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Personalised Deep Brain Stimulation Programming for Parkinson's Disease: Current Advances and Future Directions

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Abstract. Deep brain stimulation (DBS) is an established treatment for selected patients with Parkinson's disease (PD), but its effectiveness depends partly on how stimulation is programmed after surgery. Programming has become more challenging and the optimal settings differ between patients. This narrative review considers four approaches to personalising DBS programming: anatomical guidance, electrophysiological biomarkers, computational methods with algorithm-assisted and adaptive stimulation. Imaging and connectomic data can help identify contacts and stimulation settings according to electrode location and individual anatomy. Neural recordings offer more data, although beta activity alone does not adequately represent the range of motor dysfunction in PD. Computational methods can reduce the amount of clinical testing needed, but most have not yet been validated in larger clinical studies. Adaptive DBS has stronger emerging clinical evidence, including multicentre and longer-term studies, although the most useful control signal may differ by symptom. Multicentre studies combining these four approaches directly with conventional programming may be needed to establish whether they produce meaningful and durable improvements in patient outcomes.

Keywords: Parkinson's disease, deep brain stimulation (DBS), electrophysiological biomarkers, precision neuromodulation

1. Introduction

Deep brain stimulation (DBS) is one of the treatments for patients with Parkinson's disease (PD), particularly when motor fluctuations, dyskinesia or medication-refractory tremor are inadequately controlled by medication. It can improve motor function, medication requirements and quality of life [1, 2]. However, these outcomes depend not only on electrode placement but also on post-surgical stimulation programming [3]. Clinicians must decide which contacts to activate and adjust parameters according to the symptoms and adverse effects observed in each patient [3, 4].

Conventional DBS programming (cDBS) typically starts with a monopolar review, where clinicians test each contact to identify the range between clinical benefit and adverse effects [4]. Interpretation can be difficult soon after surgery because the stun effect may temporarily alter

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electrophysiological signals used to guide programming [5, 6]. Moreover, variability in settings between patients according to lead location, neuroanatomy, symptoms, medication state and neural activity has become harder to manage with directional and segmented leads, which offer more possible stimulation settings. It is impractical to test every combination in the clinic. Furthermore, it can hardly provide a full picture of symptoms and neural activity during clinical visits because they might fluctuate in daily life.

To overcome these challenges, DBS programming is shifting toward more personalised approaches. Anatomically informed approaches use patient-specific imaging to improve electrode-targeting precision, while biomarker-guided strategies use electrophysiological signals to characterise the neural state. Algorithm-assisted approaches use structured workflows and computational optimisation to manage programming complexity [4, 7]. Adaptive DBS also dynamically adjusts stimulation in real time based on each patient's physiological or behavioural signals [8]. Clinical research has moved from early feasibility studies to multicentre evaluations [9, 10]. Recent studies further highlight the potential and limitations of symptom and patient-specific programming [11-13]. Therefore, these approaches address different aspects of personalised programming.

Clinical evidence has not developed at the same pace across these approaches. This review therefore focuses on anatomically informed, biomarker-guided, algorithm-assisted and adaptive DBS programming, examining what each might currently facilitate for personalised programming, where evidence remains limited and how these approaches might be combined in clinical practice.

2. Current advances in personalised DBS programming

2.1. Anatomically informed programming

After DBS implantation, postoperative imaging allows clinicians to locate each electrode contact in relation to the intended stimulation target. Clinicians can use this information to identify contacts more likely to produce a therapeutic response and focus subsequent clinical testing on them, reducing the need to test every available contact equally. In a prospective study of 89 patients undergoing image-guided subthalamic DBS, motor symptoms and quality of life improved following surgery [1].

Connectomic approaches focus on which neural circuits are modulated. Using patient-specific tractography instead of normative connectomes, a retrospective study of 69 patients showed that greater modulation of fibres connecting the subthalamic nucleus to the supplementary motor area, premotor cortex and globus pallidus correlated with better motor outcomes, particularly for tremor and rigidity [14]. Therapeutic efficacy increasingly depends on modulating disease-relevant brain networks rather than stimulating discrete anatomical targets.

Beyond guiding initial contact selection, it also supports post-implantation adjustment. Structured image-guided programming algorithms that integrate postoperative imaging, lead localisation and volume-of-tissue-activated modelling with systematic stimulation optimisation distinguish suboptimal programming from lead misplacement [3]. So, anatomical guidance approach could identify and correct them before surgical revision is required, which might help throughout the post-DBS implantation programming phase.

However, it is insufficient for fully personalising DBS programming. In a prospective cohort of 49 patients, although reduced preoperative superior frontal cortex volume indicated poorer postoperative motor outcomes, it showed no significant association with the overall burden of non-motor symptoms, highlighting the limited ability of structural imaging alone to reflect the full

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postoperative clinical response [13]. Also, connectomic analyses are sensitive to methodological variability in image registration, tractography reconstruction and connectivity modelling, which may limit the reproducibility and clinical translation of connectivity-guided programming [14]. Therefore, while it may inform where stimulation should be delivered, it cannot determine how delivery should change in response to dynamic physiological or clinical states. It might require incorporating objective physiological biomarkers into personalised programming.

2.2. Biomarker-guided programming

Electrophysiological signals might help reflect changes in neural activity over time. It is recorded from implanted electrodes, providing data for contact selection and parameter adjustment. Researchers have studied subthalamic beta-band (13-30 Hz) local field potential activity most extensively. Beta activity is associated with bradykinesia and rigidity and decreases with dopaminergic treatment and effective DBS. In 33 patients with more than 24,000 recorded medication intakes, beta power decreased between 30 and 140 minutes after levodopa administration, with a greater reduction in the low-beta than the high-beta range [15]. Also, it remains sufficiently stable for long-term use. A longitudinal study of 33 patients followed for up to 44 months found that the spatial distribution and centre frequency of subthalamic beta activity remained largely stable after the initial postoperative period [16]. This stability makes beta activity a potential chronic marker for contact selection. However, its reliability may be lower soon after implantation. During the first three to four postoperative weeks, the stun effect can alter beta activity, so recordings from this period may not reflect the patient's longer-term neural state [5, 6].

Beta activity does not represent all aspects of motor dysfunction in PD. In a multicentre study of 119 patients, a model combining aperiodic broadband power, low-beta activity and low-gamma activity accounted for more variation in motor symptom severity than low-beta power alone [17]. This suggests that relying on beta power only may overlook neural signals relevant to a patient's motor state. Another study examined periodic and aperiodic activity and found that they related to different motor impairments, it might be related to overall motor severity, while some are more closely related to bradykinesia and change with medication state [18]. These observations argue against relying on a single electrophysiological marker for every patient or symptom.

2.3. Algorithm-assisted programming

The growing number of programmable settings in recent DBS has made parameter selection increasingly challenging. Directional leads add further choices to contact selection, while amplitude, pulse width and frequency must also be adjusted for individual patients. Therefore, algorithms can narrow these programming choices by recommending contacts or stimulation settings based on predefined clinical rules, patient-specific data, or machine-learning models. In a study with 50 patients transitioned to adaptive stimulation, a standardised workflow guided threshold selection and verified the neural signal and frequency used to control stimulation [4]. Programming remained clinician-led, but the protocol provided a consistent procedure the multidisciplinary team could reproduce.

Machine-learning-based neural decoding offers a more individualised approach to computationally assisted DBS programming. In a single-patient feasibility study, a remotely optimised movement-responsive neural decoder improved hand movement speed and typing performance and reduced dyskinesia compared with continuous stimulation [7]. The decoder could also be updated remotely using physiological data recorded outside the clinic. This allowed

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programming to be refined using the patient's neural activity over time rather than relying only on assessments performed during clinic visits. However, evidence from a single patient is insufficient to determine whether this approach will improve programming in the wider PD population.

Evidence for algorithm-assisted programming is still preliminary. Most studies have been small or single centre, with few direct comparisons against programming by experienced clinicians. Performance also depends on the quality of the data used to develop and test the models. These limitations make it difficult to determine whether it improves DBS programming or simply automates parts of the existing workflow. Larger comparative studies are needed to show whether it can reduce programming time or improve clinical outcomes. However, it could enhance clinical decision-making by improving the efficiency and consistency of complex DBS programming.

2.4. Adaptive programming

Conventional DBS delivers stimulation using parameters selected during clinical programming sessions, but a patient's motor state can change considerably between these visits. Adaptive DBS instead uses recorded physiological or behavioural signals to modify stimulation as these changes occur. In a blinded randomised crossover feasibility trial of four patients, stimulation-entrained gamma oscillations were used as patient-specific feedback signals related to dopaminergic state. Compared with clinically optimised continuous DBS, adaptive stimulation showed improved motor symptoms and quality of life [8].

Larger patient groups have examined feasibility observed in small cohorts. Adaptive DBS Algorithm for Personalised Therapy in Parkinson's Disease (ADAPT-PD) trial enrolled 68 participants previously treated with continuous DBS and evaluated single and dual-threshold adaptive stimulation. Researchers evaluated 91% of participants in dual-threshold mode and reported no serious device-related adverse events during follow-up [9]. Further analysis found that most participants had beta-band signals suitable for controlling adaptive stimulation, including across different medication states [10]. This matters for chronic use because the control signal must remain detectable as medication-related motor states change. Longer-term data are available from 19 patients followed for one year after switching from conventional to adaptive stimulation. Improvement was greater in patients with more severe wearing-off symptoms at baseline [2], suggesting that patients with motor fluctuations may benefit more from adaptive stimulation.

Symptom-specific adaptive stimulation has produced different effects across motor outcomes. In a randomised feasibility trial of five patients, gait-phase-synchronised stimulation improved gait measures and reduced falls compared with clinically optimised continuous DBS [11]. By contrast, low-frequency adaptive stimulation for freezing of gait improved gait performance but provided less effective overall motor control, including tremor, compared to conventional high-frequency stimulation [12]. Together, these studies suggest that an adaptive strategy optimised for one motor symptom may not provide equivalent control of others. Programming may therefore need to prioritise the symptom that causes the greatest clinical burden rather than applying the same adaptive strategy across motor manifestations.

Adaptive DBS allows stimulation to respond to changes in a patient's neural or clinical state rather than remaining fixed between programming visits. Its wider clinical use will depend on whether suitable signals can be recorded reliably over time and whether stimulation can be adjusted safely and consistently from those signals.

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3. Critical synthesis

The four programming approaches address different limitations of conventional DBS programming. Anatomical imaging can identify where stimulation is delivered and help prioritise contacts [1, 3, 14], but it provides limited information about a patient's changing physiological state. Electrophysiological biomarkers add this information and may help distinguish contacts or stimulation settings according to neural activity [15-17]. As the number of possible settings increases, algorithms can help clinicians narrow the combinations that require clinical testing [4, 7]. Adaptive DBS takes this a step further by using physiological or behavioural signals to modify stimulation as a patient's state changes over time [8, 9, 11].

Although technological advancements have occurred, evidential maturity varies significantly among the reviewed approaches. In Table 1, ADAPT-PD provides stronger prospective clinical evidence, with more participants enrolled across multiple centres and over months follow-up period [9, 10]. In comparison, the patient-specific gamma feasibility study involved only four participants [8], the gait-synchronised adaptive DBS trial enrolled five patients [11] and algorithm-assisted evidence is limited to a single-centre workflow implementation study and a single-patient feasibility investigation [4, 7].

Also, beta activity may not sufficiently provide a comprehensive biomarker for personalised DBS programming. It may remain stable during chronic recording, meaning that it might not fully represent a patient's motor state [16], and low-beta activity alone does not account for all variation in motor severity. A multicentre study showed improvement with motor impairment [17], while periodic and aperiodic activity showed different relationships with individual motor features [18]. A programming strategy based only on beta activity could therefore miss information relevant to particular symptoms or patients. Future biomarker-guided programming may need to consider several electrophysiological features and select them according to the clinical state being assessed.

It is difficult to compare between these four approaches directly. Since the supporting studies differ in their methods and clinical endpoints. Some investigate contact selection, others evaluate biomarker performance or adaptive stimulation and their follow-up periods and patient populations also vary. The current literature therefore does not establish that one approach is superior to another. More importantly, each approach provides different information that could contribute to an individual programming decision. The next step is to determine whether combining anatomical location, neural signals and algorithmic support produces better clinical outcomes than using these methods separately and whether such approaches remain practical outside specialist DBS centres.

Table 1. Summary of Key Studies Evaluating Personalised DBS Programming Approaches in PD

References	Strand	Design	n	Follow-up	Key finding
[1]	Anatomical (image-guided)	Prospective observational cohort	89	6 months	Image-guided asleep DBS achieved trial-comparable improvements in motor function, medication burden and quality of life following post-operative programming.
[14]	Anatomical / Connectomic	Retrospective patient-specific tractography study	69	Median 9.0 months (range 2.6-20.2)	Greater patient-specific STN-SMA/STN-GPi pathway modulation associated with improved motor outcomes, particularly tremor and rigidity.

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Table 1. (continued)

[13]	Anatomical	Prospective observational cohort	49	6 months	Reduced superior frontal cortex volume associated with poorer motor outcomes; Voxel-based morphometry showed no association with overall non-motor outcomes.
[15]	Biomarker	Longitudinal observational study (Home LFP recording)	33 (24,103 medication intakes)	Titration phase	Beta power decreased 30-140 mins after levodopa, particularly in low-beta frequencies, with a dose-response relationship.
[16]	Biomarker	Longitudinal observational study (chronic LFP recording)	33	Up to 44 months	Beta spatial distribution and centre frequency remained stable after postoperative stabilisation.
[17]	Biomarker	Multicentre cross-sectional study	119	/	Aperiodic broadband + low-beta + low-gamma features explained greater motor symptom variance than low-beta alone.
[4]	Algorithm-assisted	Quality-improvement workflow study	50	/	Standardised workflow supported consistent and reproducible adaptive programming across clinicians.
[7]	Algorithm-assisted	Single-patient feasibility study	1	435 days	Remotely optimised movement-responsive decoder improved hand speed, typing performance and dyskinesia vs. cDBS
[8]	Adaptive	Blinded randomised crossover feasibility trial	4	Short-term crossover	Patient-specific gamma feedback enabled aDBS associated with improved motor outcomes and quality of life vs. cDBS.
[9, 10]	Adaptive	International multicentre open-label non-randomised clinical trial	68	10 months	Dual-threshold aDBS met the performance goal in 91% of evaluated participants and reduced TEED without serious device-related adverse events.
[11]	Adaptive (symptom-specific)	Randomised feasibility crossover trial	5	Acute and multi-day home evaluation	Gait-phase-synchronised aDBS improved gait metrics and reduced fall frequency vs. cDBS.
[12]	Adaptive (symptom-specific)	Blinded randomised crossover study	8	Acute	60-Hz aDBS improved gait but remained inferior to 140-Hz cDBS for overall motor control.

Note. Studies are grouped into anatomically informed, biomarker-guided, algorithm-assisted and adaptive programming approaches and summarised by study design, sample size (n), follow-up and key findings.

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4. Future directions

Translating precision neuromodulation into routine clinical practice requires several priorities. Future DBS programming is unlikely to depend on beta activity alone. Although beta-band activity has become an important physiological marker, the evidence discussed above shows that it does not capture all aspects of motor impairment. Combining beta activity with other oscillatory, aperiodic and symptom-related signals may provide a more complete representation of an individual patient's motor state [17, 18]. The key question is whether these additional signals improve programming decisions and clinical outcomes, rather than simply improving predictions of symptom severity.

Another limitation of current programming is that clinicians usually make decisions based on relatively brief assessments, whereas patients' symptoms and medication effects fluctuate throughout the day. Future studies should evaluate not only whether computational approaches can identify effective settings, but whether they shorten programming sessions, reduce variability between programmers and maintain clinical outcomes when implemented across different DBS centres.

The future challenge is to determine whether combining these approaches improves the outcome and safety of DBS programming, compared with conventional programming. Neuroimaging can indicate where stimulation is delivered, chronic recordings can show how neural activity changes over time and wearable measures may provide information about symptoms during daily life. These data could potentially be used together to guide how stimulation is adjusted for an individual patient. However, greater technological complexity is only worthwhile if it produces a meaningful clinical advantage.

5. Conclusion

Personalised DBS programming can draw on anatomical targeting, neural biomarkers and computational methods, but the evidence supporting each approach is uneven. Anatomical and biomarker-based methods are already clinically informative, whereas computational programming still needs stronger validation. Adaptive DBS has also produced encouraging longer-term results, although benefit varies by patient and symptom. In the future, its clinical value will depend on whether integrating these approaches improves effectiveness and safety, compared to conventional programming.

References

[1] Roldán, P., Mosteiro, A., Rumià Arboix, J., Asín, D., Sánchez-Gómez, A., Valldeoriola, F., et al. (2025). Subthalamic deep brain stimulation under general anaesthesia for Parkinson's disease: Institutional experience and outcomes. *Stereotactic and Functional Neurosurgery, 103*(2), 102–110.

[2] Emura, T., Tani, N., Hosomi, K., Kimoto, Y., Matsuhashi, T., Fujinaga, T., et al. (2026). Adaptive deep brain stimulation benefits: Younger patients with persistent wearing-off symptoms. *Movement Disorders Clinical Practice, 13*(2), 452–463.

[3] Moraes de Falcone, A. C., França, C., Guimarães, T. G., & Cury, R. G. (2025). The electric brain: Approach to suboptimal DBS parameters. *Clinical Parkinsonism & Related Disorders, 14*, 100418.

[4] Anis, S., Clark, P. A., Shaffer, S., Hartwig, E., Moser, L., Scott, J., et al. (2026). Introducing a workflow algorithm for adaptive DBS programming in Parkinson's disease. *Parkinsonism & Related Disorders, 144*, 108199.

[5] de Neeling, M. G. J., Geraats, S., Swinnen, B. E. K. S., Stam, M. J., de Bie, R. M. A., van Wijk, B. C. M., et al. (2026). Temporal dynamics of beta power related to the stun effect in Parkinson's disease patients after deep brain stimulation surgery. *Movement Disorders, 41*(1), 222–227.

[6] Feldmann, L. K., Soriano, D. C., Habets, J., D'Onofrio, V., Kaplan, J., Mathiopoulou, V., et al. (2025). Electrophysiological changes in the acute phase after deep brain stimulation surgery. *Brain Stimulation, 18*(5), 1579–1586.

DOI: 10.54254/2753-8818/2026.HZ37585

[7] Dixon, T. C., Strandquist, G., Zeng, A., Frączek, T., Bechtold, R., Lawrence, D., et al. (2026). Movement-responsive deep brain stimulation for Parkinson's disease using a remotely optimized neural decoder. *Nature Biomedical Engineering, 10*(1), 110–124.

[8] Oehrn, C. R., Cernera, S., Hammer, L. H., Shcherbakova, M., Yao, J., Hahn, A., et al. (2024). Chronic adaptive deep brain stimulation versus conventional stimulation in Parkinson's disease: A blinded randomized feasibility trial. *Nature Medicine, 30*(11), 3345–3356.

[9] Bronte-Stewart, H. M., Beudel, M., Ostrem, J. L., Little, S., Almeida, L., Ramirez-Zamora, A., et al. (2025). Long-term personalized adaptive deep brain stimulation in Parkinson disease: A nonrandomized clinical trial. *JAMA Neurology, 82*(11), 1171–1180.

[10] Stanslaski, S., Summers, R. L. S., Tonder, L., Tan, Y., Case, M., Raike, R. S., et al. (2024). Sensing data and methodology from the Adaptive DBS Algorithm for Personalized Therapy in Parkinson's Disease (ADAPT-PD) clinical trial. *npj Parkinson's Disease, 10*(1), 174.

[11] Louie, K. H., Balakid, J. P., Bath, J. E., Song, S., Fekri Azgomi, H., Marks, J. H., et al. (2026). Adaptive deep brain stimulation for dynamic gait control in Parkinson's disease: A randomized feasibility trial. *Nature Medicine*. https://doi.org/10.1038/s41591-026-04434-2

[12] Negi, A. S., Cui, C., Wilkins, K. B., Lambert, E. F., Melbourne, J. A., Petrucci, M. N., et al. (n. d.). Low-frequency adaptive deep brain stimulation for Parkinson's disease.

[13] Loehrer, P. A., Schumacher, W., Jost, S. T., Silverdale, M., Petry-Schmelzer, J. N., Sauerbier, A., et al. (2024). No evidence for an association of voxel-based morphometry with short-term non-motor outcomes in deep brain stimulation for Parkinson's disease. *npj Parkinson's Disease, 10*(1), 91.

[14] Loução, R., Kocher, M., Brandt, G. A., Petry-Schmelzer, J. N., Barbe, M., Dafsari, H., Mana, J., Jech, R., Wirths, J., Visser-Vandewalle, V., Andrade, P., NEURIPIDES Study Group, Baqapuri, H., Luehrs, M., Linden, D. E. J., Santyr, B., Lozano, A., Bongioanni, A., Jarraya, B., & Cukur, T. (2025). Structural connectivity of the basal ganglia from patient-individual tractography is key for understanding the effects of deep brain stimulation in Parkinson's disease. *Stereotactic and Functional Neurosurgery, 103*(4), 279–294. https://doi.org/10.1159/000546716

[15] de Neeling, M. G. J., Keulen, B. J., Stam, M. J., Hubers, D., de Bie, R. M. A., van Wijk, B. C. M., Schuurman, P. R., Buijink, A. W. G., & Beudel, M. (2025). Beta power response after levodopa intake in Parkinson's disease patients with chronic sensing-enabled deep brain stimulation. *Annals of Neurology, 98*(6), 1352–1363. https://doi.org/10.1002/ana.78025

[16] Behnke, J. K., Peach, R. L., Habets, J. G. V., Busch, J. L., Kaplan, J., Roediger, J., Mathiopoulou, V., Feldmann, L. K., Gerster, M., Vivien, J., Schneider, G. H., Faust, K., Krause, P., & Kühn, A. A. (2025). Long-term stability of spatial distribution and peak dynamics of subthalamic beta power in Parkinson's disease patients. *Movement Disorders, 40*(6), 1070–1084. https://doi.org/10.1002/mds.30169

[17] Gerster, M., Waterstraat, G., Binns, T. S., Darcy, N., Wiest, C., Köhler, R. M., Vanhoecke, J., West, T. O., Sure, M., Todorov, D., Radzinski, L., Habets, J., Busch, J. L., Feldmann, L. K., Krause, P., Faust, K., Schneider, G. H., Ashkan, K., Pereira, E., ... Nikulin, V. (2025). Beyond beta rhythms: Subthalamic aperiodic broadband power scales with Parkinson's disease severity—A cross-sectional multicentre study. *EBioMedicine, 122*, 105988. https://doi.org/10.1016/j.ebiom.2025.105988

[18] Sayfulina, K., Filyushkina, V., Usova, S., Gamaleya, A., Tomskiy, A., Belova, E., & Sedov, A. (2025). Periodic and aperiodic components of subthalamic nucleus activity reflect different aspects of motor impairment in Parkinson's disease. *European Journal of Neuroscience, 61*(1), e16648. https://doi.org/10.1111/ejn.16648