Visual Cueing in Parkinson's Disease: Neuroanatomy, Neurophysiology, Clinical Evidence, and Future Directions

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Abstract

Visual cueing — the use of external visual stimuli to guide movement — is one of the most effective and widely studied non-pharmacological interventions for the motor symptoms of Parkinson's disease (PD). This white paper reviews the historical development of visual cueing techniques, the neuroanatomical and neurophysiological mechanisms by which they exert their effects, the quantitative clinical evidence for improvements in gait, stride length, balance, fall rates, freezing of gait, and patient-reported outcomes including confidence and independence, and the current boundaries of knowledge. Key open questions and the most promising hypotheses for future investigation are identified, drawing on the perspectives of leading researchers and clinicians in the field.

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1. Introduction

Parkinson's disease is the second most common neurodegenerative disorder worldwide, affecting an estimated 10 million people globally and projected to reach 17 million by 2040.[1] Its hallmark motor features — resting tremor, bradykinesia, rigidity, and postural instability — arise primarily from the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc), leading to dopamine depletion in the striatum. Among the most disabling consequences of this neurodegeneration are gait disturbances: a characteristic shuffling walk with shortened stride length, reduced velocity, and impaired postural stability. In advanced disease, up to 63–90% of patients experience freezing of gait (FOG), a sudden, episodic inability to initiate or continue walking that is a leading cause of falls and loss of independence.[2]

Pharmacological treatment with levodopa and dopamine agonists provides substantial relief for many motor symptoms, but gait disturbances — particularly FOG — are often poorly responsive to medication, especially in later disease stages.[3] This therapeutic gap has driven decades of research into non-pharmacological strategies, of which **visual cueing** has emerged as one of the most clinically compelling. The observation that people with PD who are virtually unable to walk can step over lines on the floor, or follow a laser beam projected in front of their feet, has fascinated clinicians and neuroscientists alike and has opened a unique window into the neural architecture of voluntary movement.

2. Historical Background

The earliest systematic clinical observations of visual cueing in PD are attributed to the British neurologist **J.P. Martin**, who in his 1967 monograph *The Basal Ganglia and Posture* described patients with PD who could walk more normally when stepping over lines or cracks on the floor.[4] Martin recognized that the visual environment could substitute for something the diseased brain was failing to provide internally, though the neurological mechanism remained unexplained at the time.

Systematic experimental investigation began in earnest in the 1980s and 1990s. **Forssberg and colleagues** (1984) demonstrated that visual marking of the walking surface could improve gait in PD patients, establishing the basic paradigm of transverse floor lines as spatial cues.[5] **Bagley and colleagues** (1991) conducted one of the first controlled studies showing that visual cues on the floor significantly improved stride length and cadence in independently mobile PD patients.[6] The pivotal work of **Morris, Iansek, and colleagues** at La Trobe University in Melbourne through the 1990s provided a rigorous biomechanical framework: their 1994 and 1996 studies in *Brain* demonstrated that PD patients have a fundamental deficit in scaling stride length — they walk with a hypokinetic gait not because of weakness or spasticity but because the internal motor program fails to generate appropriately sized steps — and that visual cues could normalize this scaling deficit, increasing stride length by approximately 18% in controlled conditions.[7,8]

Azulay and colleagues (1999) in Marseille extended this work by distinguishing between static cues (lines on the floor) and dynamic optic flow cues, and by demonstrating that PD patients are more dependent on dynamic visual information for locomotor control than healthy adults, reflecting a compensatory reliance on visual feedback.[9] The 2000s brought the first large-scale randomized controlled trials. The **RESCUE (Rehabilitation in Parkinson's Disease: Strategies for Cueing) trial** (Nieuwboer et al., 2007), a multi-centre European study of 153 PD patients, provided the most robust evidence to date for the clinical effectiveness of a structured, home-based cueing programme.[10] More recently, research has shifted toward wearable and technology-enabled cueing devices, including laser shoes, smart glasses with augmented reality overlays, and sensor-triggered on-demand systems.

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3. Neuroanatomy and Neurophysiology

3.1 The Basal Ganglia and Dopamine Deficiency

To understand why visual cues work, it is necessary to understand what they are compensating for. The **basal ganglia** — comprising the striatum (caudate nucleus and putamen), globus pallidus (internal and external segments, GPi and GPe), subthalamic

nucleus (STN), and substantia nigra — form a critical subcortical loop that modulates voluntary movement. In the healthy brain, dopamine released from SNpc neurons into the striatum facilitates the **direct pathway** (striatum → GPi → thalamus → cortex), which promotes movement, while inhibiting the **indirect pathway** (striatum → GPe → STN → GPi → thalamus → cortex), which suppresses movement. The net effect is that dopamine enables smooth, appropriately scaled, internally generated movements.

In PD, dopamine depletion reverses this balance: the indirect pathway becomes overactive, the direct pathway is suppressed, and the GPi exerts excessive inhibition on the thalamus and motor cortex. The result is not simply slowness but a specific failure of **internal rhythm generation** — the basal ganglia can no longer provide the tonic drive that the **supplementary motor area (SMA)** requires to self-initiate and sustain rhythmic movements such as walking.[11] The SMA, located in the medial portion of Brodmann's area 6, is critically dependent on basal ganglia input for self-initiated, internally cued movements; when this input is disrupted, the SMA becomes hypoactive, and automatic motor programs — including the rhythmic stepping pattern of gait — deteriorate.[12]

Electrophysiological evidence has confirmed this model. In PD, the STN and other basal ganglia structures exhibit pathological **beta-band oscillations (10–30 Hz)** that are associated with motor suppression and correlate with the severity of bradykinesia and rigidity. Kühn and colleagues (2004) demonstrated that these oscillations decrease immediately after a visual go-cue is presented, with the onset of suppression correlating with reaction time — suggesting that visual cues directly modulate pathological basal ganglia activity.[13] Sarma and colleagues (2012) showed in intraoperative recordings from PD patients that predictably timed visual cues significantly decreased STN beta oscillations and increased directional tuning of STN neurons, facilitating motor output.[14]

3.2 The Alternative Pathway: Visual-Cerebellar-Premotor Circuit

The key to understanding visual cueing lies in an alternative motor pathway that remains relatively preserved in PD. The **lateral premotor cortex (PMC)**, unlike the SMA, receives its primary input not from the basal ganglia but from the **cerebellum** (via the ventrolateral thalamus) and from the **visual cortex** and **posterior parietal cortex**.[15] This cerebello-thalamo-cortical circuit is specialized for **externally guided movements** — movements triggered and regulated by sensory information from the environment rather than by internally generated timing signals.

When a person with PD sees a transverse line on the floor, the visual cortex processes this as a spatial target. The posterior parietal cortex integrates this visual information with proprioceptive and motor signals to compute the required step size. This information is relayed to the lateral PMC, which can directly activate the primary motor cortex (M1) and spinal motor circuits to generate a step — **bypassing the dysfunctional basal ganglia–SMA loop entirely**.[16] The cerebellum contributes by providing forward models of the expected sensory consequences of movement, enabling smooth, well-calibrated steps in response to the visual target.

Neuroimaging studies have confirmed this compensatory activation. Functional MRI and fNIRS studies consistently show that visual cueing increases activation in the **prefrontal**

cortex (PFC), **premotor cortex**, **primary somatosensory cortex (S1)**, and **visual association cortex (V2)** in PD patients with FOG, while also enhancing functional connectivity between these regions.[17] The cerebellum shows increased activity during cued versus uncued walking in PD, consistent with its role in externally guided movement and its compensatory hyperactivation in the context of basal ganglia dysfunction.[18] Importantly, the **primary motor cortex (M1)** itself is not the primary site of cue-related activation, suggesting that visual cues act primarily at the level of motor planning and preparation rather than execution per se.[19]

3.3 Attentional Mechanisms

A complementary mechanism involves **attention**. Walking, which is largely automatic in healthy individuals, requires explicit attentional resources in PD because the automatic motor programs are degraded. Visual cues may work in part by directing focused attention to the act of stepping — effectively converting an automatic motor task into a consciously controlled one. This "attentional strategy" hypothesis is supported by evidence that dual-task conditions (which divide attention) reduce the effectiveness of cueing, and that patients with greater cognitive impairment show attenuated cue responses.[20] The **prefrontal cortex**, which mediates executive function and attentional control, is consistently activated by visual cueing, and its activation correlates with gait improvement — suggesting that top-down attentional modulation is an integral part of the cueing mechanism.[17]

3.4 Eye Movements and Visuo-Cognitive Processing

Recent research using mobile eye-tracking has revealed that visual cueing also changes how people with PD explore their visual environment while walking. Graham and colleagues (2023) found that visual cues increased saccade frequency (the rate of rapid eye movements between fixation points) in both PD patients and healthy controls, but that the relationship between changes in visual exploration and gait improvement was specific to PD and differed between those with and without FOG.[21] This suggests that visual cueing engages **visuo-cognitive processing** — the integration of visual attention, spatial cognition, and motor planning — and that deficits in this processing may underlie the variable responses to cueing observed in clinical practice.

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4. Clinical Evidence: Quantitative Outcomes

4.1 Gait Speed and Stride Length

The most consistently reported benefits of visual cueing are improvements in **stride length** and **gait speed**. Morris and colleagues (1996) demonstrated an approximately 18% increase in stride length with visual floor cues in PD patients during the "off" medication state.[8] Suteerawattananon and colleagues (2004), in a controlled study of 39 PD patients, found that visual cues significantly improved stride length, while auditory cues primarily improved

cadence — establishing that the two modalities act through partially distinct mechanisms.[22] A meta-analysis by Lim and colleagues (2010) confirmed that visual cueing produced a significant improvement in stride length (Hedge's g = 0.554; 95% CI, 0.072–1.036), while auditory cueing produced broader improvements across multiple gait parameters.[23]

Tang and colleagues (2019), using full three-dimensional gait analysis with force platforms in 34 PD patients with FOG, found that laser-line cues significantly improved stride length (from 0.93 ± 0.20 m to 1.06 ± 0.18 m), gait velocity (from 0.87 ± 0.17 m/s to 1.01 ± 0.19 m/s), and cadence, bringing these parameters close to the values observed in healthy age-matched controls.[24] Kinematic improvements included increased ankle dorsiflexion, hip range of motion, and ankle push-off power — indicating that visual cues improve not just the temporal and spatial parameters of gait but its underlying biomechanical quality.

4.2 Freezing of Gait

Visual cues are considered the most effective non-pharmacological intervention specifically for FOG.[25] Velik and colleagues found that continuous visual cueing reduced the duration of freezing episodes by 51% and the number of episodes by 43%, while on-demand cueing (triggered by sensor detection of FOG) reduced freeze duration by 69% — though with a smaller reduction in episode frequency (9%), highlighting the complementary advantages of different cueing strategies.[26] Zhao and colleagues demonstrated that a Google Glass-based visual cueing application produced a statistically significant reduction in Freezing of Gait Questionnaire (FOGQ) scores and fall frequency in PD patients with FOG.[27]

4.3 Balance and Postural Stability

Zhang and colleagues (2023) used dynamic center-of-pressure (COP) analysis to show that wearable laser-line cues significantly improved postural stability during walking in PD patients, reducing the anterior-posterior displacement of the COP and normalizing the "distorted butterfly" COP trajectory characteristic of parkinsonian gait.[28] The RESCUE trial demonstrated significant improvements in timed balance tests ($p = 0.003$) following three weeks of cued gait training, alongside improvements in gait speed ($p = 0.005$) and step length ($p < 0.001$).[10] A 2024 scoping review by Giorgi and colleagues confirmed that cueing interventions consistently improve dynamic balance measures, though effects on static balance are less consistent.[29]

4.4 Falls, Confidence, and Independence

Falls are a leading cause of injury, hospitalization, and loss of independence in PD, occurring in approximately 60% of patients annually. While direct evidence for visual cueing reducing fall rates in long-term community settings remains limited, the RESCUE trial reported that patients receiving cued training showed greater confidence in carrying out daily activities — a finding of particular clinical significance given that fear of falling is itself a major contributor to reduced activity and social isolation in PD.[10] Donovan and colleagues, in an open-label study of 26 PD patients with FOG using laser-cane devices, found modest but meaningful reductions in freezing and falls in real-world settings.[30] Patient-reported outcomes consistently describe improved confidence in walking, reduced anxiety about

navigating challenging environments (doorways, turns, crowds), and greater independence in activities of daily living — outcomes that are not fully captured by laboratory gait measures but are central to quality of life.

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5. Limits of Current Knowledge

Despite substantial progress, several important questions remain unresolved.

Mechanism specificity. While the broad outlines of the neural mechanism are established — visual cues engage the premotor-cerebellar pathway and suppress pathological STN oscillations — the precise sequence of events from visual stimulus to improved gait remains incompletely understood. It is not clear, for example, whether the primary effect is on motor planning, motor execution, or sensorimotor feedback during the step itself, nor how the attentional and direct motor-pathway mechanisms interact.

Responder heterogeneity. Clinical responses to visual cueing vary considerably across patients. Some individuals show dramatic improvements; others show little or no benefit. The predictors of response are not well established. Disease stage, dopaminergic medication state, cognitive status (particularly executive function and visuospatial ability), and the specific neural substrate of FOG (dopaminergic-sensitive versus dopaminergic-resistant) all appear to modulate response, but no validated clinical algorithm exists for predicting who will benefit most from which type of cue.[25]

Cue parameters. The optimal characteristics of visual cues — spacing, color, contrast, static versus dynamic, continuous versus on-demand — have not been systematically determined. Evidence suggests that patient preference for cue parameters is associated with greater gait improvement, but the neurophysiological basis of this preference effect is unknown.[31]

Long-term effects and neuroplasticity. Most studies demonstrate acute, immediate benefits of visual cueing that diminish when the cue is removed. Whether sustained cueing training can induce lasting neuroplastic changes — reorganizing motor circuits to produce durable gait improvements independent of the cue — is a critical open question. The RESCUE trial found that improvements were not fully retained at three-month follow-up, suggesting limited long-term consolidation with current protocols.[10] De Icco and colleagues (2015) similarly found that chronic gait improvements from a four-week cueing rehabilitation programme were not retained at three-month follow-up in any cueing group.[32]

Real-world applicability. Laboratory studies are conducted under controlled conditions that do not reflect the complexity, variability, and cognitive demands of real-world walking. The effectiveness of cueing in community environments — navigating crowds, uneven surfaces, dual-task conditions — is less well established, and wearable cueing devices have shown more variable results outside the laboratory.[33]

6. Fruitful Hypotheses and Future Directions

Leading researchers and clinicians in the field have identified several priority areas for investigation.

Closed-loop, adaptive cueing systems. Rather than providing continuous or fixed-interval cues, next-generation systems would use wearable inertial sensors or electromyography to detect the early neural or kinematic signatures of an impending freeze and deliver a cue precisely when needed. This "on-demand" approach may be more effective and less cognitively fatiguing than continuous cueing, and could be personalized to each patient's freeze patterns.[25,33]

Augmented reality and immersive cueing. Smart glasses capable of projecting virtual transverse lines, 3D obstacles, or dynamic optic flow patterns into the patient's visual field offer the possibility of context-sensitive, spatially accurate cueing in any environment. Early studies of AR cueing are promising, and Cleveland Clinic researchers are currently investigating AR visual cues as a treatment for FOG, noting that visual cues are "the only promising means for improving freezing of gait in Parkinson's disease" in real-world settings.[34]

Combining cueing with neuroplasticity-based training. The hypothesis that cueing can serve as a scaffold for motor relearning — if paired with intensive, repetitive practice — has not been fully tested. If cueing allows patients to practice normal-amplitude gait, the resulting sensorimotor experience might drive cortical reorganization and reduce dependence on the cue over time. This approach would require protocols designed specifically to promote motor learning rather than simply compensate for the deficit.[35]

Cerebellar stimulation as an adjunct. Given the central role of the cerebellum in the cueing mechanism, non-invasive cerebellar stimulation (transcranial magnetic stimulation or transcranial direct current stimulation) may enhance the effectiveness of visual cueing by increasing the excitability of the cerebello-thalamo-cortical pathway. This is an emerging area of investigation with early supportive evidence.[18]

Biomarker-guided patient stratification. The identification of neuroimaging, electrophysiological, or genetic biomarkers that predict cueing response would allow clinicians to match patients to the most appropriate intervention. Wróbel and colleagues (2025) have shown that SMA microstructure, assessed by diffusion MRI, predicts gait impairment and potentially cueing response in PD — suggesting that structural neuroimaging may eventually guide therapeutic decisions.[36]

Multimodal cueing. Combining visual and auditory cues might be expected to produce additive benefits, but evidence is mixed: Zhang and colleagues (2026) found that combined rhythmic visual and auditory cues did not produce meaningful gait improvements beyond either modality alone, and actually decreased premotor cortex activation — suggesting

possible interference between modalities that needs to be better understood before multimodal approaches are widely adopted.[17]

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7. Conclusion

Visual cueing represents a remarkable example of how a simple, low-cost intervention can exploit preserved neural pathways to compensate for the consequences of neurodegeneration. By providing an external spatial target that engages the visual-premotor-cerebellar circuit, visual cues bypass the dysfunctional basal ganglia–SMA loop and restore more normal gait in people with Parkinson's disease. The clinical evidence is compelling: stride length increases of 18–25%, meaningful reductions in freezing episodes, improvements in balance and postural stability, and patient-reported gains in confidence and independence. Yet the field is far from mature. The variability of response, the limited durability of effects, and the challenges of real-world implementation all point to the need for more sophisticated, personalized, and neuroplasticity-informed approaches. The convergence of wearable sensor technology, augmented reality, closed-loop systems, and advanced neuroimaging offers a genuine opportunity to move visual cueing from a clinical observation to a precision therapeutic tool — one that could substantially improve the daily lives of millions of people living with Parkinson's disease.

References

[1] Dorsey ER, Bloem BR. The Parkinson pandemic — a call to action. *JAMA Neurology*. 2018;75(1):9–10.

[2] Nutt JG, Bloem BR, Giladi N, Hallett M, Horak FB, Nieuwboer A. Freezing of gait: moving forward on a mysterious clinical phenomenon. *Lancet Neurology*. 2011;10(8):734–744.

[3] Keus SH, Bloem BR, Hendriks EJ, Bredero-Cohen AB, Munneke M. Evidence-based analysis of physical therapy in Parkinson's disease with recommendations for practice and research. *Movement Disorders*. 2007;22(4):451–460.

[4] Martin JP. *The Basal Ganglia and Posture*. London: Pitman Medical Publishing; 1967.

[5] Forssberg H, Johnels B, Steg G. Is parkinsonian gait caused by a regression to an immature walking pattern? *Advances in Neurology*. 1984;40:375–379.

[6] Bagley S, Kelly B, Tunnicliffe N, Turnbull GI, Walker JM. The effect of visual cues on the gait of independently mobile Parkinson's disease patients. *Physiotherapy*. 1991;77(6):415–420.

[7] Morris ME, Iansek R, Matyas TA, Summers JJ. The pathogenesis of gait hypokinesia in Parkinson's disease. *Brain*. 1994;117(5):1169–1181.

[8] Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease: normalization strategies and underlying mechanisms. *Brain*. 1996;119(2):551–568.

[9] Azulay JP, Mesure S, Amblard B, Blin O, Sangla I, Pouget J. Visual control of locomotion in Parkinson's disease. *Brain*. 1999;122(1):111–120.

[10] Nieuwboer A, Kwakkel G, Rochester L, et al. Cueing training in the home improves gait-related mobility in Parkinson's disease: the RESCUE trial. *Journal of Neurology, Neurosurgery & Psychiatry*. 2007;78(2):134–140.

[11] Georgiou N, Iansek R, Bradshaw JL, Phillips JG, Mattingley JB, Bradshaw JA. An evaluation of the role of internal cues in the pathogenesis of parkinsonian hypokinesia. *Brain*. 1993;116(6):1575–1587.

[12] Rahimpour S, Rajkumar S, Hallett M. The supplementary motor complex in Parkinson's disease. *Journal of Movement Disorders*. 2022;15(1):21–32.

[13] Kühn AA, Williams D, Kupsch A, et al. Event-related beta desynchronization in human subthalamic nucleus correlates with motor performance. *Brain*. 2004;127(4):735–746.

[14] Sarma SV, Cheng ML, Eden U, Williams Z, Brown EN, Eskandar E. The effects of cues on neurons in the basal ganglia in Parkinson's disease. *Frontiers in Integrative Neuroscience*. 2012;6:40.

[15] Lewis MM, Galley S, Johnson S, Stevenson J, Huang X, McKeown MJ. The role of the cerebellum in the pathophysiology of Parkinson's disease. *Canadian Journal of Neurological Sciences*. 2013;40(3):299–306.

[16] Glickstein M, Stein J. Paradoxical movement in Parkinson's disease. *Trends in Neurosciences*. 1991;14(11):480–482.

[17] Zhang Z, Sheng L, Cui G, Shan C, Xiang J. The mechanism of external cueing interventions in improving freezing of gait in Parkinson's disease: an fNIRS study. *Frontiers in Aging Neuroscience*. 2026;18:1776882.

[18] Lewis MM, Galley S, Johnson S, Stevenson J, Huang X, McKeown MJ. The role of the cerebellum in the pathophysiology of Parkinson's disease. *Canadian Journal of Neurological Sciences*. 2013;40(3):299–306.

[19] Zadeh AK, et al. Connecting the dots: sensory cueing enhances functional connectivity but not primary motor cortex activation. *European Journal of Neuroscience*. 2024;60:1437.

[20] Azulay JP, Mesure S, Blin O. Influence of visual cues on gait in Parkinson's disease: contribution to attention or sensory dependence? *Journal of the Neurological Sciences*. 2006;248(1–2):192–195.

[21] Graham L, Armitage J, Vitorio R, et al. Visual exploration while walking with and without visual cues in Parkinson's disease: freezer versus non-freezer. *Neurorehabilitation and Neural Repair*. 2023;37(10):734–743.

[22] Suteerawattananon M, Morris GS, Etnyre BR, Jankovic J, Protas EJ. Effects of visual and auditory cues on gait in individuals with Parkinson's disease. *Journal of the Neurological Sciences*. 2004;219(1–2):63–69.

[23] Lim I, van Wegen E, de Goede C, et al. Cueing and gait improvement among people with Parkinson's disease: a meta-analysis. *Archives of Physical Medicine and Rehabilitation*. 2010;91(11):1832–1836. [Note: Hedge's g = 0.554 for visual cueing on stride length, 95% CI 0.072–1.036.]

[24] Tang L, Xu W, Li Z, et al. Quantitative gait analysis for laser cue in Parkinson's disease patients with freezing of gait. *Annals of Translational Medicine*. 2019;7(14):325.

[25] Ginis P, Nackaerts E, Nieuwboer A, Heremans E. Cueing for people with Parkinson's disease with freezing of gait: a narrative review of the state-of-the-art and novel perspectives. *Annals of Physical and Rehabilitation Medicine*. 2018;61(6):407–413.

[26] Velik R, Hoffmann U, Zabaleta H, Marti Masso JF, Keller T. The effect of visual cues on the number and duration of freezing episodes in Parkinson's patients. *Proceedings of the IEEE Engineering in Medicine and Biology Society*. 2012:4656–4659.

[27] Zhao Y, Nonnekes J, Storcken EJ, et al. Feasibility of external rhythmic cueing with the Google Glass for improving gait in people with Parkinson's disease. *Journal of Neurology*. 2016;263(6):1156–1165.

[28] Zhang W, Han Y, Shi Y, et al. Effects of wearable visual cueing on gait pattern and stability in patients with Parkinson's disease. *Frontiers in Neurology*. 2023;14:1077871.

[29] Giorgi F, Donati D, Tedeschi R. Cueing interventions for gait and balance in Parkinson's disease: a scoping review of current evidence. *Applied Sciences*. 2024;14(24):11781.

[30] Donovan S, Lim C, Diaz N, et al. Laserlight cues for gait freezing in Parkinson's disease: an open-label study. *Parkinsonism & Related Disorders*. 2011;17(4):240–245.

[31] Nishikawa R, Nishizawa H, Fukushima K, et al. The effects of visual cues from optical stimulation devices on gait disturbance in patients with Parkinson's disease. *Journal of Physical Therapy Science*. 2023;35(3):230–236.

[32] De Icco R, Tassorelli C, Berra E, et al. Acute and chronic effect of acoustic and visual cues on gait training in Parkinson's disease: a randomized, controlled study. *Parkinson's Disease*. 2015;2015:978590.

[33] Sweeney D, Quinlan LR, Browne P, Richardson M, Meskell P, ÓLaighin G. A technological review of wearable cueing devices addressing freezing of gait in Parkinson's disease. *Sensors*. 2019;19(6):1277.

[34] Cleveland Clinic. Studying augmented reality visual cues to treat freezing of gait in Parkinson's. *Consult QD*. May 10, 2023. https://consultqd.clevelandclinic.org/studying-augmented-reality-visual-cues-to-treat-freezing-of-gait-in-parkinsons

[35] Nieuwboer A. Cueing for freezing of gait in patients with Parkinson's disease: a rehabilitation perspective. *Movement Disorders*. 2008;23(Suppl 2):S475–S481.

[36] Wróbel PP, et al. Supplementary motor area microstructure defines the response to cueing in Parkinson's disease. *npj Parkinson's Disease*. 2025;11:119.