scientific reports

Check for updates

OPEN

Wearable inertial device for monitoring Parkinson's disease symptoms: a pilot study in a controlled environment

Margaux Poleur[1,8]✉, Teresa Gidaro[2,8], Stéphanie Delstanche[1], Jean-Marc Gurruchaga[3], Alexis Tricot[4], Léopold Bancel[4], Stéphane Palfi[3], Laurent Servais[1,5,8] & Bertrand Degos[6,7,8]

Clinical assessments for Parkinson's disease depend on clinician-administered scales, which have limitations in sensitivity and real-world applicability. Wearable inertial sensors offer a promising approach for objective and continuous monitoring of PD motor symptoms. This study aimed to evaluate the feasibility and accuracy of a magneto-inertial wearable device in detecting key PD motor manifestations—tremor, akinesia, and dyskinesia—on an individual movement basis. Ten PD patients undergoing pre-surgical evaluation for deep brain stimulation were included in a pilot multicentric study. Participants performed a Levodopa challenge test while wearing an inertial measurement unit on the most affected wrist and ankle. MDS-UPDRS Part III and video recordings were obtained to compare sensor performance to expert evaluations. Algorithms analyzed acceleration and angular velocity data to detect tremor, akinesia and dyskinesia. The sensor demonstrated high sensitivity (100%) and specificity (≥ 93%) for tremor and akinesia detection, with an overall accuracy exceeding 94%. Performance metrics were less promising for dyskinesia detection. Levodopa significantly reduced tremor ($p = 0.0247$) and increased dyskinesia ($p = 0.0169$), confirming sensor responsiveness to pharmacological effects. Magneto-inertial wearable device showed promising accuracy for the objective assessment of PD motor symptoms in a controlled environment. These findings support further validation in real-life conditions.

Keywords Wireless sensor, Parkinson's disease, Levodopa-induced dyskinesia, Akinesia, Tremor

Parkinson's disease (PD) is a chronic neurodegenerative disease primarily affecting dopaminergic neurons of the Substantia nigra pars compacta. It is the second most common neurodegenerative disease after Alzheimer's disease[1]. Clinically, PD is defined by bradykinesia combined with either rest tremor or rigidity[2]. Diagnosis remains challenging, especially at an early stage, as no definitive biomarkers exist[3]. A good and sustained response to Levodopa (L-Dopa) may help support the diagnosis[4].

Current treatments aim to compensate for dopamine loss using L-Dopa or dopamine receptor agonists[5]. However, none of these drugs prevent neuronal degeneration and disease progression. As the disease progresses, patients ineluctably develop L-Dopa-induced motor complications, such as motor fluctuation and dyskinesia partly linked to the iterative administration of L-Dopa and its pulsatile effects on dopaminergic receptors[6].

Clinical assessment of PD typically relies on scales such as the Movement Disorder Society-Unified Parkinson's Disease Rating Scale (MDS-UPDRS)[7]. However, these assessments have psychometric limitations, including

[1]University Department of Neurology, Citadelle Hospital, Liège, Belgium. [2]Institute of Myology, GH La Pitié-Salpêtrière, Paris, France. [3]Groupe de recherche clinique translationnelle OPTIMA, Institut Mondor de Recherche Biomédical, Henri Mondor Academic Hospital, Assistance Publique Hôpitaux de Paris, Université Paris Est Créteil, INSERM Laboratory U955 of Translational NeuroPsychiatry, Institut Mondor de Recherche Biomédicale, Créteil, France. [4]SYSNAV, Vernon, France. [5]Department of Paediatrics, MDUK Oxford Neuromuscular Centre, University of Oxford, Oxford, UK. [6]Neurology Department, Réseau NS-PARK/FCRIN, Avicenne Hospital, APHP, Universitaires de Paris-Seine Saint Denis (HUPSSD), Sorbonne Paris Nord, Bobigny, Hôpitaux, France. [7]Dynamics and Pathophysiology of Neuronal Networks Team, Center for Interdisciplinary Research in Biology, UMR7241/INSERM U1050, Collège de France, CNRS, Université PSL, 75005 Paris, France. [8]Margaux Poleur, Teresa Gidaro, Laurent Servais and Bertrand Degos have contributed equally to this work. ✉email: margaux.poleur@citadelle.be

lack of inter-rater reliability[8], poor sensitivity to subtle changes, particularly in early stages[9], and dependence on hospital-based, episodic evaluations that fail to capture daily fluctuations[6].

With recent advances in the therapeutic field, an increasing number of promising trials are emerging for PD[10]. As therapeutic decisions and drug development rely on accurate, objective, and representative evaluations of patient's condition, the ability to continuously monitor a patient's activity and symptoms remains a significant challenge.

The emergence of wearable sensor technology offers new opportunities for continuous, objective symptom monitoring[11]. Accelerometers, gyroscopes, and their combination, known as inertial measurement units (IMUs), are the most widely used wearable sensors for assessing symptoms and motor complications in neurological diseases[12]. They have already been used for various purpose in the neurological field[13,14]. Objective measurement of various PD motor manifestations, including gait impairments, bradykinesia, tremors, dyskinesia and motor fluctuations, outside clinical settings using wearable inertial devices has proven feasible, achieving accuracies exceeding 90%[13]. In recent year, the use of wearable sensors to objectively measure PD motor manifestations has known a growing interest, as highlighted in recent comprehensive reviews[15]. Some studies have focused on postural and gait disturbances, while others investigated PD symptoms and their fluctuations aiming to detect bradykinesia, tremor, dyskinesia, and ON/OFF state episodes in real-life conditions[13]. Only few devices have shown their ability to assess all aspects of the disease[16]. Machine learning approach for identifying PD motor manifestations[17] has shown remarkable potential in detecting patients with prodromal PD[18]. While this could be a valuable tool for selecting participants in clinical trials for neuroprotective treatments, it remains uncertain if these models could be generalized outside the studied population, raising concerns about their clinical applicability. Additionally, most existing devices and algorithms have yet to prove their efficiency in real-life conditions[17], likely due to the lack of validated reference outcome for comparison.

Recently, the European Medicines Agency (EMA) qualified the Stride Velocity 95th Centile (SV95C) as an appropriate primary endpoint for clinical trial in Duchenne Muscular Dystrophy (DMD)[19]. This regulatory qualification highlights the potential of wearable-based digital measures in clinical research. The underlying magneto-inertial technology supports an increase of statistical power for clinical trials not only for DMD[19], but also for Spinal Muscular Atrophy[20] and Facio-scapulo-humeral dystrophy[21]. While these neuromuscular diseases share characteristics such as muscle weakness, they do not involve movement disorders.

The aim of this study was to evaluate whether movement reconstruction using a wearable inertial device can accurately identify key PD motor manifestations, including tremor, akinesia, and dyskinesia, on an individual movement basis. This work is a first step toward real-world detection of PD motor symptoms, this study lays the foundation for future research on continuous, real-life symptom monitoring.

Results

Study population

Baseline demographic and clinical characteristics of the study population are presented in Table 1. A total of 10 PD patients were included. All patients were treated with L-Dopa with a median LEDD of 1139.3 mg/day. All patients reported dyskinesia at inclusion. Among them 90% reported peak dose dyskinesia. No participants reported any adverse event related to the study.

Demographic characteristics	
Sample size	10
Gender : number of female (%)	4 (40)
Age at inclusion (years)	55.2 ± 9.8 [40–69]
Disease Duration (years)	10.7 ± 3.6[6–16]
Treatment	
Number of intake/day	5.2 ± 1.4[2–7]
L-Dopa equivalent dose* (mg)	1302.3 ± 295.0 [850–1722.5]
Clinical characteristics	
MDS-UPDRS	
Total score (/260)	60 ± 15.9 [42–90]
Part 1 (/52)	12.3 ± 4.8[7–21]
Part 2 (/52)	14.5 ± 3.9[10–24]
Part 3 (/132)	23.8 ± 11.7 [7–43]
Part 4 (/24)	9.4 ± 3.4[2–14]
RDRS score	
Total score	1.3 ± 1.1 [0–3]

Table 1. Demographic and clinical characteristics of participants. Data are expressed in mean ± SD [Min–Max]. *Calculated as described by Schade et al.[32]Abbreviations: MDS-UPDRS = Movement Disorder Society—Unified Parkison's Disease Rating Scale; RDRS = Rush Dyskinesia Rating Scale; SD = Standard Deviation.

Sensor algorithm performance

During the LCT, the algorithm of PD motor manifestations detection by the sensor showed an overall good result. The sensor detects an improvement of PD motor manifestations during the L-Dopa test as shown by the reduction of tremor and akinesia after L-Dopa administration. Precision, sensitivity, specificity and accuracy are shown in Table 2. The performance of the algorithm for the detection of PD motor manifestations is illustrated in Fig. 1B.

L-Dopa challenge test (LCT) results

L-Dopa test was positive in all 10 patients with PD. Mean MDS-UPDRS motor scores were 48.7 ± 11.5 in OFF state and 37.7 ± 14.1, 25.7 ± 5.5, 18.6 ± 5.0 and 18.4 ± 5.5 at 15, 30, 60 and 90 min after acute L-Dopa intake, respectively. After 90 min, a 62.1 ± 18.7% (− 30 points) mean reduction in MDS-UPDRS motor score has been achieved. The evolution of PD motor manifestations detected by sensors using 15-min windows is shown for each patient in Fig. 1A. Based on the Mann–Whitney U test results, we observed a significant difference between the proportion of time with dyskinesia ($p = 0.0169$) and tremor recorded at the wrist ($p = 0.0247$) before (15 min before) and after (between 45 and 60 min after) L-Dopa intake, but no significant difference for the tremor recorded at the ankle ($p = 0.5415$) and akinesia recorded at wrist and ankle ($p = 0.3173$ and $p = 0.0798$, respectively). Figure 1**.** Sensor detection during LCT for each PD motor manifestation for all patients per 15-min windows (A). Sensor performance during LCT for each PD motor manifestation for all patients (B). In green, sensor detection of events and in orange the false negatives. The intake of L-Dopa is marked with a dotted black line.

Discussion

Our pilot study suggested that a magneto-inertial wearable sensor could accurately detect akinesia, tremor and dyskinesia; the main parkinsonian symptoms observed in PD patients. Recent technological advances have made continuous monitoring of neurological manifestations possible, with growing interest in wearable devices for real-life data collection[22]. The COVID-19 pandemic has further highlighted the need for reliable, validated tools to assess patients remotely, both in clinical trials and routine practice.

Previous studies have suggested that remote monitoring can help identify patients who may be candidates for advanced therapies[18] or improved management of PD[23]. However, these data were often based on global indices derived from spectral analysis of movement. Our results demonstrate that individual movements associated with PD can be accurately identified in a controlled environment using a magneto-inertial device. The lower agreement with physicians observed for dyskinesia reflects the challenge of distinguishing these movements from normal ones with similar trajectories.

Importantly, our algorithms were validated against a set of synchronized videos independently and blindly analyzed by two neurologists, providing for each symptom negative and positive predictive values. While video-based validation has been applied for freezing of gait[24] and balance assessment[25], it has not yet been applied to the detection of individual PD movements. This constitutes the key novelty of our study and provides a crucial methodological foundation for future large-scale validation efforts.

These preliminary findings provide a strong rationale for further investigations in a larger and more diverse population, particularly in unsupervised settings where symptom monitoring can more reflect real-life conditions. To assess the clinical relevance of wearable-based symptom detection, future studies should establish a robust reference for symptom occurrence, as traditional symptom diaries remain unreliable due to subjective reporting and recall bias[26]. A key challenge will be identifying an objective and accurate "ground truth" for symptoms, possibly through a combination of clinician assessments and real-time patient feedback. Expanding data collection to a longitudinal, real-world framework will also allow better tracking of symptom fluctuations, disease progression, and treatment responses over time, refining algorithms and improving their accuracy.

Our algorithms were designed using a standard methodology based on movement disorder and PD knowledge rather than machine learning. While simple algorithms effectively detect clear manifestations such as tremor, they are less reliable for complex movements like dyskinesia. Distinguishing akinesia from inactivity in real life is also expected to be challenging. A machine learning approach could improve accuracy but would require extensive data collection[27]. The main drawback of such an approach is the lack of interpretability for clinicians.

	Dyskinesia	Wrist tremor	Ankle tremor	Wirst akinesia	Ankle akinesia
True positive (%)	2.9	19.0	11.0	3.2	17.0
True negative (%)	93.0	78.0	83.0	97	78.0
False positive (%)	1.5	3.0	5.8	0.0	4.8
False negative (%)	2.2	0.0	0.0	0.0	0.0
Precision (%)	65.9	86.4	65.5	100.0	77.9
Sensitivity (%)	56.9	100.0	100.0	100.0	100.0
Specificity (%)	98.4	96.3	93.5	100.0	94.2
Accuracy (%)	96.3	97.0	94.2	100.0	95.2

Table 2. Precision, Sensitivity, Specificity, Accuracy of the wearable inertial device or detection of each PD symptom.

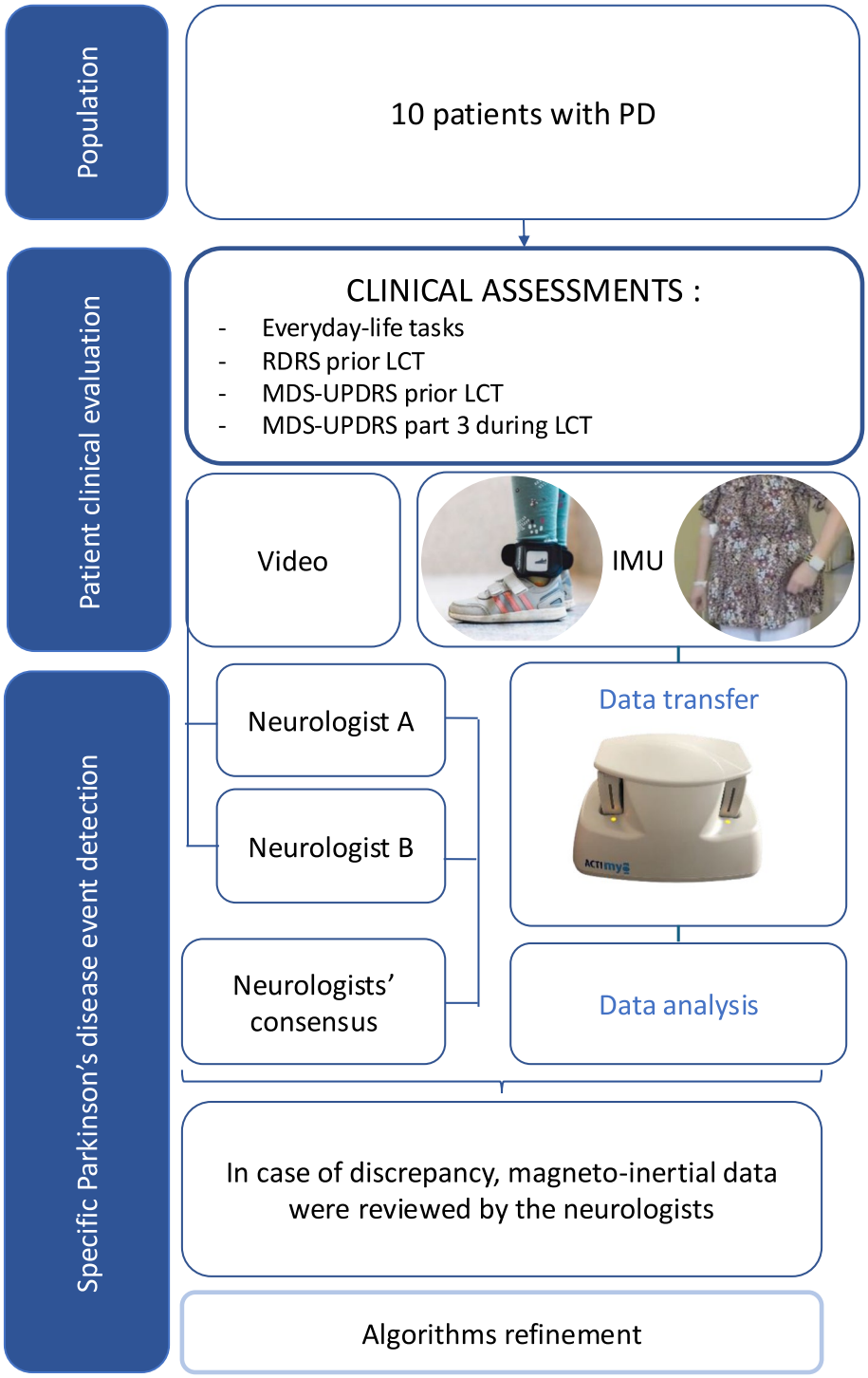

Fig. 1. Flow chart and method for specific Parkinson's disease events detection. Abbreviations: MDS-UPDRS = Movement Disorder Society—Unified Parkison's Disease Rating Scale; LCT = L-Dopa challenge test; RDRS = Rush Dyskinesia Rating Scale; IMU = inertial measurement unit.

Despite these promising results, certain limitations exist at this stage. This study was primarily designed as a proof of concept in a controlled setting, and participants were DBS candidates with a complex PD phenotype, including L-Dopa-induced motor complications. This selection bias may have complicated the distinction between different motor events. In addition, the size of the studied population was very small and highly selected, which limits the generalizability of our findings. Moreover, as the algorithms were initially developed and tested on a prior unpublished cohort of PD patients and subsequently refined during the present study, there is a potential risk of an overestimation of algorithm accuracy. Furthermore, the current algorithm for dyskinesia detection could not capture episodes occurring simultaneously with walking or tremor due to

technical limitations. We acknowledge that this may lead to underestimation of dyskinesia, particularly during walking, and that further engineering work will be required to address this limitation.

Consequently, the results should be interpreted with caution and viewed as exploratory and hypothesis-generating, warranting validation in larger and more diverse cohorts to ensure external validity and reproducibility. The next step will also be to assess PD motor manifestations in real-life conditions, the ultimate goal of continuous digital assessment. Future studies should refine algorithms for detecting dyskinesia and akinesia. Since the device used here has already been well-validated for gait impairment detection in DMD[19,28] including comparison with controls and is currently being validated in other neurological disorders[21,29,30], future research should aim to integrate gait and Parkinson's disease symptom assessment in real-life environments.

Beyond methodological considerations, regulatory qualification remains a critical challenge. Except for the Parkinson's Kinetigraph, which received FDA clearance in September 2016, wearable devices and the variables they generate lack qualification from regulatory bodies such as the EMA and FDA. Despite the promise of wearable devices for remote monitoring, technical issues, poorly validated outcome measures and regulatory limitations remain significant barriers. A few years ago, the Movement Disorder Society Task Force on technology published concrete steps to facilitate adoption of mobile health technologies in clinical practice[31]. The recent qualification of a digital outcome, the SV95C, in Duchenne Muscular Dystrophy and its use as a primary endpoint in several pivotal trials open the perspective of further qualification of digital outcome, and Parkinson's disease is certainly an area of considerable unmet need.

Achieving regulatory qualification will require large, prospective, multi-sites real-world studies to establish usability, validity, reliability, clinical meaningfulness and sensitivity to change. Massive data acquisition, algorithm refinement, and psychometric validation of digital outcomes will be necessary. This can only be achieved through strong collaboration between patients, physicians, engineers, regulators, and the pharma industry. This study represents an initial step toward real-life monitoring.

Method

Study design

This pilot multicentric study on patients with PD (Pre-QuantiPark), was sponsored by the Institute of Myology at La Pitié-Salpêtrière University Hospital. The 12-months study was conducted at two French investigational sites of the APHP: La Pitié-Salpêtrière Hospital, Paris, and Henri Mondor Hospital, Créteil.

Standard protocol approvals, registration and patient consents

The study protocol was approved by the local ethics committee (CPP-Ile de France VI – Groupe Hospitalier La Pitié-Salpêtrière, ID RCB: 2016-A00121-50; ClinicalTrials.gov Identifier: NCT02785978). All patients gave written informed consent prior to participation in this exploratory study. The study was conducted in accordance with relevant guidelines and regulations, including the Declaration of Helsinki and Good Clinical Practice guidelines.

Participants

Ten patients with PD were enrolled. Participants were recruited among PD patients undergoing pre-surgical evaluation for deep brain stimulation (DBS). The inclusion criteria were: subjects aged ≥ 18 years old with a clinical diagnosis of idiopathic PD (MDS criteria); stable parkinsonian medication regimen including L-Dopa for at least 4 weeks; experience of motor fluctuations and dyskinesia, and a normal MoCA test (Montreal Cognitive Assessment) ≥ 26 (out of 30). The exclusion criteria were: previous surgery for the treatment of PD, patients treated with apomorphine or L-Dopa pump, patients treated with DBS, patients with drug-induced parkinsonism, vascular parkinsonism or Parkinson plus syndromes (such as multiple system atrophy, progressive supranuclear palsy, and cortico-basal syndrome), as well as pregnant and breastfeeding women.

Data collection

Demographic and clinical data, along with treatment and medical histories, were collected before the L-dopa acute challenge, which was scheduled during a planned hospitalization as part of routine care.

Controlled environment tests, including a series of everyday-life tasks, Movement Disorder Society–Unified Parkinson's Disease Rating Scale (MDS-UPDRS), and Rush Dyskinesia Rating Scale (RDRS) assessments, were performed before the L-dopa challenge test (LCT). All test sessions, including the LCT evaluation periods, were videotaped and simultaneously recorded using a wearable IMU device (Fig. 2).

LCT was was conducted by physicians and/or nurses experienced in movement disorders. The trial involved a single-dose L-dopa challenge. Prior to the visit, patients discontinued all dopaminergic medications for at least 12 h (over 18 h for dopaminergic agonists and 24 h for long-acting agonists) to induce a practically defined "OFF medication" state. The LCT was performed early in the morning on an empty stomach. A L-dopa equivalent dose (LEDD) corresponding to the patient's first morning dose, increased by 50% or 100 mg, was administered.

Clinical evaluations were conducted before any drug intake ("OFF state") and then every 15 min for 90 min following the L-dopa administration. These evaluations included the MDS-UPDRS Part III (motor section).

Controlled environment tests were recorded with the ActiMyo magneto-inertial device (SYSNAV, Vernon, France) as depicted in Fig. 2. Simultaneously, video recordings were captured using a tripod-mounted camera. Recording started as soon as the sensors were removed from the docking station and ended at the conclusion of the test. A synchronization signal was applied at the start of the session to align the video and wearable device data.

Videos were independently and blindly analyzed by two neurologists (TG and BD), who assessed each movement detected by the wearable device and calculated corresponding negative and positive predictive values. Their assessments were then compared, and any discrepancies were resolved through qualitative consensus.

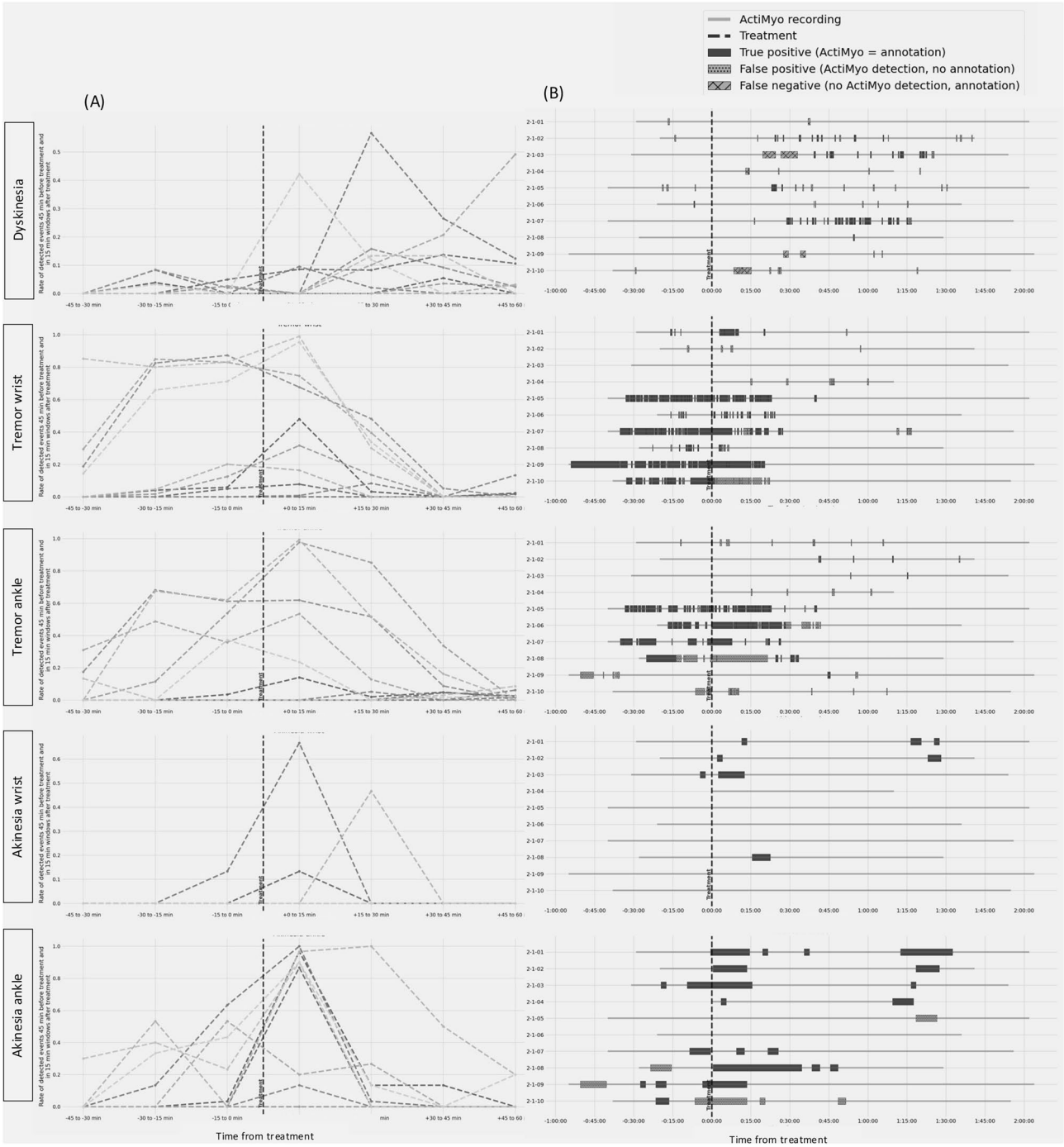

Fig. 2. Sensor detection during LCT for each PD motor manifestation for all patients per 15-min windows (**A**). Sensor performance during LCT for each PD motor manifestation for all patients (**B**). In green, sensor detection of events and in orange the false negatives. The intake of L-Dopa is marked with a dotted black line.

Drug-related dyskinesia (such as OFF-period dystonia, diphasic or peak-dose dyskinesia) has been evaluated by an expert in movement disorder (BD) and reported on the dedicated worksheet. Adverse events have been monitored.

Data proceeding and extracted parameters

Algorithms for PD manifestations detection

The device includes two sensors attached to the wrist and ankle of the side most affected by PD. Each sensor contains high precision triaxial accelerometers, gyrometers, magnetometers, a temperature sensor, and a barometer. These sensors recorded passively upper and lower limbs linear acceleration and angular velocity in 3-dimensions. Data are recorded at a sampling frequency of 130.69 Hz. The sensors are encased in a lightweight

plastic housing (43 × 37 × 16 mm; 28 g) and are individually factory-calibrated to ensure accuracy across a range of temperature, pressure, and dynamic conditions. These measurements have been used to develop an automatic detection of parkinsonian manifestations. These manifestations are tremors, akinesia and dyskinesia. The data were analyzed using MATLAB and Python.

Tremors are characterized by repeated, regular and oscillatory movements at the frequencies between 4 and 7 Hz. The frequency spectrum is computed using a fast Fourier transform on a sliding window of 8 s. To demonstrate the presence of a tremor, a peak should be detected between 4 and 7 Hz in one of the axes of the accelerometer and in one of the axes of the gyroscope. In addition, this peak must be of large magnitude (at least twice as large) compared to the signal measured between 0.8 and 2.5 Hz.

Akinesia is detected when the sensor could only detect very few movements. The absence of movement is identified by computing the standard deviation of the norm of the gyroscope and the accelerometer on a sliding 5-min window. If both standard deviations are between 0.2 and 4 degrees per second in the window, we assumed that the patient presented akinesia during this time window.

Dyskinesia was detected in the ankle and defined as prolonged, high-amplitude movement which is not associated with walking or tremor. The frequency spectrums of the norm of the accelerometer and the gyroscope were calculated on 8 s sliding window. A candidate dyskinesia is considered in the window if we observe a peak in the lower frequency band (0.8–2.5 Hz). Windows containing tremor activity or physiologically normal leg movements were discarded, and the remaining events were classified as dyskinesia. The movement intensity threshold was set to exclude most voluntary actions, thereby improving the discrimination between involuntary dyskinetic and intentional movements.

Algorithms for event detection are based on prior validated signal processing approaches developed for motion reconstruction in neuromuscular disorders. These algorithms were initially developed and tested on a prior unpublished cohort of PD patients and were refined and optimized during the present study, as planned in the study protocol.

As illustrated in Fig. 2, algorithms for event detection and analysis have been compared to the simultaneous and independent evaluation of the set of videos, by two neurologists (TG and BD).

Statistical analysis

The accuracy of the proposed algorithms was tested by using the confusion matrix comparing data from the algorithms and the observations of neurologists. This confusion matrix includes the following details: True positive (TP) for correctly predicted event values; False negative (FN) for incorrectly predicted no-event values; True negative (TN) for correctly predicted no-event values and False positive (FP): for incorrectly predicted event values. Metrics for performance were computed for the duration of time spent on each symptom. Sensitivity, specificity, precision and accuracy are expressed, as described below, in terms of TP, TN, FN and FP.

- Precision (or positive predictive value) = TP/(TP + FP) = (Number of correct positive assessments)/(Number of positive assessments)
- Sensitivity = TP/(TP + FN) = (Number of true positive assessment)/(Number of all positive assessment)
- Specificity = TN/(TN + FP) = (Number of true negative assessment)/(Number of all negative assessment)
- Accuracy = (TN + TP)/(TN + TP + FN + FP) = (Number of correct assessments)/Number of all assessments)

We used Mann–Whitney U test to compare differences between PD motor manifestations detected by sensors before and after LCT.

Data availability

The data supporting the findings of this study may be made available for academic, non-commercial purposes upon request to the corresponding author. Access may be granted after review and approval of a written research proposal compatible with patient consents & legitimate use, and contingent upon the signing of a data access agreement.

Received: 14 August 2025; Accepted: 13 November 2025
Published online: 27 November 2025

References

1. Erkkinen, M. G., Kim, M.-O. & Geschwind, M. D. Clinical Neurology and Epidemiology of the Major Neurodegenerative Diseases. *Cold Spring Harb Perspect Biol* **10**, a033118 (2018).
2. Postuma, R. B. et al. MDS clinical diagnostic criteria for Parkinson's disease. *Mov. Disord.* **30**, 1591–1601 (2015).
3. Tolosa, E., Wenning, G. & Poewe, W. The diagnosis of Parkinson's disease. *Lancet Neurol.* **5**, 75–86 (2006).
4. D'Costa, D. F., Sheehan, L. J., Phillips, P. A. & Moore-Smith, B. The levodopa test in Parkinson's disease. *Age Ageing* **24**, 210–212 (1995).
5. Armstrong, M. J. & Okun, M. S. Diagnosis and Treatment of Parkinson Disease: A Review. *JAMA* **323**, 548–560 (2020).
6. Ahlskog, J. E. & Muenter, M. D. Frequency of levodopa-related dyskinesias and motor fluctuations as estimated from the cumulative literature. *Mov. Disord.* **16**, 448–458 (2001).
7. Goetz, C. G. et al. Movement Disorder Society-Sponsored Revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS): Scale presentation and clinimetric testing results. *Mov. Disord.* **23**, 2129–2170 (2008).
8. Marras, C. & Lang, A. E. Outcome measures for clinical trials in Parkinson's disease: Achievements and shortcomings. *Expert Rev Neurother* **4**, 985–993 (2004).
9. Regnault, A. et al. Does the MDS-UPDRS provide the precision to assess progression in early Parkinson's disease? Learnings from the Parkinson's progression marker initiative cohort. *J Neurol* **266**, 1927–1936 (2019).
10. McFarthing, K. et al. Parkinson's Disease Drug Therapies in the Clinical Trial Pipeline: 2023 Update. *J Parkinsons Dis* **13**, 427–439 (2023).

11. Piwek, L., Ellis, D. A., Andrews, S. & Joinson, A. The Rise of Consumer Health Wearables: Promises and Barriers. *PLoS Med* **13**, e1001953 (2016).
12. Yang, C.-C. & Hsu, Y.-L. A review of accelerometry-based wearable motion detectors for physical activity monitoring. *Sensors* **10**, 7772–7788 (2010).
13. Sica, M. et al. Continuous home monitoring of Parkinson's disease using inertial sensors: A systematic review. *PLoS ONE* **16**, e0246528 (2021).
14. Zhao, H. et al. Wearable sensors and features for diagnosis of neurodegenerative diseases: A systematic review. *Digit Health* **9**, 205520762311735 (2023).
15. Moreau, C. et al. Overview on wearable sensors for the management of Parkinson's disease. *NPJ Parkinsons Dis* **9**, 153 (2023).
16. Rodríguez-Martín, D. et al. A New Paradigm in Parkinson's Disease Evaluation With Wearable Medical Devices: A Review of STAT-ONTM. *Front Neurol* **13**, 912343 (2022).
17. Del Din, S., Kirk, C., Yarnall, A. J., Rochester, L. & Hausdorff, J. M. Body-Worn Sensors for Remote Monitoring of Parkinson's Disease Motor Symptoms: Vision, State of the Art, and Challenges Ahead. *J Parkinsons Dis* **11**, S35–S47 (2021).
18. Kadirvelu, B. et al. A wearable motion capture suit and machine learning predict disease progression in Friedreich's ataxia. *Nat Med* **29**, 86–94 (2023).
19. Servais, L. et al. First regulatory qualification of a digital primary endpoint to measure treatment efficacy in DMD. *Nat Med* **29**, 2391–2392 (2023).
20. Annoussamy, M. et al. Natural history of Type 2 and 3 spinal muscular atrophy: 2-year NatHis-SMA study. *Ann Clin Transl Neurol* **8**, 359–373 (2021).
21. Gidaro, T. et al. Home-based gait analysis as an exploratory endpoint during a multicenter phase 1 trial in limb girdle muscular dystrophy type R2 and facioscapulohumeral muscular dystrophy. *Muscle Nerve* **65**, 237–242 (2022).
22. Lu, L. et al. Wearable Health Devices in Health Care: Narrative Systematic Review. *JMIR Mhealth Uhealth* **8**, e18907 (2020).
23. Woodrow, H. et al. A blinded, controlled trial of objective measurement in Parkinson's disease. *NPJ Parkinsons Dis* **6**, 35 (2020).
24. Capecci, M., Pepa, L., Verdini, F. & Ceravolo, M. G. A smartphone-based architecture to detect and quantify freezing of gait in Parkinson's disease. *Gait Posture* **50**, 28–33 (2016).
25. Stack, E. et al. Identifying balance impairments in people with Parkinson's disease using video and wearable sensors. *Gait Posture* **62**, 321–326 (2018).
26. Papapetropoulos, S. S. Patient diaries as a clinical endpoint in Parkinson's disease clinical trials. *CNS Neurosci Ther* **18**, 380–387 (2011).
27. Schalkamp, A.-K., Peall, K. J., Harrison, N. A. & Sandor, C. Wearable movement-tracking data identify Parkinson's disease years before clinical diagnosis. *Nat Med* **29**, 2048–2056 (2023).
28. European Medicines Agency. EMA qualification opinion for SV95C as primary endpoint in studies in ambulatory Duchenne Muscular Dystrophy studies. https://www.ema.europa.eu/en/documents/scientific-guideline/qualification-opinion-stride-velocity-95th-centile-primary-endpoint-studies-ambulatory-duchenne-muscular-dystrophy-studies_en.pdf.
29. Poleur, M. et al. Digital Outcome Measure Assessing the Motor Function of Patients with Amyotrophic Lateral Sclerosis. *Neurology* **102**, 5214 (2024).
30. Poleur, M. et al. Reliability and External Validity of Digital Passive Gait Tracking in MS. *Neurology* **102**, 5890 (2024).
31. Espay, A. J. et al. A roadmap for implementation of patient-centered digital outcome measures in Parkinson's disease obtained using mobile health technologies. *Mov Disord* **34**, 657–663 (2019).
32. Schade, S., Mollenhauer, B. & Trenkwalder, C. Levodopa Equivalent Dose Conversion Factors: An Updated Proposal Including Opicapone and Safinamide. *Mov Disord Clin Pract* **7**, 343–345 (2020).

Acknowledgements

We would like to thank Mélanie Villeret for her contribution to this study.

Author contributions

TG, LS, and BD conceived and designed the study. TG, SD, JMG, SP, and BD were responsible for data acquisition. MP, AT, and LB conducted the data analysis, while MP, AT, LB, LS, and DB contributed to data interpretation. AT and LB designed the algorithms used for sensor data processing. MP drafted the manuscript. All authors reviewed the manuscript for important intellectual content and approved the final version.

All authors reviewed the manuscript for important intellectual content and approved the final version.

Declarations

Competing interests

The authors declare no competing interests.

Additional information

Correspondence and requests for materials should be addressed to M.P.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.