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A New Therapeutic Approach to Improving Movement Disorders

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ABSTRACT

Objective: This study aims to propose a novel multimodal therapeutic framework for movement disorders that extends beyond symptom control toward functional reorganization of motor networks, enabling more stable and meaningful long-term recovery.

Methods: A network-based conceptual model was developed consisting of three integrated components: pharmacological optimization within network physiology, non-invasive and invasive neuromodulation strategies, and task-specific motor retraining at optimized therapeutic doses to promote adaptive circuit reorganization. The model is grounded in systems neuroscience and functional imaging evidence involving basal ganglia, thalamocortical, and cerebellar networks.

Results: The proposed approach targets not only symptom reduction but also normalization of abnormal connectivity, oscillatory activity, and maladaptive plasticity within motor networks. Although direct clinical validation is still required, theoretical and experimental evidence supports the potential of combining pharmacological, neuromodulatory, and motor training interventions to enhance long-term functional outcomes.

Conclusions: A multimodal, network-based therapeutic strategy may offer advantages over conventional approaches by facilitating adaptive motor network reorganization in addition to symptom relief. This integrated approach may reduce variability in treatment response and improve long-term functional recovery.

Keywords: Movement disorders, Basal ganglia, Neuromodulation, Functional reorganization, Pharmacological optimization

INTRODUCTION

Movement disorders have significant clinical and social impacts, including Parkinson's disease (PD), essential tremor, dystonia, chorea, myoclonus, and tics [1, 2]. While the phenotypes of these disorders differ, closer examination has revealed shared mechanistic underpinnings including basal ganglia-thalamocortical and cerebellothalamocortical circuits [3, 4]. Early neuroscientific models tended to conceptualize each disorder as end-stage pathology localized to a specific nucleus, particularly loss of dopaminergic neurons characteristic for PD [5]. This framework was instrumental in the development of pharmacological agents, but today it is understood that this viewpoint represents only part of the underlying mechanism[6].

In parallel, the field has increasingly focused on movement disorders as network disturbances rather than isolated lesions, supported by advances in functional neuroimaging, electrophysiology and computational modeling [7-11]. Though therapeutic progress has been significant, treatment continues to be a challenge. It is well established that many patients develop an incomplete response to standard pharmacological treatment with time or suffer from medication-related motor complications such as drug-induced dyskinesias and gait problems [11-13]. DBS has significantly improved treatment outcomes for some patients, but significant limitations remain [14, 15]. These difficulties have encouraged the search for treatments that extend beyond symptomatic control [16]. The overarching goal of this article is to describe and discuss a nascent network-based therapeutic framework, the network-based approach, which conceptually focuses on orchestrating: (1) the balance of neurochemical systems; (2) neuromodulation with targeted control over dysfunctional circuits; and (3) adaptive motor learning by means of structured, task-specific training. Each component has demonstrated efficacy

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in isolation, yet it remains unclear how best to integrate these approaches into a unified intervention. In this paper, we present a conceptual design to integrate these aspects and speculate on its possible implications for long-term functional recovery.

EPIDEMIOLOGY

The worldwide prevalence of movement disorders is increasing as a result of demographics and enhanced recognition [17]. For example, PD is one of the fastest growing neurological diseases worldwide [18]. Recent epidemiological studies indicate that the prevalence rate is almost doubled with every 10 years over age of 60, and it belongs to one of the most frequent aging-related neurodegenerative diseases [19]. The increase is particularly marked in countries with rising life expectancy, due to the increasing burden on health systems to provide long term care and rehabilitation [20].

ET, although often undervalued when compared to PD, is much more prevalent. Community studies from several areas calculate its prevalence between 0.4% and 1% in the general population increasing with age, specifically over the age of 65 [21, 22]. Adjacent to the fact that essential tremor used to be considered a ‘’benign’’ condition it has become increasingly clear that ETS can lead to a marked impairment of daily life especially when severity and spread of tremor increases [23].

Hyperkinetic movement disorders like dystonia, chorea, and myoclonus are less common but can also have significant impact on the patient's life [24]. Some young people with genetic dystonias present in adolescence or early adult life, causing disturbance to educational and occupational trajectories [25]. Chorea, whether genetic (e.g., as in Huntington’s disease) or acquired, places a significant burden on care givers and is frequently associated with psychiatric or cognitive features that can make long-term treatment challenging [26].

Increasingly, however, the medical costs of hospitalization, medication and surgery are being directly associated with movement disorders, but also indirect costs attributable to falls and fractures increased rates of institutionalization occupational disablement and psychosocial burden on families [27]. This wider scope increases the demand for alternatives in treatment focusing on long-term effect indicators rather than on mere symptomatic relief [28].

ETIOLOGY

The etiology of movement disorders is complex and a combination of genetic susceptibility, environmental exposure, endogenous metabolic effects, and network-level perturbations [29].

Genetic Contributions

Classic monogenic disorders, such as Huntington’s disease (HTT gene expansion) and Wilson’s disease (ATP7B mutations), demonstrate single-gene defects that lead to defined movement disorder phenotypes. However, many other disorders such as PD and different dystonia’s are a result of complex polygenic factors. Extensive genetic analyses have associated a large number of loci with synaptic transmission, mitochondrial robustness and protein degradation pathways indicating that the architecture is multifactorial rather than a single dominant mechanism [30].

Environmental and Acquired Factors

Exposures in the environment have long been associated with parkinsonism and other movement disorders. Exposure to pesticides, industrial solvents, and heavy metals are commonly identified risk factors especially in populations with long-term occupation exposure. Cumulative head injury, including mild traumatic brain injury, has also been linked to delayed-onset parkinsonism and has been reported in some athletic and military groups [31].

Another significant etiologic category is medications. Dopamine receptor blockade by many antipsychotic agents used in psychiatry can cause drug-induced parkinsonism, tardive dyskinesia or mixed hyperkinetic syndromes [32]. These entities frequently share degenerative patterns, which makes both diagnosis and treatment difficult.

Network, Inflammatory, and Metabolic Influences

According to Table 1, emerging evidence from electrophysiological and neuroimaging studies indicates that movement disorders are not solely caused by focal structural lesions [33]:

Table 1. Neural Network and Metabolic Contributions to Movement Disorders.

Finding / Mechanism	Details
Altered functional connectivity	Within basal ganglia–thalamocortical networks, affecting movement regulation.
Pathological cerebellar–ganglia communication	Particularly relevant in tremor and dystonia.
Neuroinflammation and oxidative stress	Subtle changes that exacerbate network dysfunction and contribute to variability in clinical presentation.
Interdependent network–metabolic action	The interaction between distributed neural networks and metabolic liability explains diverse patient phenotypes and therapeutic responsiveness.

PATHOPHYSIOLOGY

An in-depth understanding of the path mechanism is crucial for developing therapeutic strategies that extend beyond mere symptom relief.

Basal Ganglia and Parallel Motor Circuits

The basal ganglia function within a series of parallel-linked circuits which convey information among the cortex, thalamus and brainstem. The simple direct–indirect pathway model appears to be more valid than one might think. In PD, depletion of dopaminergic input biases activity opposed to the effective broadcast within thalamocortical circuits onto direct pathway cells in the SNr, thereby reducing output from SNr that inhibits thalamus (thalamocortical facilitation) which generate bradykinesia and rigidity. On the other hand, hyperkinetic disorders frequently appear as a result of decreased inhibitory output or abnormal firing pattern from basal ganglia output nuclei, which produce excessive or unwanted movements [34].

Network Oscillations and Synchrony

Recently, similar emphasis has been put on oscillatory activity and large-scale synchronization [35]:

- Abnormal beta-band oscillations (13–30 Hz) in PD are associated with rigidity and akinesia;
- Disorders with tremor are characterized by atypical frequency behaviour coupled among the cerebellum, thalamus and cortex.

These oscillatory signatures are now more and more considered as biomarkers that can help to target therapeutic interventions.

Cerebellar Involvement

The role of the cerebellum as a coordinator is no longer its sole function, and it has been implicated in tremor and dystonia. In essential tremor and tremor-dominant PD, imaging studies demonstrate cerebellar hyperactivity and abnormal patterns of connectivity. In dystonia, the concept of cerebellar involvement has emerged to challenge this older basal ganglia centric model and identify alternative targets for neuromodulation [36].

Maladaptive Plasticity

Persistently altered synaptic plasticity, such as exaggerated long-term potentiation (LTP) or diminished long-term depression (LTD), may lock in suboptimal motor programs. Therapeutic interventions that focus on activity-dependent plasticity for remodeling maladaptive networks could be essential to reset these patterns.

CLINICAL FEATURES

Clinical presentations of movement disorders show extensive variation, among patients and also within diagnostic categories.

Hypokinetic Manifestations

The first, and often most disabling feature of Parkinson's disease (PD)and related diseases is bradykinesia or slowness of movement. Patients frequently complain about everyday activities such as buttoning a shirt, standing up from a chair or beginning to walk being much slower than in the past. Stiffness can cause pain and reduced arm swing, as well as a sense of resistance that comes and goes during the day. Reduced vocal intensity and facial hypomimia are common, and may lead patients' emotional states to be misread [38].

Hyperkinetic Manifestations

The heterogeneous cluster of hyperkinetic symptoms is displayed in **Table 2** [39]:

Table 2. Hyperkinetic Manifestations and Clinical Impact.

Symptom	Description / Characteristics	Clinical Impact
Tremor	Can manifest at rest (e.g., Parkinson's disease), during posture, or during action (e.g., essential tremor).	Interferes with daily activities such as eating, writing, and fine motor tasks.
Dystonia	Sustained muscle contractions resulting in abnormal postures or repeated movements.	May cause disability; e.g., cervical dystonia can lead to sustained head tilt affecting work and driving.
Chorea	Rapid, random, irregular, purposeless, involuntary movements that flow unpredictably from one body part to another.	Can disrupt coordinated movement and functional tasks.
Myoclonus	Abrupt, shock-like jerks, which can be focal or generalized.	Can interfere with voluntary movements and daily functioning.

Gait, Balance, and Postural Instability

Parkinsonian gait and balance are more frequently affected. Freezing of gait, or the temporary inability to take a step when attempting to walk, can result in falls and significant loss of independence." This tight association between cognitive and motor pathways is reflected in the dual-task interference, i.e., when gait declines during a concurrent cognitive performance [40].

Non-Motor Features

While traditionally described as movement disorders, some conditions such as PD are characterized by a range of non-motor symptoms [41]:

- Cognitive slowing and/or impairment of the executive functions
- Feeling depressed, anxious, or apathetic
- Sleep disturbances
- Disorder of the autonomic nervous system (e.g., orthostatic hypotension, constipation)

Such symptoms can even occur many years before motor signs, and emphasize that motion disorders are manifestations of disordered neural activity rather than simplistic motor defects.

DIAGNOSIS AND IMAGING

Diagnosis is made by integrating clinical examination and selective investigations and excluding other mimics.

Clinical Examination and Standardized Scales

A neurologists er round evaluation are a crucial part of the diagnostic approach. Clinicians systematically evaluate [42]:

- Time of appearance, changes in and spread of symptoms;
- Family history;
- Previous or current exposure to dopaminergic or antidopaminergic (including dopamine antagonists) agents;
- Fluctuating symptoms.

A number of standardized scoring instruments aid in diagnostic accuracy and follow-up. The Movement Disorder Society–Unified Parkinson's Disease Rating Scale (MDS-UPDRS) is frequently used to assess bradykinesia, rigidity, tremor, gait impairment and non-motor symptoms in PD. Invalid tremor (Fahn–Tolosa–Marín Tremor Rating Scale) and dystonia (Burke–Fahn–Marsden Dystonia Rating Scale) scales are also available [43].

Laboratory and Genetic Tests

Laboratory tests are useful when the etiology may be autoimmune, viral or metabolic. Although polygenic diseases, such as Parkinson's disease (PD), remain mainly diagnosed clinically, genetic testing is of particular value in early onset or familial cases [44].

Imaging and Neurophysiology

As seen in Figure 1, magnetic resonance imaging (MRI) is the foundation of the inquiry to rule out structural etiologies such malignancies, vascular lesions, or demyelinating illness. Dopaminergic transporter single-photon emission computed tomography (DAT-SPECT) and 18F-fluorodopa positron emission tomography (FDOPA-PET) help differentiate neurodegenerative parkinsonism from drug-induced or functional forms. Functional MRI has further substantiated disease-specific alterations of brain connectivity in PD, tremor and dystonia. In rare or ambiguous cases, tremor frequency and patterns may be characterized with accelerometry and electromyography (EMG). Network-based imaging methods are currently still restricted primarily to research paradigms, but may rise in relevance for clinical applications such as for example guiding and rationalizing neuromodulation [45].

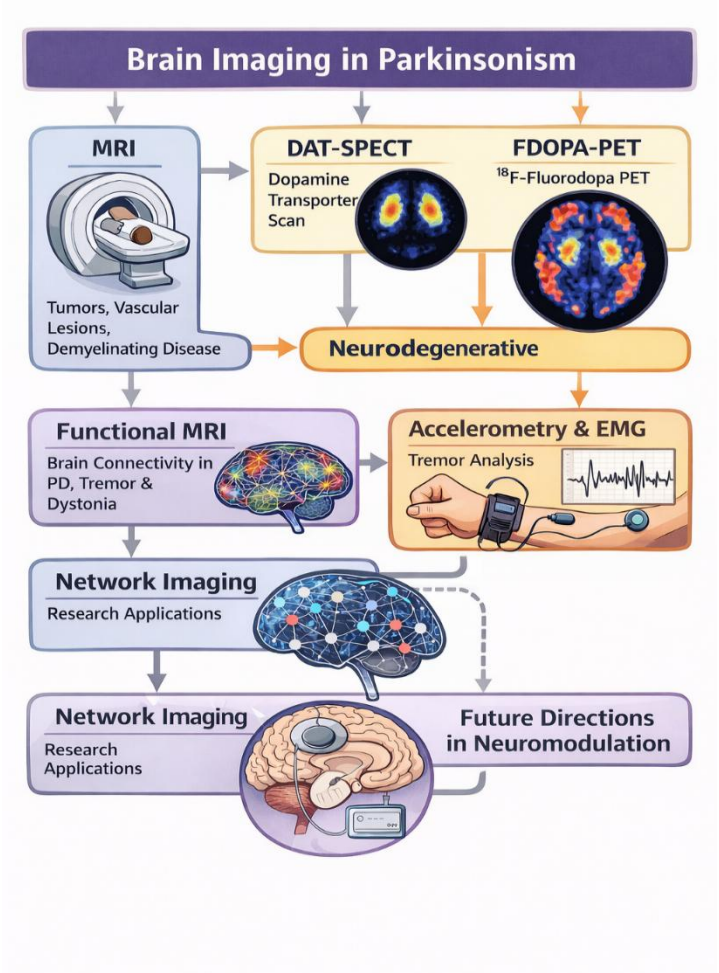

Figure 1: Overview of neuroimaging and electrophysiological techniques used in the diagnosis of movement disorders. Structural imaging (MRI) is used to exclude anatomical lesions, while functional techniques such as DAT-SPECT and FDOPA-PET assess dopaminergic deficits. Functional MRI and electrophysiological tools (EMG, accelerometry) provide insights into abnormal network connectivity and oscillatory activity.

CURRENT THERAPEUTIC APPROACHES

Current treatment modalities are derived from years of clinical practice yet frequently fail at providing lasting functional recovery.

Pharmacological Therapies

Dopaminergic drugs continue to be the mainstay of therapy for PD. Levodopa remains the most effective for symptomatic treatment, but long-term complications are also known to exist like [46].

- Motor fluctuations (on-off periods)
- LID (Levodopa-induced dyskinesia)
- Limited benefit in axial symptoms (gait, balance, and posture)

Appetitive: Augmenting agents such as the dopamine agonists, MAO-B inhibitors and COMT inhibitors may also have additional benefits; however, they could increase the risk of side effects (e.g. hallucinations, somnolence, or impulse control disorders).

The symptomatic relief of anxiolytics or anticholinergics, beta-blockers and benzodiazepines for example in tremor and dystonia respectively, often does little to influence the underlying network disturbances. Botulinum toxin in focal dystonia and tremor syndromes At Forty years, from its initial use in 1974 [47], botulinum is one of the main first lines therapies in these diseases.

Deep Brain Stimulation (DBS)

Deep brain stimulation (DBS) has revolutionized the management of advanced Parkinson's disease, essential tremor and some forms of dystonia. Subthalamic nucleus (STN), GPi, or VIM has been found to substantially reduce motor symptoms and improve quality of life and reduces medication needs. However, there are several limitations [48]:

- DBS does not restore motor function to normal levels.
- Speech and postural instability, freezing of gait frequently remain.
- Setting up stimulation protocol can be complicated and time consuming.
- Existing DBS is open-loop and does not adjust itself to physiological variations that may fluctuate from minute-to-minute.

Non-Invasive Brain Stimulation (NIBS)

Approaches like rTMS and tDCS are being considered as supplementary treatment options. The findings point to moderate but clinically relevant improvements in [49]:

- Gait performance
- Bradykinesia
- Mood and cognitive dimensions.

Despite the disparate adoption of this treatment, active research is further defining stimulation targets and parameters.

Rehabilitation and Task-Specific Training

Physical activity and physiotherapy are well-established in the treatment but often utilized insufficiently. Emerging evidence indicates that [50]:

- High-intensity balance training enhances postural control and decreases fall risk.
- Dual-task exercise enhances gait stability and cognitive set-shifting.
- Task-targeted training produces cognitive and QoL benefits that can be quantified.

Despite this weight of evidence, rehabilitation is often delivered at a dose that may not be high enough to contribute to persistent neuroplastic change. In addition, the cocktail of tDCS, medications and motor training is little harmonized.

MECHANISTIC RATIONALE FOR A NEW THERAPEUTIC APPROACH

While legitimate progress has been made in the area of medicine, it is still limited by structural inefficiencies. Invasive or not, neuromodulation often complements more than compounds rehabilitation [51].

Limitations of Current Care

As seen in **Table 3**, three limitations jump out.

Table 3. Limitations of Current Care in Movement Disorder Management [52].

Limitation	Description / Details
Over-reliance on dopaminergic correction	Pharmacological treatments alleviate symptoms but often fail to restore atypical rhythmic activity patterns (e.g., pathological beta synchronization in PD) that are critical to motor function.
Discontinuous application of neuromodulation and rehabilitation	DBS programming, physiotherapy, and medication adjustments are typically performed separately in clinical settings, preventing alignment of interventions with periods of elevated plasticity or network responsiveness.
Underutilization of network-based biomarkers	Investigation of beta band activity, connectivity patterns, and task-related oscillatory changes highlights explanatory gaps that are not yet regularly applied in clinical decision-making.

Emerging Evidence and Scientific Basis

Recent studies indicate that:

- Specific frequency bands can be modulated through DBS, adaptive DBS or rTMS to change network dynamics and enhance motor function.
- The cerebellum can act as a potent gateway to influence abnormal synchrony in tremor and dystonia via its interactions with the basal ganglia and motor cortex.

Together, these results support a new approach for the treatment of SSLP which intentionally combines chemical, electrical and behavioral interventions to promote adaptive reorganization of motor networks [53].

PROPOSED NEW THERAPEUTIC APPROACH

Our proposed model is based on the three following aspects. Different MoA are provided by each of the pillars, but their real benefit is found in their complementary functioning [54].

1. Network-Based Pharmacological Optimization [55]

Key Components

- Adaptive dopaminergic therapy to decrease pathological beta synchrony and increase modulation responsiveness.
- Addition of non-dopaminergic drugs (i.e., NMDA modulators, GABAergic enhancers, cholinergic stabilizers) as needed with the understanding that numerous neurotransmitter systems are involved in circuit pathology.
- Use of objective measures such as motor diaries, wearable sensors, and patient-reported feedback to define performance levels for instances in which patients are most "motor available" for therapy.

Functional Purpose

- This sets up circuits to be more sensitive to subsequent neuromodulation, and to secure motor learning better.

2. Network-Level Neuromodulation

This pillar integrates non-invasive and minimally invasive techniques into a cohesive procedure (refer to **Table 4**).

Table 4. Network-Level Neuromodulation Approaches in Movement Disorders.

Category	Approach / Strategy	Details
Invasive Neuromodulation	DBS and Adaptive DBS	For suitable patients, DBS remains highly effective. Adaptive DBS adjusts in real-time based on biomarkers (e.g., beta activity), relieving motor symptoms [56].
Non-Invasive Brain Stimulation	rTMS	Applied to primary motor cortex, supplementary motor area, or cerebellum depending on the clinical phenotype.
	tDCS	Provides milder, cumulative neuromodulation effects by increasing motor excitability or suppressing maladaptive activity patterns [57].
Timing & Personalization	Coupling with Intensive Training	Neuromodulation sessions are administered alongside intensive motor training, leveraging periods of heightened plasticity to optimize motor learning and retention [58].

3. Intensive Task-Oriented Motor Retraining

According to **Table 5**, this pillar transforms rehabilitation from an adjuvant to a primary treatment approach [59].

Table 5. Principles and Rationale of Functionally Targeted Neurorehabilitation.

Category	Principle / Rationale	Details
Principles	Task Specificity	Practice is specific to functionally meaningful activities such as gait, turning, balance, and hand use.
	High Repetition / Dose	Intensity is needed to stimulate permanent reorganization.
	Progression Complexity	Activities introduce variation and dual-task demands as skills develop.
	Digital Tool Integration	Wearable sensors, VR-enriched practice, and home-based platforms extend training outside a clinic setting.
Rationale	Reinforcing Favorable Patterns	When neuromodulation diminishes pathological synchronization, specialized training can reinforce favorable patterns and suppress unfavorable ones.

Safety and Clinical Implementation

Safety Considerations

- Pharmacotherapies: Monitoring of hypotension, psychiatric symptoms, dyskinesias and sleep disorders are needed [60].
- DBS: It is associated with the risk of infection, hardware problems and rare intracranial hemorrhage; long-term follow-up is required.
- rTMS/tDCS – Relatively safe when procedures are carried out as per established protocols, with few and mild side effects which are transient.
- High-intensity training: Might lead to fatigue or risk of falling, particularly in frail subjects; monitoring and regular re-evaluation are required.

Clinical Delivery Model

Optimal delivery of the described protocol is likely to occur in tertiary centres with multidisciplinary input, such as:

- Neurologists, neurosurgeons and physiatrists
- Physical and occupational therapists
- Neuropsychologists and speech-language pathologists
- Nursing staff specifically trained for DBS programming and NIBS delivery

However, dedicated centres can facilitate the optimization and synchronization of medication timing, neuromodulation windows and structured training blocks as part of a single treatment pathway [61].

Practical Implementation Phases

Phased rehabilitation is described (**Figure 2**): evaluation, optimization, relearning with the biological readout and neuromodulation, and long-term stability with continuous training and adjustment of parameters.

1. **Assessment Phase**
   - Comprehensive clinical evaluation
   - Imaging and electrophysiological biomarkers
   - Identification of oscillatory targets
2. **Optimization Phase**
   - Medication calibration
   - DBS programming or rTMS/tDCS protocol determination
3. **Relearning Phase**
   - High-intensity, task-oriented training integrated with neuromodulation sessions
   - Real-time feedback from wearable sensors
4. **Maintenance Phase**
   - Long-term training cycles
   - Periodic re-optimization of stimulation parameters and pharmacotherapy

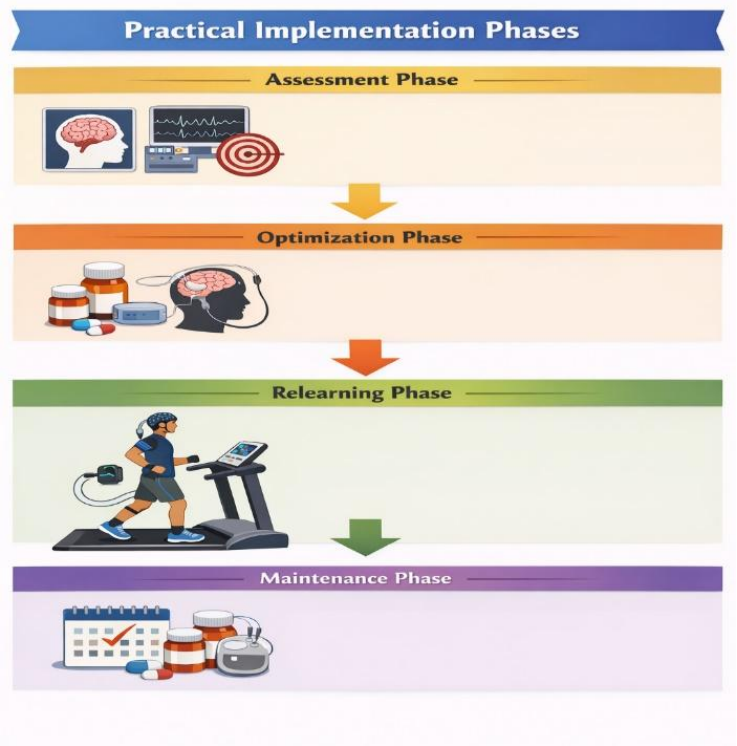

Figure 2: Schematic representation of the proposed phased therapeutic approach. The model includes four stages: assessment, optimization, relearning, and maintenance. Each phase integrates pharmacological adjustment, neuromodulation, and task-specific motor training to promote functional reorganization of motor networks.

Literature Survey

New therapeutic approaches for movement disorders: a literature review focusing on cognition physiology. Novel therapies such as DBS, rTMS and tDCS with and without high intensity feedback-driven training have demonstrated promising results on enhancing motor function and quality of life. The most important studies, summarized in **Table 6**, show a comparison between methods applied and patient populations and clinical outcomes achieved in recent research.

Table 6: Literature Survey on New Therapeutic Approaches to Movement Disorders.

Authors & Year	Key Focus & Findings	Methodology/Approach	Relevance to Proposed Model
Ferrazzoli, D. et al. (2022) [62]	Reviews how tailored rehabilitation strategies and goal-based aerobic and motor-cognitive training drive neuroplasticity and improve movement disorder outcomes by restoring circuit dysfunctions.	Systematic/narrative review of rehabilitation evidence	Supports task-specific, high-intensity motor training to modulate plasticity and networks. (PubMed)
Martinez-Nunez, A. E. et al. (2024) [63]	Summarizes new neuromodulation interventions (e.g., adaptive stimulation, targeted network modulation) for PD, emphasizing network-specific strategies.	Narrative review of neuromodulation advancements	Relates to network-level neuromodulation and optimization beyond traditional DBS. (ScienceDirect)
Xia, Y. et al. (2023) [64]	Cerebellar rTMS shows promise for improving motor dysfunction in PD, ET, dystonia, and ataxia with minimal safety concerns.	RCT systematic review	Highlights non-invasive neuromodulation targeting cerebellar networks. (PubMed)

Lefaucheur, J. P. et al. (2024) [65]	Comprehensive review of neuromodulation methods (DBS, rTMS, tDCS, tACS, FUS) and mechanisms, including emerging therapeutic protocols.	Handbook chapter summarizing clinical and research data	Validates the integration of invasive & non-invasive neuromodulation in therapeutic strategies. (ScienceDirect)
Desai, D., & Desai, S. (2025) [66]	Focuses on circuit-level dysfunction in functional movement disorders and the clinical potential of neuromodulation integrated with multidisciplinary care.	Narrative review	Shows value of network-based and personalized neuromodulation (including TMS, tDCS). (PubMed)
Wang, J. et al. (2023) [67]	Illustrates expanding understanding of circuits beyond classical basal ganglia, including novel targets for stimulation.	Review article	Supports network-based physiological understanding guiding therapeutic targets. (ScienceDirect)

LIMITATIONS

The proposed model is primarily conceptual and lacks direct clinical validation. Although it is supported by existing evidence, its real-world applicability and long-term effectiveness remain uncertain. Future prospective clinical trials are needed to evaluate its safety, feasibility, and therapeutic impact.

CONCLUSION

Thought is no longer considered a mere derangement of separate areas within the brain. However, most patients continue to suffer from detrimental functional limitations and unpredictable responses, although symptomatic treatment using existing agents is dramatically effective.

This paper proposes an integrative therapeutic model based on three core pillars:

- Network-accelerated pharmacological optimization
- Network-level neuromodulation (invasive and noninvasive).
- Task-specific intensive motor retraining.

The combined use of these strategies aims to shift treatment from symptomatic relief toward functional reorganization of motor networks. Each component has strong evidence supporting its effectiveness in isolation, and their integration may offer a promising strategy for improving long-term outcomes.

Future studies should examine protocols that optimize timing, intensity, and customization of interventions, incorporate network biomarkers to guide therapy, and evaluate functional outcomes through longitudinal studies.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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ETHICS STATEMENTS

Not applicable.

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نهج علاجي جديد لتحسين اضطرابات الحركة

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الخلاصة

الهدف: تطوير ونشر مفهوم علاجي متعدد الوسائط جديد لاضطرابات الحركة، لا يقتصر على تخفيف الأعراض فحسب، بل يهدف أيضًا إلى إعادة تنظيم الشبكات الحركية وظيفيًا، مما يسمح بتعافي أكثر استقرارًا وفعالية.

الطرق: تم استخدام نموذج قائم على الشبكات العصبية، يتألف من ثلاث وحدات متكاملة:

- تعديل دوائي لوظائف الشبكة العصبية.
- تعديلات مشتركة، تدخلية وغير تدخلية، للتعديلات التدخلية.
- إعادة تأهيل حركي مُخصصة لكل مهمة بجرعات علاجية مثلى، بهدف إعادة بناء الدوائر العصبية المختلة.

تم بناء هذا النموذج من خلال ملاحظات في علم الأعصاب النظمي والتصوير الوظيفي لديناميكيات الشبكة العصبية التي تشمل العقد القاعدية، والقشرة الدماغية، والمهاد، والمخيخ.

النتائج: يهدف التدخل إلى ما هو أبعد من تخفيف الأعراض، وهو تطبيع الاتصال غير الطبيعي، والنشاط التذبذبي، واللدونة غير التكيفية في الشبكات الحركية المعنية. على الرغم من أن التحقق السريري تجريبي، إلا أن التحليل النظري يدعم فكرة أن الجمع بين العلاج الدوائي، ومعدلات الأعصاب، وإعادة تأهيل الحركة قد يعزز إعادة تنظيم الوظائف الحركية، ويكون فعالاً في التحسن طويل الأمد.

الاستنتاج: قد يتفوق هذا العلاج متعدد الوسائط، القائم على فهم الشبكة العصبية، على أساليب علاج اضطراب الحركة الحالية، ليس فقط بتخفيف الأعراض، بل أيضاً بتسهيل إعادة تنظيم الشبكة الحركية التكيفية. ومن المتوقع أن يؤدي هذا المزيج من العلاجات إلى نتائج وظيفية فائقة على المدى الطويل، وتقليل التباين في استجابة المرضى.

الكلمات المفتاحية: اضطرابات الحركة، العقد القاعدية، التعديل العصبي، إعادة التنظيم الوظيفي، التحسين الدوائي