Parkinsonism and Related Disorders 87 (2021) 105–110

Contents lists available at ScienceDirect

Parkinsonism and Related Disorders

journal homepage: www.elsevier.com/locate/parkreldis

Comparing subjective and objective response to medications in Parkinson's disease patients using the Personal KinetiGraph™

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ARTICLE INFO

Keywords:
Parkinson's disease
Wearable technology
Personal kinetigraph
Objective assessment

ABSTRACT

Background: Management of motor symptoms in Parkinson's Disease(PD) relies on subjective information provided by patients, the quality of which can be affected by many factors.
Rationale: Objective data collected during daily life could complement this information and improve management of motor symptoms.
Objectives: To assess the usefulness of the Personal KinetiGraph (PKG) in characterizing the intensity and timing of motor symptoms in PD patients.
Methods: Retrospective study of all PD patients followed at a tertiary academic movement disorders center assessed by PKG between December 1, 2016 and October 30, 2018. PKG was worn for 7 days prior to the clinical visit. We compared the information obtained from the interview and the clinical visit, and assessed the impact of the PKG on treatment decision making.
Results: 170 PKG results were reviewed. PKG complemented patient input in 82.9%(141/170) and led to medication changes in 71%(100/141) of the complemented inputs. PKG contributed the least to correcting or complementing patients' input when patients self-reported as undertreated (22%) and the most when patient were unable to answer all questions regarding motor response to individual doses (100%) (Fisher, $p < 0.0001$). The majority of patient undergoing 3 or 4 PKG encounters did not reach a controlled state as defined by PKG until the 3rd or 4th encounter, suggesting that repeated use of the PKG might be needed to help optimize motor control as therapy changes done after one encounter might not be enough.
Conclusions: PKG might be useful in supplementing patient-provided information for accurate assessment and treatment plan.

1. Introduction

Parkinson's disease (PD) is one of the most common neurodegenerative disorders affecting more than ten million people worldwide [1]. As one aim of the management of motor symptoms of PD is to improve bradykinesia and tremor while minimizing dyskinesia, the treating clinician must establish the extent and timing of motor symptoms and modify the timing and dose of medications accordingly. One of the most frequent challenges is reliance on self-reporting. For example, patients are frequently unaware of the presence of levodopa-induced dyskinesia [2]or can confuse them with tremor. In addition, patients often find it difficult to specify the response to medication, giving the treating physician approximate responses in term of amplitude and duration of improvement with individual medication doses [3]. This can potentially affect changes in pharmacotherapy and ultimately symptom control, with up to 50% of PD patients having uncontrolled motor symptoms [4]. Data collection during patient interview can be misleading due to subjectivity with patient recall, variations in clinician skill, as well as possible PD related impaired cognition [5,6]. Finally, detecting poor compliance on interview alone can also be challenging. As such, the lack of longitudinal objective measurement can prevent effective and timely management of PD [7]. The Movement Disorder Society Task Force on Technology recently acknowledged these challenges and suggested a roadmap for the implementation of wearable technology, including developing selection criteria for the use of this technology [8].

The Personal KinetiGraph™ (also known as the Parkinson's KinetiGraph™ or PKG™, Global Kinetics Corporation, Australia) is approved for use in Australia and Europe [9], and has FDA clearance in the United States [10]. However, there is no data comparing the results of the PKG with the information provided by patients during routine clinical care.

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https://doi.org/10.1016/j.parkreldis.2021.05.008
Received 1 February 2021; Received in revised form 4 May 2021; Accepted 6 May 2021
Available online 11 May 2021

We decided to address this lack and evaluate the usefulness of PKG in characterizing the amplitude and timing of bradykinesia, motor fluctuations, tremor and dyskinesia in PD patients. For that reason, we compared the information typically gathered in clinic by a movement disorder specialist to the information provided by the PKG, and assessed the impact of the PKG on treatment decision making.

2. Methods

2.1. Technology

Although other similar devices were more recently made clinically available, PKG is the oldest and most studied commercially available system providing continuous, objective, and ambulatory assessment of bradykinesia [11]. The PKG system consists of a wrist worn logger, proprietary algorithms to produce bradykinesia, dyskinesia, and tremor scores, and a clinically intuitive presentation of this data in graphical and numerical format. The logger is a smart watch weighing 26 g (46 g with the wrist strap) worn on the most affected wrist (Fig. 1A). It contains a rechargeable battery as well as a 3-axis iMEMS accelerometer set to record 12-bit digital measurements of acceleration with a range of ±4 g and a sampling rate of 32 samples per second using a digital microcontroller [5]. It has data storage on flash memory that allows it to record continuously for more than 10 days. It can also be programmed to remind the patient to take the PD medications through a scheduled vibration. The patient then acknowledges taking the medications by swiping the logger's smart screen [5]. The algorithms were developed using an expert system approach to model how neurologists recognize bradykinesia and dyskinesia on accelerometry data. The inputs from the accelerometer included the Mean Spectral Power (MSP) of acceleration between 0.2 and 4 Hz, as well as peak acceleration, and the amount of time without any movement. These inputs were weighted to model how neurologists rate bradykinesia and dyskinesia and to produce a bradykinesia score (BKS) and a dyskinesia score (DKS) every 2 min [5]. The final report is a graphical representation of these scores collected every 2 min over an extended period (typically 6 days). When worn continuously for 6 days, 4320 2-min data points are generated. The PKG plots the mean BKS and DKS (with a smoothing function) against the time of the day (Fig. 1B) and also shows the scheduled time of medications intake, making it possible to assess for dose-related variation in BKS or DKS and to compare the median value at any time of the day with these of a normal subject. Finally, numerical scores for median BKS and DKS, as well as their 25th and 75th percentile are presented, along with an objective motor fluctuation score [12] and the percent of the time with tremor or asleep [13,14] (Fig. 1 C). The PKG was previously validated for the measurement of bradykinesia and dyskinesia by comparing it to the motor section of the Unified Parkinson's Disease Rating Scale, and to the Abnormal Involuntary Movements Scale [15].It was also validated for the measurement of rest tremor [13] and showed reliability on repeated measures of the response to levodopa [15]. Targets have been developed to characterize patients with PD as controlled or uncontrolled based on the various motor scores provided by the PKG and are detailed elsewhere [16].

2.2. Participants

This was a retrospective evaluation of all PD patients followed by a movement disorders specialist(R.M.) at a tertiary academic center and who were assessed by PKG between December 1, 2016 and October 30, 2018. The study was approved by the institutional review board (IRB number HSC-MS-18-0917). Patients were included in the study if they had been diagnosed with PD by a movement disorders specialist and were able to understand the instructions for the use of PKG. Patients were excluded if they were diagnosed with another cause of parkinsonism than PD, such as atypical or vascular parkinsonism, as the PKG was not previously validated for non-idiopathic PD. Patients diagnosed with dementia or with a language barrier with poor understanding of English were also excluded, as instructions about PKG and communication with the PKG managing and mailing office were exclusively in English. Patients signed an informed consent allowing the managing and mailing office to contact them and to mail them the PKG.

2.3. Data collection

Each patient was instructed to wear the device on the side of worse motor symptoms for 7 days (with a minimum of 5 days for the data collected to be representative of the patient's pattern) while performing routine activities of daily living. The patient only had to swipe the logger after taking each dose of medication. After completion of each encounter, the patient mailed the PKG back. The results were uploaded on the PKG portal. During the course of the clinical visit, the interview and examination were performed with the clinician (R.M.) blinded to the PKG results. Using layman's terms, the clinician asked the patient about the response of motor symptoms to individual doses of medication throughout the day, including latency to onset of effect, amplitude of effect and duration of effect. The clinician also asked about presence of motor fluctuations and their intensity, as well as the presence of dyskinesia, their timing in regards to dose intake, and their intensity. Finally, the clinician asked if the patient felt well controlled with the medication regimen or would request a change to it. The patient and clinician's impressions as well as a treatment plan were noted in the chart before the PKG results were reviewed. The clinician then interpreted the PKG results to answer the same questions he had asked the patient, looking for latency, amplitude and duration of effect of each medication intake, presence of dyskinesia and motor fluctuation as well as reviewing the overall bradykinesia, dyskinesia, tremor and fluctuation scores. Based on these results, the clinician modified the initial treatment plan when appropriate (adding a new medication, stopping a medication, or changing dosage and/or frequency of medication intake). The PKG report, recommendations for change and patient's acceptance or refusal of these recommendations were documented in the chart.

2.4. Data analysis

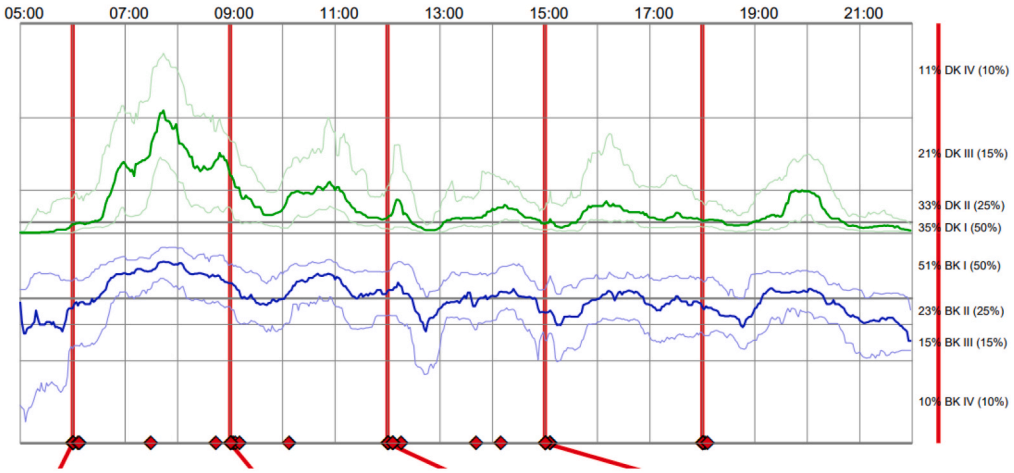
An electronic medical record chart review was performed for eligible patients by E.K. and J.R. PKG encounters with incomplete data or early discontinuation (less than 5 days of recording) were excluded from the study. The chart review recorded patient's age, gender, disease duration, category of patient's ability to provide details about motor response to PD medications, and any changes in medications attributed to the use of PKG. Patients' categories were determined based on the movement disorder's specialist previous experience with the various answers obtained from patients when asked about their response to PD medication in routine care. The PKG having been previously compared to diaries [17], we do not routinely use written diaries in our clinic as they are redundant to the use of PKG, have several imitations [18] and are cumbersome to use according to our patients. The categories were defined as follows:

1 Patient was unable to give all characteristics of response to individual doses of medication (latency from medication intake to initial therapeutic effect, percentage of improvement in the ON phase, duration of benefit/wearing off before the next dose, presence, timing and severity of dyskinesia).
2 Patient did not feel any response to medication individual doses but overall felt better.
3 Patient felt no response at all to medication, i.e. considered him/herself undertreated.
4 Patient was able to give all characteristics of response to individual doses of medication.

To ensure the impartiality of this study, the manufacturer of the PKG

A: picture of the PKG logger

B: picture of the first page of the PKG report in a patient.



C: Picture of the score sheet of the PKG report in a patient

	Patient	Control	(Percentiles)
BK (09:00 - 18:00)	12.7 - 18.6 - 27.7	12.7 - 18.6 - 26.1	25 - 50 - 75
DK (09:00 - 18:00)	1.5 - 5.9 - 15.3	0.9 - 4.3 - 16.5	25 - 50 - 75
FDS (09:00 - 18:00)	10.3		
PTI (09:00 - 18:00)	3.2%		
PTT (09:00 - 18:00)	0.5%		

Fig. 1. A: picture of the PKG logger

Fig. 1 B: picture of the first page of the PKG report in a patient.

Legend: the red vertical lines indicate the times of the day at which the medication reminders were set. The red diamonds at the bottom indicate the times at which the patient swiped the logger to acknowledge taking the medication. The green line represents the dyskinesia score: the further up, the higher the score and the worse the dyskinesia. The blue line represents the bradykinesia score: the further down, the higher the score and the worse the bradykinesia. The heavy colored line represent the median, and the 2 lighter lines represent the 25th and 75th percentile of each score at each time of the day, over 6 days. At the right of the picture, the percentage of the recording spent in each category, compared to healthy control between parentheses. For more details, please see Farzanehfar and Horne[5].

Fig. 1C: Picture of the score sheet of the PKG report in a patient

Legend: BK = Bradykinesia, DK = Dyskinesia, FDS = fluctuation score, PTI = percentage of time immobile, PTT = percentage of time with tremor. This figure shows the numeric scores for BK and DK. The middle number is the median, with the 25th and 75th percentile to the left and to the right respectively. Numeric scores for healthy controls are provided for comparison. The FDS is positively correlated to the severity of motor fluctuations. A high PTI can be suggestive of excessive daytime sleepiness. Targets to characterize patients with PD as controlled or uncontrolled based on the various scores above are detailed elsewhere [16]. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

was not involved in funding, study design, data collection or data analysis.

2.5. Statistical analysis

Using the Fisher test, we compared the proportion of patients whom history was corrected or complemented by PKG between the 4 categories described above. The statistical significance was set at $p < 0.05$.

3. Results

181 total PKG encounters were reviewed. Eleven encounters were excluded due to defective equipment, equipment lost in mail, or patient withdrawing consent. 170 total PKG encounters corresponding to 104 patients were eligible for the study, as 49 patients received multiple encounters. There were 62 males and 42 females with average age of 68.6 years and an average levodopa equivalent daily dose of 831.14 (±484.97). The characteristics of the PKG encounters are summarized in Table 1. Eleven patients had bilateral subthalamic nucleus deep brain stimulation (DBS) devices in place at the time of PKG recording, corresponding to 12 PKG encounters. PKG complemented patient input in 82.9%(141/170) and led to medication changes in 71%(100/141) of the complemented inputs (Table 2).

The PKG led to a change in medication (increases, decreases, or introduction of new drug) in 100 out of the 141 encounters (71%) where it complemented the patient's input. Of these, 79 (79%) led to increase in medications and 23 (23%) led to the introduction of a new drug, 7 of which were amantadine immediate or extended release for dyskinesia. On the other hand, 6 PKG encounters (6%) led to decrease in medications: 4 because of levodopa induced dyskinesia; and 2 for lack of response to carbidopa/levodopa, in order to assess patient off medications and reconsider the diagnosis. The total number of medication changes (108) was superior to the number of PKG encounters leading to medication changes (100) as some encounters led to more than one medication change. PKG encounters in patients with DBS were distributed between the categories as follows: 2 encounters in category 1, 1 in category 3 and 9 in category 4. PKG results prompted programming in 4 encounters (all in category 4) with objective and subjective improvement.

The difference in the proportion of encounters in which the PKG modified the patient's input was statistically significant between the 4 categories (Fisher, $p < 0.0001$), with PKG contributing the least to correcting or complementing patients' input when patients self-reported as undertreated (22%) and the most when patient were unable to answer all questions regarding motor response to individual doses (100%).

Out of the 104 patients, 49 (46%) had more than 1 PKG encounter: 37 patients had 2 encounters, 7 had 3 encounters and 5 had 4 encounters (Table 3). Among those with 2 encounters, 27% changed from an uncontrolled to a controlled state from the first to the second encounter. The majority of patient undergoing 3 or 4 PKG encounters did not reach a controlled state as defined by PKG until the 3rd or 4th encounter.

Table 1
Encounter characteristics.

	Category 1	Category 2	Category 3	Category 4
Age - mean (SD)	71 (10)	64 (10)	75 (5)	67 (10)
M/F ratio	25/21	21/7	12/6	43/35
Time since PD onset in years- mean (SD)	7.41 (5.69)	5.55 (3.06)	7.83 (4.32)	7.09 (4.8)
Time since PD dx in years- mean (SD)	5.103 (4.5)	3.9 (3.46)	4.83 (4.26)	5.16 (4.17)
LEDD mg –mean (SD)	962.11 (574.91)	662.23 (379.66)	717.78 (357.24)	855.84 (469.33)
% on C/L	100	78.6	94	93.7
% on DA	30.4	71.4	33	44.3
% amantadine	13	0	0	16.5
% on MAOi	8.7	14.3	0	15.2
% on COMTi	17.4	0	5.6	15.2

Legend: SD = standard deviation, M = male, F = female, LEDD: levodopa equivalent daily dose C/L = carbidopa/levodopa, DA = dopamine agonist, MAOi = Monoamine oxidase inhibitor, COMTi = Catechol-O-methyltransferase inhibitor. Note: medications reported are before PKG induced changes in therapy.
Of note, there was no difference between the categories' average age (Anova F = 5.66, p = 0.75).

Table 2
Impact of PKG results on assessment and management.

Category	Number of PKG encounters	PKG modified	Treatment changed after PKG (%)
		patient input	
1	46	46 (100%)	25 (54%)
2	28	24 (86%)	19 (79%)
3	18	4 (22%)	0 (as these patient felt uncontrolled but were found to be adequately controlled on PKG and thus did not require medication change)
4	78	67 (86%)	56 (including 4 DBS programming) (84%)
Total	170	141 (83%)	100 (71%)

PKG: Personal Kinetigraph.

4. Discussion

In this retrospective series, PKG data was reviewed to assess motor symptoms and response to medications in PD patients. This objective data was utilized by the physician to verify patient's reported symptoms and optimize treatment decisions.

4.1. PKG input by category

Overall, the PKG corrected or complemented patient input in 141 cases (83%) and confirmed it in 29 (17%). It complemented or corrected the data in all PKG encounters (100%) in category 1 as expected, with 38/46 (82.6%) of these being uncontrolled while the rest were well controlled on their medication regimen. Following the input from the PKG, a medication regimen change was initiated in 25/46 (54%) while the rest did not undergo medication escalation either because of good control on PKG, side effects such as hallucinations or orthostatic hypotension, or in one case because of patient refusal.

However, in patients reporting overall feeling better despite a lack of response to medication individual doses (category 2), the PKG demonstrated under-treatment in 24/28 encounters (86%). While these patients might have otherwise been considered well controlled since they had reported feeling good overall and initially declined any change in medications, the use of the PKG led to medication change in 19/24 encounters (79%). In the remaining 5 encounters, patients declined increase in medications, for fear of medication side effects or dyskinesia.

Interestingly, 4/18 encounters (22%) in category 3 showed patients were actually well controlled according to the PKG and did not require an increase in medications doses, which would have likely happened in the absence of the PKG. Among those who were not well controlled, patients agreed with medication increase in 8/14 encounters (57%) and refused in the rest, most often for fear of medication side effects or dyskinesia.

The most notable, however, is that 67 of the 78 encounters (86%)

Table 3
PKG changes in patients with more than 1 PKG encounter.

Number of PKG encounters	Number of patients	Interval between first and last PKG (mean, +/-SD; range) in months	Number of patients uncontrolled at first PKG, but controlled at last PKG	Number of patients uncontrolled at first PKG and uncontrolled at last PKG	Number of patients controlled at first PKG and controlled at last PKG	Number of patients controlled at first but uncontrolled at last PKG
2	37	6.3 (±3.8; 1 to 17)	10	21 (PKG numbers improved but did not reach target)	3	3 (seen 9–12 months later and missed at least one appointment)
3	7	11.4 (±1.4; 10 to 14)	2 (one at the 2nd and one at the 3rd encounter)	2 (1 refused changes to treatment, 1 improved but did not reach target)	2 (uncontrolled at the 2nd but controlled again at the 3rd encounter)	1 (became uncontrolled at the 3rd encounter)
4	5	15.8 (±2.3; 12 to 18)	3 (1 at the 3rd encounter, 2 at the 4th encounter)	1 (was controlled only at the 3rd encounter)	1 (controlled through all 4 encounters)	0

PKG: Personal Kinetigraph.

where patients confidently provided all requested details regarding timing and amplitude of motor symptoms and potential motor complications (category 4) were actually corrected by the PKG, and 56/67 (84%) had a modification of the medication regimen as a result.

4.2. Comparison between categories

Overall, PKG contributed the least to correcting or complementing patients' input when patients self-reported as undertreated (22%) and the most when patient were unable to answer all questions regarding motor response to individual doses (100%) (Fisher, $p < 0.0001$). PKG contribution remained high, complementing the history intake in more than 80% of the encounters when patients confidently reported all details or reported overall improvement without clear response to individual doses.

4.3. Serial PKG

Notably, 50%(1/2) of the patients with 3 encounters who were initially uncontrolled but were controlled at the end of the study did not reach the controlled state until the 3rd encounter. Similarly, 100%(3/3) of those with 4 PKG encounters who were initially uncontrolled but were controlled at the last visit, did not reach the controlled state until the 3rd or 4th encounter. As such, the relatively low percentage (27%) of patients with 2 PKG encounters changing from an uncontrolled to a controlled state could possibly increase after subsequent treatment modification at following visits, since one therapy change might not be enough to move the PKG results to target, but change built over multiple encounters might.

4.4. Positive findings

In summary, our results underline the importance of characterizing and verifying patient's input with wearable devices such as PKG in order to optimize medical management, even in those patients who sound like reliable historians (category 4 in our study), supporting previously published expert opinions [6,19–21]. Our results also suggest that repeated use of the PKG might be needed to help optimize motor control as therapy changes done after one encounter might not be enough and 50–100% of our patients with 3 or 4 PKG respectively did not change from their uncontrolled initial state to controlled final state until their 3rd or 4th encounter.

We are aware of only one other study assessing the post-validation use of PKG in clinical practice [16], involving 103 PD patients, none of them included in our cohort, and reporting suboptimal control in 78% of patients, of which 89% underwent treatment modification while the remaining 11% could not because of the concern for side effects at higher doses. While that study also reported improvement in quality of life as measured by the PDQ39 in patients treated to target as defined by the PKG, it did not compare the PKG results to the patient's interview. That comparison, performed in our study, underlines the usefulness of this objective method in supplementing information provided by the patient for accurate clinical assessment and treatment plan. Furthermore, our results might suggest which patients might be more likely to benefit and that more than one recording might be needed overtime to optimize motor control, although the design of our study does not allow for definitive conclusions.

4.5. Limitations

Being worn on one wrist, usually on the side most affected by PD, PKG might not capture a patient's full motor picture and, for example, underestimate dyskinesia affecting the opposite arm, the trunk or the legs; or tremor affecting the legs or the jaw. Similarly, low frequency high rhythmicity dyskinesia can rarely be captured as tremor on the PKG, but would still be captured as dyskinesia as well, making the distinction between low frequency dyskinesia and parkinsonian tremor usually easy. Clinical assessment by a trained physician is still necessary to integrate the information provided by the PKG with other symptoms that are not measured by this technology such as hallucinations and lightheadedness that could worsen on higher dose of medications if a patient was categorized as poorly controlled by PKG; or dystonic or truncal symptoms that could require treatment modifications but went undetected by the PKG.

In addition to the limitations inherent to the PKG, our study has some other limitations. First, it is a retrospective chart review, carrying the risk of incomplete or inaccurate data recorded in the patient charts. However, all the data was collected prospectively by the same clinician following an established template followed in clinic. Second, the quality of the patient's interview might have been suboptimal or heterogeneous and thus artificially increase the value of PKG. However, all the interviews were performed by the same movement disorders formally trained clinician, with more than 6 years of experience in the field of movement disorders at the time the use of PKG began in our clinic. Actually, awareness that the information collected during the interviews will be compared to data collected by the PKG might have led the clinician to be more thorough in his evaluation, thereby potentially artificially decreasing the value of PKG. Regarding possible bias, the interview and examination were performed with the clinician blinded to the PKG reports, with patient's and clinician's impression noted in the chart before the PKG results were reviewed. The PKG results were then reviewed and noted in the chart, as well as any potential change in management that would have resulted. In addition, retrospective data collection was performed by another clinician to avoid any collection bias. Global Kinetic Corporation, the manufacturer of the PKG, was not involved in funding, study conception, data collection, data analysis, manuscript writing or manuscript revision. Finally, while amantadine was not used in category 2 and 3, and while patients in category 2 had the lowest percentage of carbidopa/levodopa use but the highest percentage of dopamine agonist use likely related to the shorter duration of

disease, these differences are unlikely to create any bias that could impact our findings or their interpretation. While repeated PKG in a subgroup of our patients showed an improvement in motor control per PKG targets, our study did not formally assess the impact of the therapy changes on patients' symptoms and quality of life. Randomized control trials addressing these questions are ongoing.

5. Conclusion

We present the first study demonstrating the added benefit of PKG to the patient's interview and its impact on treatment decision. We report than even a priori confident patients reporting good response to treatment need their input supplemented by an objective device, while those reporting complete lack of response are most often correct. As such, PKG can be a useful tool in the management of PD, when used in the appropriate context and correlated to the clinician's observations. Furthermore, more than one recording might be needed overtime to optimize motor control, although the design of our study does not allow for definitive conclusions. The implications on global healthcare will still have to be formally assessed. It has been demonstrated that better motor control in PD patients decreased global health care cost essentially by preventing additional clinic visits or urgent care visits [22–24]. Financial incentives of wearable technology have been suggested based on informed treatment-decision processes [5,21,25,26]. Prospective randomized controlled trials with double blind approach including two independent groups of patients (a first subgroup of patients undergoing only clinical evaluation versus a second subgroup undergoing clinical and PKG evaluation) are ongoing. Until these trials are completed and published, our study suggests additional benefits from the use of PKG in clinical practice by complementing the information provided by patients.

Funding sources

None.

Disclosures

R Mehanna serves as a consultant for Global Kinetic Corporation, and is on the speaker bureau for TEVA, Adamas Pharmaceuticals, Kyowa Kirin, Sunovion and Accorda Therapeuthics. He has received research grants from Lundbeck, Solstice neurosciences, Prilenia, Global Kinetic Corporation, Northera, Neurocrine and Abbvie. E Krause and J Randhawa have nothing to disclose.

Authors' contribution

Krause: data collection, data analysis, writing of the first manuscript, approval of the final version.

J Randhawa: partial data collection, approval of the final version.

R Mehanna: conception and design, data analysis, review and critique, editing, approval of the final version.

Declaration of competing interest

R Mehanna is a paid consultant for Global Kinetics Corporation (GKC), the manufacturer of the personal kinetigraph (PKG). However, GKC was not involved in study conception, data collection, data analysis, manuscript writing or manuscript revision. E Krause and J Randhawa report no conflict of interest.

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