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Review

Deep Brain Stimulation as a Rehabilitation Amplifier A Precision, Network-Guided Framework for Functional Restoration in Movement Disorders

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Abstract

Deep brain stimulation (DBS) is increasingly understood as a precision neuromodulation therapy capable of influencing distributed basal ganglia–thalamo–cortical and cerebellothalamic networks. Although its symptomatic benefits in Parkinson’s disease, essential tremor, and dystonia are well established, the extent to which DBS supports motor learning, adaptive plasticity, and participation in rehabilitation remains insufficiently defined. Traditional interpretations of DBS as a focal or lesion-like intervention are being challenged by electrophysiological and imaging evidence demonstrating multiscale modulation of circuit dynamics. DBS may enhance rehabilitation outcomes by stabilizing pathological oscillations and reducing moment-to-moment variability in motor performance—conditions that enable consistent task execution and more effective physiotherapy, occupational therapy, and speech–language interventions. Yet this potential is not fully realized in clinical practice due to interindividual variability, incomplete mechanistic understanding, and the limited specificity of current connectomic biomarkers for predicting functional gains. Technological advances such as tractography-guided targeting, directional leads, sensing-enabled devices, and adaptive stimulation are expanding opportunities to align neuromodulation with individualized circuit dysfunction. Despite these developments, major conceptual and empirical gaps persist. Few studies directly examine how stimulation-induced changes in neural stability interact with structured rehabilitation to promote long-term functional recovery. Heterogeneity in therapeutic response and rehabilitation access further complicates interpretation of outcomes. Clarifying these relationships is essential for developing precision frameworks that integrate DBS with rehabilitative strategies. This review synthesizes mechanistic, imaging, and technological evidence to outline a network-informed perspective of DBS as a potential facilitator of rehabilitation-driven functional improvement and identifies priorities for future research aimed at optimizing durable functional restoration.

Keywords: deep brain stimulation; connectomics; movement disorders; Parkinson’s disease; essential tremor; dystonia; adaptive neuromodulation; rehabilitation; tractography; sensing-enabled DBS

1. Introduction

Essential tremor (ET), Parkinson's disease (PD), and dystonia are the most prevalent chronic movement disorders and major contributors to long-term motor disability worldwide [1–4]. Their global prevalence continues to rise, driven largely by population ageing and increased life expectancy, underscoring the need for therapeutic strategies that sustain long-term functional independence [5–7]. This growing burden highlights the limitations of purely symptomatic approaches and reinforces the importance of interventions that not only control motor features but also enhance functional capacity and participation in daily life.

Deep brain stimulation (DBS) has become the most effective neurosurgical therapy for medication-refractory movement disorders, marking a transition from irreversible ablative procedures to reversible and adjustable neuromodulation [8–11]. Early interpretations framed DBS as a “functional lesion,” reflecting its ability to suppress pathological activity within targeted nuclei [12,13]. However, accumulating electrophysiological, neurochemical, and imaging evidence has reframed DBS as a network-level intervention, acting through modulation of distributed motor circuits rather than by focal suppression at the stimulation site [14–17]. Recent advances in connectomics have further highlighted the limitations of classical nucleus-centric models, showing that DBS outcomes are shaped not solely by electrode proximity to anatomical targets but by the broader patterns of circuit engagement and network reorganization enabled through stimulation [18–20]. This shift from anatomical to systems-based reasoning challenges traditional assumptions about target selection, mechanisms of action, and the therapeutic reach of DBS.

Despite these mechanistic and technological advances, DBS remains underutilized as a partner to structured rehabilitation, even though neuromodulation can provide a stable physiological substrate for task-specific training, motor learning, and long-term skill acquisition [21–23]. By stabilizing motor fluctuations and reducing performance variability, DBS may enable patients to participate more effectively in physiotherapy, occupational therapy, and speech–language interventions. Viewed in this context, DBS acts as a rehabilitation amplifier, creating neural conditions under which plasticity can be harnessed to promote durable functional restoration.

The purpose of this review is to synthesize mechanistic concepts, imaging frameworks, and technological innovations that support a precision, network-based integration of DBS with multidisciplinary rehabilitation. We outline the evolution from lesion-based and nucleus-centric models to modern connectomic paradigms; examine phenotypic and circuit-level heterogeneity; and discuss how DBS, when combined with structured rehabilitation, can optimize motor performance, functional independence, and long-term outcomes in movement disorders.

2. Materials and Methods

Literature Search Strategy and Scope of the Review

This review synthesizes mechanistic, imaging, technological, and clinical evidence relevant to the interaction between DBS and rehabilitation. Literature was identified through searches in PubMed, Scopus, and Web of Science up to December 2025 using combinations of the terms “deep brain stimulation,” “motor learning,” “plasticity,” “connectomics,” “rehabilitation,” “adaptive DBS,” “tractography-guided targeting,” and disorder-specific terms including “Parkinson's disease,” “essential tremor,” and “dystonia.” Reference lists of key publications were examined to supplement database searches.

No language or date restrictions were applied, although priority was given to mechanistic, imaging, and clinically relevant studies published within the last decade alongside foundational historical work. As this is a conceptual narrative review, no systematic screening, risk-of-bias assessment, or meta-analysis was performed. Studies were selected for relevance to (i) multiscale DBS mechanisms; (ii) imaging supporting circuit-level targeting; (iii) technological innovations in neuromodulation; and (iv) rehabilitation approaches intersecting with DBS.

This review is based solely on previously published studies and does not involve any new research with human participants or animals.

3. Integrated Framework for Network-Level DBS Mechanisms and Clinical Translation

3.1. Epidemiology and Global Burden of Movement Disorders

ET, PD, and dystonia are among the most common chronic movement disorders and together represent substantial long-term sources of disability (Figure 1). ET affects approximately 4–5% of adults over 65 years of age, underscoring its significant public-health impact in ageing populations [1]. The global prevalence of PD is projected to surpass 12 million by 2040, largely driven by demographic ageing and increased life expectancy [2]. Dystonia represents the third most frequent movement disorder encountered in tertiary care, encompassing focal, segmental, generalized, and genetic forms with heterogeneous clinical trajectories [3,4]. Although considerably less prevalent, rare and highly disabling dystonic phenotypes continue to be reported, and emerging case-based evidence demonstrates that DBS can restore meaningful functional capacity even in exceptional or atypical presentations [24,25].

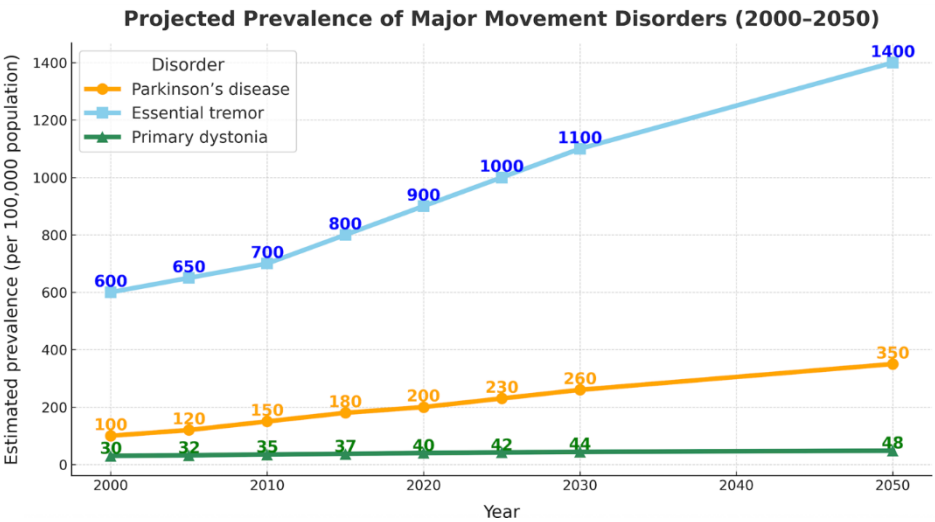


Figure 1. Projected prevalence of major movement disorders (2000–2050). Values reflect approximate population-level trends derived from published epidemiological data. These projections illustrate the growing global burden of movement disorders and underscore the need for therapeutic strategies that enhance functional capacity and reduce long-term disability.

Beyond their cardinal motor manifestations, these conditions impose substantial functional, psychosocial, and socioeconomic burdens. Global modelling indicates a sustained rise in PD-associated disability, reflecting both increased prevalence and progression-related morbidity [5–7]. ET, long mischaracterized as a benign disorder, is now recognized as a major contributor to functional dependence, reduced quality of life, and increased healthcare utilization [26]. Although less frequently quantified in burden-of-disease frameworks, dystonia also contributes significantly to disability-adjusted life years, chronic pain, vocational limitations, and long-term care needs [27].

These epidemiological trends emphasize the need for therapeutic strategies that move beyond symptom suppression to restore functional capacity and support sustained independence. When embedded within structured rehabilitation paradigms, DBS offers an opportunity to modulate pathological networks and enhance adaptive plasticity, thereby promoting more durable improvements in daily functioning.

3.2. Historical Evolution of DBS Concepts

3.2.1. From Lesion-Based Surgery to Reversible Neuromodulation

The development of DBS marked a major shift from irreversible ablative procedures toward adjustable neuromodulation (Figure 2). Lesioning techniques such as thalamotomy and pallidotomy demonstrated that interrupting aberrant basal ganglia signaling could reduce tremor, rigidity, and dyskinesias, but concerns regarding irreversibility and adverse effects limited their widespread use [10,11].

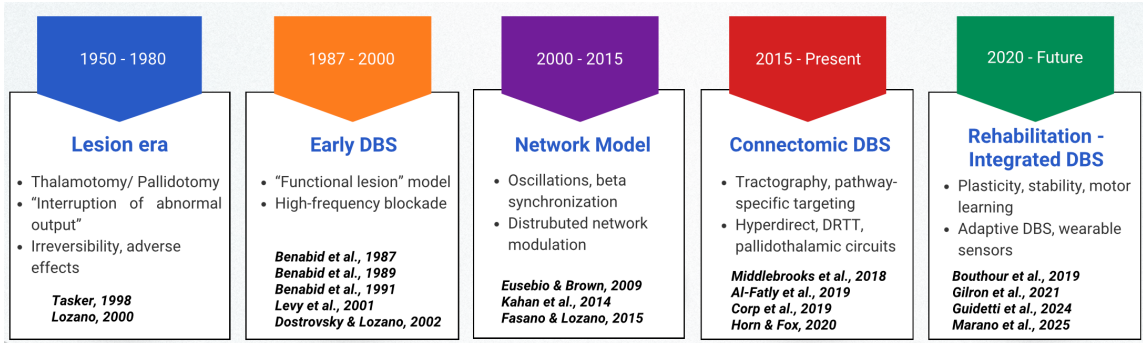


Figure 2. Conceptual evolution of deep brain stimulation (DBS). Five major eras illustrate the progression from lesion-based interventions to modern, network-guided neuromodulation. Early irreversible lesioning was replaced by high-frequency DBS, later reinterpreted within network-level frameworks. Advances in connectomics enabled pathway-specific targeting, and from 2020 onward, sensing-enabled and adaptive DBS technologies have driven the transition toward rehabilitation-integrated neuromodulation.

The introduction of high-frequency stimulation by Benabid and colleagues in the late 1980s was transformative. Ventral intermediate nucleus (VIM) stimulation reproduced the therapeutic effects of lesions while preserving reversibility and allowing postoperative titration [8,28]. This approach rapidly expanded to Parkinson’s disease (PD), establishing DBS as a viable alternative to lesioning [9].

Early mechanistic interpretations framed DBS as a “functional” or “adjustable lesion,” reflecting its capacity to suppress pathological activity within targeted nuclei [12,13]. Subsequent electrophysiological evidence, however, demonstrated that high-frequency stimulation does not simply silence local neuronal activity. Instead, it imposes a structured exogenous drive that disrupts pathological oscillatory dynamics [14]. This transition from a lesion-based to a network-based explanatory model began to suggest that clinical benefits extend beyond focal inhibition.

3.2.2. Nucleus-Centric DBS and Emerging Complexity

Classical DBS strategies aligned each movement disorder with a canonical target—VIM for essential tremor, the subthalamic nucleus (STN) for PD, and the globus pallidus internus (GPi) for dystonia [29,30], as depicted in Figure 3. This nucleus-based model produced major therapeutic advances, with randomized trials confirming durable improvements across tremor, bradykinesia, rigidity, and dystonia [31–35].

As clinical experience grew, several observations challenged this framework. Physiological responses to stimulation proved more complex than simple suppression, with heterogeneous axonal and somatic effects extending beyond the stimulation site [36–38]. Phenotypic variability within ET, PD, and dystonia complicated attempts to generalize stimulation responses [4,18,39].

Additionally, early safety concerns regarding MRI in patients with implanted devices limited high-resolution characterization of stimulation-induced changes [40,41]. The subsequent development of device-specific MRI protocols enabled higher-resolution interrogation of

stimulation-related network effects [42], solidifying the conceptual transition from a focal intervention toward a therapy that exerts its benefits by engaging distributed motor circuits.

	PARKINSON'S DISEASE	TREMOR SYNDROMES	DYSTONIA
KEY PATHWAY	- Hyperdirect pathway - Basal ganglia – thalamo-cortical loop (beta synchrony)	- Dentatorubrothalamic - Cerebellothalamic pathway	- Pallidothalamic fibers - Sensorimotor / premotor cortex
DBS TARGETS	STN GPI	VIM PSA/ZI	GPI STN
KEY EFFECT	Beta desynchronization Improves bradykinesia Improves rigidity	Cerebellothalamic regularization Reduces tremor Facilitates fine motor training	Normalization of output signaling Enhances motor control Facilitates motor retraining
REFERENCES	Eusebio & Brown, 2009 Kahan et al., 2019	Middlebrooks et al., 2018 Al-Fatly et al., 2019	Corps et al., 2019 Ostrem et al., 2011

Figure 3. Pathways, DBS targets, and key physiological effects across movement disorders. Schematic overview of dominant dysfunctional circuits, preferred DBS targets, and associated physiological effects across movement disorders.

3.3. Phenotypes and Network Level Heterogeneity

Clinical experience with DBS rapidly demonstrated that major movement disorders exhibit substantial phenotypic heterogeneity, reflected in the variable engagement of basal ganglia–thalamo-cortical and cerebellothalamic networks (Table 1). In PD, tremor-dominant, akinetic-rigid, postural instability / gait disorder (PIGD), and cognitively vulnerable subtypes show distinct circuit dependencies that shape therapeutic response [39,43]. ET and dystonia show comparable diversity, with symptom profiles reflecting differential involvement of motor, cerebellar, and associative pathways [4,44].

Table 1. Phenotypes, Dominant Circuits, and Rehabilitation Implications in DBS.

Technology	Core feature	Clinical / rehabilitation relevance
High-field MRI + tractography	Patient-specific visualization of relevant pathways	Improves targeting precision and reduces side effects, supporting alignment of stimulation with functional goals
Directional leads	Current steering toward therapeutic pathways	Widens therapeutic window; improves stability for high-intensity rehabilitation
Sensing-enabled DBS	Continuous monitoring of physiological biomarkers	Enables objective programming and reduces variability affecting therapy performance
Adaptive DBS (closed-loop)	Stimulation delivered when biomarkers exceed thresholds	Improves gait/tremor stability and supports timing of rehabilitation tasks
Wearable motor sensors	Continuous monitoring of gait, tremor, bradykinesia	Enables therapy personalization and home-based training
Connectomic programming platforms	Lead reconstructions + pathway-activation modeling	Supports individualized programming based on patient-specific networks

Table 1. Phenotype–circuit–rehabilitation mapping in movement disorders. Dominant dysfunctional networks are summarized alongside key rehabilitation implications; PD: Parkinson’s disease; DRTT: dentato–rubro-thalamic tract; STN: subthalamic nucleus; GPI: globus pallidus internus.

Electrophysiological and imaging evidence increasingly supports the view that these conditions represent spectra of circuit dysfunction [15,37]. Tremor severity aligns with cerebellothalamic pathways—especially the dentato-rubro-thalamic path (DRTT)—[16,23], whereas bradykinesia and rigidity relate to hyperdirect cortico–subthalamic projections, and cognitive vulnerability maps onto associative–limbic networks [21,45,46]. In dystonia, responsiveness to GPi stimulation corresponds to pallidothalamic and sensorimotor network engagement [19,33,47,48].

Network specificity also explains therapeutic variability within tremor syndromes: while classic ET responds to VIM stimulation, ET-plus and cerebellar variants may benefit from posterior subthalamic area (PSA) or zona incerta (ZI) targeting, where cerebellothalamic fibers converge [16,18]. Dystonic tremor, reflecting hybrid cerebellothalamic–pallidal involvement, often requires individualized targeting [49]. Rare dystonic presentations, including GA1-related and KMT2B-associated dystonia, can improve meaningfully with GPi-DBS, though outcomes remain variable across syndromes [24,25].

Overall, these findings, summarized in Table 1, demonstrate that identifying the dominant dysfunctional circuit—cerebellothalamic, hyperdirect, pallidothalamic, or associative–limbic—can guide individualized DBS strategies and improve clinical outcomes, as illustrated in Figure 3.

3.4. Local Effects and Multiscale Biological Mechanisms

Building on these network-level distinctions, mechanistic insights into DBS derive from studies of STN stimulation in PD, where electrophysiological and cellular responses have been characterized in greatest detail (Table 2). At the microscale, DBS preferentially activates large myelinated axons while suppressing intrinsic somatic firing, generating orthodromic and antidromic signals that reshape information flow across basal ganglia circuits [12,38]. High-frequency stimulation introduces a more regular firing pattern that counteracts pathological bursting and excessive beta synchrony, reducing moment-to-moment variability in motor output [50,51]

Table 2. Multiscale Biological Mechanisms of DBS and Their Relevance for rehabilitation.

Mechanistic level	Key mechanisms	Rehabilitation relevance
Microscale (neuronal)	Axonal activation; somatic suppression; altered firing patterns	Stabilizes motor output and supports consistent performance during training
Mesoscale (oscillatory)	Beta suppression; ERNA; short-latency entrainment	Enhances motor learning and improves within-session stability
Macroscale (network)	Modulation of hyperdirect, cerebellothalamic, and pallidothalamic circuits	Aligns stimulation with gait, fine-motor, and functional rehabilitation goals
Non-neuronal/ molecular	Astrocytic modulation, adenosine release, trophic signaling	Supports adaptive plasticity and learning-dependent improvement

Table 2. Multiscale neuronal, oscillatory, network-level, and non-neuronal mechanisms of DBS relevant to functional rehabilitation, illustrating how these mechanisms relate to rehabilitative potential.

Local responses differ across targets. Human recordings demonstrate that STN stimulation rapidly suppresses intrinsic firing, VIM stimulation evokes a brief activation burst before silencing, and GPi stimulation produces transient inhibition followed by recurrent reactivation—patterns reflecting the unique convergence of afferent and efferent connections at each site [37]. At the mesoscale, DBS suppresses pathological beta oscillations, induces short-latency cortico–subcortical entrainment, and evokes resonant neural activity (ERNA), a high-frequency signature whose amplitude scales with stimulation intensity and differs between STN and GPi [37]. These oscillatory dynamics represent a physiological bridge between local neuronal effects and broader functional improvements, consistent with macroscale network modulation summarized in Table 2.

Beyond neuronal elements, DBS also modulates non-neuronal components of the microenvironment. Astrocytes regulate neurotransmission through activity-dependent glutamate

and adenosine release, influence extracellular matrix signaling, fostering conditions that support plasticity. Electrode implantation and chronic stimulation additionally induce glial and epigenetic adaptations—including reductions in astrocytic reactivity and changes in DNA methylation and microRNA expression—that may contribute to long-term functional stability [36].

Together, these multiscale responses illustrate that DBS operates through layered mechanisms encompassing cellular, oscillatory, and network domains. Although not yet indicative of a definitive disease-modifying effect, this multilevel modulation provides a neurophysiological foundation upon which rehabilitation can build by promoting more consistent motor performance and enhancing the capacity for adaptive plasticity, an alignment illustrated in Figure 4, which outlines the principal rehabilitation goals after DBS.

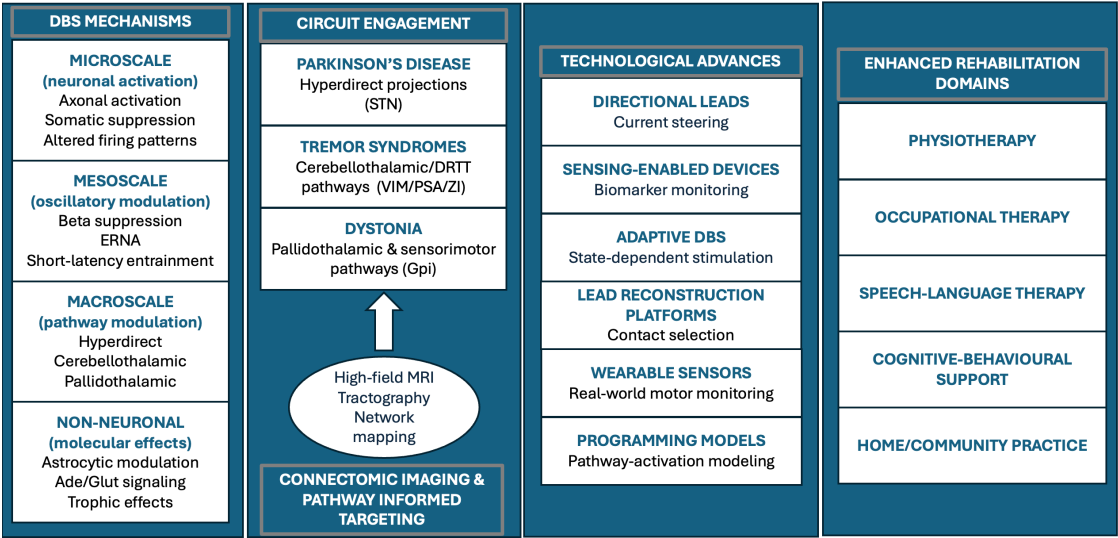


Figure 4. Integrative overview of DBS mechanisms, disease-specific circuit engagement, key technological advances, and rehabilitation domains. Connectomic imaging and pathway-informed targeting link multiscale neuromodulation effects with clinical and rehabilitative applications.

3.5. Imaging Evidence for Network-Level Mechanisms

Imaging studies have been central to reframing DBS mechanisms. Before MRI protocols compatible with implanted hardware became available, PET and SPECT provided the first in vivo evidence that stimulation induces metabolic and perfusion changes in regions distant from the electrode site— including motor cortex, cerebellum, thalamus, and basal ganglia—supporting a circuit-level interpretation of DBS effects [41,52]. Although limited by radiation exposure and temporal resolution, these modalities established the foundational concept that DBS modifies activity across broader motor systems.

The advent of device-specific MRI protocols enabled higher-resolution characterization of these network effects [42] (Table 1). Structural MRI and diffusion tractography studies have demonstrated that clinical outcomes correlate more strongly with stimulation of symptom-relevant pathways than with proximity to nuclear boundaries [48,53,54]. These structural imaging findings reinforce the previously outlined symptom–circuit relationships: stimulation is most effective when it interfaces with the pathways that underlie each motor or cognitive phenotype. Across disorders, pathway-level alignment—not anatomical proximity—emerges as the principal determinant of DBS efficacy.

Functional imaging further supports this distributed-network perspective [21]. Resting-state fMRI identifies connectivity signatures that predict DBS responsiveness—stronger cerebellothalamic coupling in tremor syndromes and restoration of cortico–subcortical flexibility in PD [55,56]. In dystonia, intact GPi connectivity with premotor and cingulate regions predicts faster and more durable clinical improvement [19,48,57].

Longitudinal imaging studies indicate that DBS induces progressive reorganization of motor networks rather than fixed, stimulation-dependent effects. STN stimulation alters cortico–subcortical communication patterns in PD, GPi stimulation normalizes sensorimotor network architecture in dystonia, and VIM/PSA stimulation modulates cerebellothalamic pathways in tremor syndromes, with tract involvement correlating with sustained symptom reduction [16,57,58]. Experimental work further suggests that thalamic stimulation may enhance motor learning, reinforcing the concept that DBS can facilitate adaptive network changes [59].

When considered together, imaging evidence—from early metabolic studies to contemporary structural and functional connectomics—demonstrates that DBS exerts its clinical effects through distributed circuits whose organization shapes symptom expression and treatment responsiveness.

3.6. Surgical and Technological Advances Enabling Precision DBS

Advances in imaging, computational planning, and implantable hardware have progressively shifted DBS from a nucleus-based therapy to a circuit-informed, precision neuromodulation approach (Table 3). High-field MRI now enables reliable visualization of subcortical anatomy, while diffusion tractography provides patient-specific reconstructions of pathways relevant to tremor, bradykinesia–rigidity, and dystonia [15,16,18,19,54]. These modalities support targeting strategies that align electrode placement with symptom-relevant fiber trajectories and reduce reliance on indirect anatomical landmarks [40,42].

Table 3. Technological Innovations in DBS and Their Functional/Rehabilitative Implications.

Technology	Core feature	Clinical / rehabilitation relevance
High-field MRI + tractography	Patient-specific visualization of relevant pathways	Improves targeting precision and reduces side effects, supporting alignment of stimulation with functional goals
Directional leads	Current steering toward therapeutic pathways	Widens therapeutic window; improves stability for high-intensity rehabilitation
Sensing-enabled DBS	Continuous monitoring of physiological biomarkers	Enables objective programming and reduces variability affecting therapy performance
Adaptive DBS (closed-loop)	Stimulation delivered when biomarkers exceed thresholds	Improves gait/tremor stability and supports timing of rehabilitation tasks
Wearable motor sensors	Continuous monitoring of gait, tremor, bradykinesia	Enables therapy personalization and home-based training
Connectomic programming platforms	Lead reconstructions + pathway-activation modeling	Supports individualized programming based on patient-specific networks

Table 3. Key DBS technologies and their clinical and rehabilitation implications.

Trajectory planning has also evolved with the integration of multimodal image fusion, tractography constraints, and vascular avoidance algorithms, enabling safe implantation while maximizing engagement of intended therapeutic structures [18,42,54]. Intraoperative workflows increasingly incorporate imaging-guided verification or microelectrode recording, offering complementary methods to confirm lead positioning and reducing operative variability[60–62].

Hardware innovations have expanded the capacity to tailor stimulation to individual network architecture (Table 3) [63]. Directional leads allow current steering away from structures associated with side effects and toward pathways linked to therapeutic benefit, effectively broadening the clinical stimulation range[64,65]. Sensing-enabled pulse generators can record neural biomarkers such as beta bursts or tremor-related oscillations, providing objective data for programming and chronic physiological assessment [15,66,67]. These systems form the basis for adaptive (closed-loop)

DBS, in which stimulation continuously adjusts according to real-time neural states, improving gait and tremor stability and reducing unnecessary energy delivery.

Modern programming platforms incorporate postoperative lead reconstructions and tractography-based pathway models, enabling clinicians to visualize which contacts most effectively engage the patient’s dominant dysfunctional circuit [15,68]. This approach supports efficient, rational programming while facilitating alignment of stimulation with individual functional goals.

Table 4. Rehabilitation Domains After DBS and Main Functional Goals.

Domain	Main goals
Physiotherapy	Improve gait, balance, amplitude, and dual-task performance
Occupational therapy	Enhance dexterity, handwriting, and ADLs
Speech-language therapy	Improve articulation, phonation, and intelligibility
Cognitive-behavioral support	Maintain executive functioning, mood, and therapy engagement
Home / community training	Promote task-specific practice and generalization to daily life

Table 4. Primary rehabilitation domains and goals following DBS. These domains outline the therapeutic framework typically integrated with neuromodulation-based care.

3.7. *Rehabilitation-Integrated DBS: Towards Network Restoration*

DBS may facilitate rehabilitation by stabilizing neural dynamics and reducing moment-to-moment motor variability—conditions essential for motor learning [14,67,69]. This physiological regularization enhances responsiveness to physiotherapy and improves task-specific retraining efficiency, partly by reducing motor variability [70]. Clinically, this may translate into improved capacity for locomotor training, including gait and balanced-focused interventions. This clinical effect aligns with recent mechanistic summaries demonstrating that DBS restores more physiologically regular network activity and supports functional performance [71].

Wearable sensor data show that DBS reduces gait irregularity and enhances responsiveness to physiotherapy, especially in balance and lower-limb programs[72]. Remote monitoring studies similarly indicate that periods of lower subthalamic beta activity may correspond to more favorable windows for gait rehabilitation [73]. Balance and postural control remain only partially responsive to DBS alone, reinforcing the need for targeted rehabilitation strategies [74].

A recent Delphi consensus supports early, task-specific physiotherapy after DBS, highlighting benefits in gait amplitude, dual-task performance, and postural stability when training is delivered once stimulation parameters are clinically optimized [70]. Case-based evidence also suggests that combining DBS with intensive, goal-directed rehabilitation can produce additional gains in functional mobility and daily activities beyond stimulation alone [75]. Additional clinical data suggest that structured rehabilitation improves mobility and functional performance even years after DBS implantation [76].

Newer DBS technologies strengthen these clinical applications. Sensing-enabled and adaptive systems offer physiological markers that may help clinicians time therapy sessions to periods of optimal neural stability [77–79]. Neuromodulation-enhanced rehabilitation frameworks further propose that DBS may support adaptive plasticity [72,75,80]. Advances in functional mobility assessment techniques, such as integrated motion-capture and pressure-based gait analysis, may further enhance the ability to quantify rehabilitation response after DBS [81].

In this view, DBS may serve as a physiological facilitator of therapy-driven improvement, though confirmation requires mechanistic and clinical validation. Complementary rehabilitative technologies such as virtual reality may also enhance gait and balance training in DBS patients in selected phenotypes [82].

4. Limitations

This review is narrative rather than systematic, and not all relevant literature may have been captured. Heterogeneity in study design, imaging methods, and follow-up restricts comparability. Mechanistic insights derive disproportionately from PD studies.

Rehabilitation protocols after DBS remain highly heterogeneous, with limited evidence to guide optimal timing, intensity, or phenotype-specific adaptations.

5. Future Directions

Future progress in deep brain stimulation (DBS) will require integration of physiological biomarkers, individualized connectomic targeting, and adaptive neuromodulation strategies. Sensing-enabled systems may align stimulation and rehabilitation with neural states favorable for motor learning, while patient-specific pathway modeling could refine circuit selection across phenotypes. Real-world motor monitoring through wearable sensors may also help quantify long-term plasticity and guide rehabilitation dosing. Ultimately, mechanistically informed clinical trials are needed to determine how DBS-induced network stabilization interacts with structured rehabilitation to support durable functional recovery.

6. Conclusions

DBS has progressed from a focal intervention to a systems-level neuromodulation therapy informed by circuit physiology, imaging biomarkers, and technological innovation. Evidence suggests that DBS modulates distributed networks, stabilizes motor output, and may enhance conditions for rehabilitation-driven improvement.

Technological developments, including directional leads, tractography-guided targeting, and adaptive systems, strengthen the interface between stimulation and therapy. Yet integrated frameworks linking neuromodulation with functional recovery remain nascent.

Overall, DBS may act as a physiological facilitator of rehabilitation, a model requiring further mechanistic refinement and clinical validation.

7. Key highlights

- Deep brain stimulation (DBS) enhances the stability of motor performance, creating favorable conditions for structured rehabilitation.
- Connectivity-informed targeting improves clinical outcomes by aligning stimulation with patient-specific circuit architecture.
- Technological advances—including tractography-based planning, directional leads, and sensing-enabled systems—support more precise and individualized neuromodulation.
- Rehabilitation integrated with DBS can amplify functional gains by leveraging stabilized neural dynamics.
- Future frameworks will incorporate adaptive stimulation, biomarker-guided therapy, and real-world motor monitoring to optimize long-term functional restoration.

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Informed Consent Statement: Not applicable. This study did not involve human participants, identifiable data, or new clinical interventions requiring informed consent.

Data Availability Statement: No new data were generated or analyzed in this study. All data discussed in this review are derived from previously published sources.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

DBS	Deep brain stimulation
DRTT	Dentato–rubro–thalamic tract
ET	Essential tremor
ERNA	Evoked resonant neural activity
fMRI	Functional magnetic resonance imaging
GA1	Glutaric aciduria type I
Gpi	Globus pallidus internus
KMT2B	Lysine methyltransferase 2B
MRI	Magnetic resonance imaging
PD	Parkinson’s disease
PET	Positron emission tomography
PIGD	Postural instability/gait disorder
PSA	Posterior subthalamic area
SPECT	Single-photon emission computed tomography
STN	Subthalamic nucleus
VIM	Ventral intermediate nucleus
ZI	Zona incerta

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