced central fatigue is influenced by several factors, including neurotransmitter imbalance, metabolic and inflammatory responses, and impaired neuromuscular activation. Understanding the mechanisms of central fatigue and nervous system recovery is essential for optimizing training programs, preventing overtraining, and improving athletic performance.

Keywords: central fatigue, nervous system recovery, exercise physiology

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Exercise-Based Strategies to Enhance BDNF and Cognitive Recovery After Stroke: A Clinical review.

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Introduction. According to the latest World Stroke Organization Global Stroke Fact Sheet 2025, nearly 12 million new strokes and over 7 million stroke-related deaths occur globally each year, while around 94 million people live with post-stroke consequences. Stroke remains the second leading cause of death and the third leading cause of disability-adjusted life years (DALYs) worldwide. From a neurophysiological perspective, brain-derived neurotrophic factor (BDNF) is crucial for neuronal survival, synaptogenesis, and long-term potentiation—key mechanisms underlying neuroplasticity and cognitive recovery. Animal studies have shown that physical exercise enhances post-stroke recoveryby increasing BDNF expression and synaptic plasticity. However, clinical findings remain inconsistent, and the relationship between exercise-induced BDNF changes and cognitive improvement in humans is still unclear.

Aim. To compare and summarize clinical studies evaluating the effects of exercise interventions on BDNF levels and cognitive function in post-stroke patients.

Materials and Methods. Six clinical trials (n = 315) including post-stroke patients at various recovery stages (2 weeks–26 months) were analyzed. Aerobic interventions of different intensities (30–95 % HRR) lasting from 3 to 12 weeks were included. Studies assessed serum BDNF using ELISA and cognitive performance using standardized tests such as ACE-III, ACE-R, MoCA, RBANS, and TMT.

Results. Short interventions (≤ 3 weeks) led to a decrease in BDNF despite significant cognitive improvement ($r = -0.34$, $p = 0.04$). In contrast, longer programs (≥ 5 weeks) increased BDNF levels (by 25–80 %) alongside cognitive gains ($r = 0.59$, $p = 0.047$). The

most pronounced effects were observed with moderate-intensity exercise (50–60 % HRR), whereas high-intensity protocols (≥ 80 % HRR) enhanced BDNF but did not further improve cognition.
Conclusions. Moderate aerobic training sustained for 5–8 weeks (40–60 min sessions, 50–60 % HRR) appears most effective for stimulating BDNF and improving cognitive outcomes after stroke. Very short or excessively intense programs may increase BDNF without translating into cognitive benefits. Further
research should define optimal training parameters and explore combining physical exercise with neuromodulatory therapies (e.g., rTMS, tDCS) to enhance neuroplasticity and recovery.
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From Biomarker to Therapeutic Target: The multifaceted role of miR-124 in major depressive disorder
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Introduction. MicroRNA-124 (miR-124) is currently recognized as a key biomarker and potential therapeutic target in Major Depressive Disorder (MDD). Modulating its expression may offer a novel therapeutic approach, providing hope for more effective treatment and improved quality of life for individuals affected by MDD.
Aim. To present the potential role of miR-124 as a therapeutic target in depression.
Material and Methods. A literature review was conducted using PubMed, Scopus, and ResearchGate databases, covering publications from 2016 to 2025. The search terms included miR-124, MDD, biomarkers, and therapy. Studies investigating the impact of miR-124 on MDD were included, while those lacking quantitative and/or clinical data were excluded.
Results. Animal studies demonstrated that miR-124 expression was significantly decreased in the hippocampus of mice exposed to chronic unpredictable mild stress (CUMS; an animal model of depression). Restoration of miR-124 levels alleviated depressive-like behaviors and suppressed CUMS-induced microglial activation.
In a clinical study involving 91 patients, miR-124 expression was shown to modulate depressive symptoms via altered large-scale brain network connectivity, including the default mode (DMN), dorsal attention (DAN), salience (SN), and cingulo-opercular (CON) networks. miR-124 also appears to regulate appetite-related mechanisms in depression: miR-124-3p downregulates NPY expression, contributing to weight loss—particularly relevant for patients with symptoms linked to body image or somatic factors. Furthermore, miR-124 dysregulation has been associated with impaired neurotransmitter signaling and reduced neurotrophic support, notably affecting serotonin, dopamine, and brain-derived neurotrophic factor (BDNF). These changes may disrupt mood-regulating circuits, exacerbating the severity and persistence of depressive symptoms.

Conclusion. Although current findings highlight miR-124 as a promising target for MDD treatment, further research, standardized therapeutic protocols, and robust clinical validation are required to fully establish its clinical utility.
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Keywords: mir-124, MDD, biomarkers, therapy, gene expression regulation
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Clinical and Genetic Insights into Spinocerebellar Ataxia type 29 – a Case Report

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Background: Spinocerebellar ataxias (SCAs) denote a heterogeneous group of hereditary neurodegenerative disorders, most commonly presenting in adulthood. Nevertheless, few entities have their onset in childhood, including SCA29. It is associated with dominant pathogenic variants in the ITPR1 gene, which encodes a cation channel essential for Purkinje cell function. Clinically, SCA29 is characterized by early-onset cerebellar ataxia, global motor delay, and infantile muscular hypotonia.
Case report: The patient is a 5-year-old boy born at term after an uneventful pregnancy through cesarean section to healthy parents. From infancy, he exhibited hypotonia, delayed attainment of motor milestones, and reduced responsiveness to visual stimuli. Since the age of 4 years, he has been walking only with bilateral support. Independent ambulation has not been reached. The patient's cognitive development is normal, speech is adequate for the age, though dysarthric. Magnetic resonance imaging (MRI) revealed cerebellar hypoplasia, a significant feature of SCA29. Trio-based whole exome sequencing (WES) identified a pathogenic heterozygous missense variant in the ITPR1 gene c.722G>A p.(Arg241Lys). The variant was absent from both parents, confirming its de novo character. Combined clinical and molecular findings led to a diagnosis of SCA29.
Conclusions: SCA29 is an ultra-rare disorder, which clinical manifestation overlaps with various other neurological entities, posing difficulties in differential diagnosis. Genetic analysis should be considered in any child exhibiting signs of ataxia of unknown etiology. Early diagnosis is of particular importance, as timely implemented interprofessional rehabilitation may enhance the children's development, while allowing for proper genetic counseling and facilitating access to patient support groups.
Keywords: spinocerebellar ataxia, early-onset spinocerebellar ataxia
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