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Research Article

Muscle Activation Signals During Gait Parkinson's Disease are More Rhythmic than in Healthy Controls

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Abstract

Background

To quantify the underlying rhythmic patterns observed in surface electromyography (SEMG) in patients with Parkinson's Disease (PD), to compare rhythmicity of PD gait with normal gait, and to compare overground walking to treadmill gait.

Methods

Eight individuals with idiopathic PD and ten individuals with no history of neurological disorders participated. SEMG was recorded from the vastus lateralis, tibialis anterior, lateral gastrocnemius and biceps femoris muscles bilaterally. Each participant performed two trials of overground walking and two trials of treadmill walking at a self-selected comfortable walking speed. SEMG was analyzed using a non-linear statistical model that identified underlying recurrent locomotor patterns, which estimated the relative contribution of central pattern generators to the observed muscle activation signals. An index of rhythmicity was determined from the statistic, R^2.

Results

The rhythmicity of PD gait was significantly higher than that of normal gait (p = .0458). There was also a significant difference between the rhythmicity of overground walking and treadmill walking (p = .0097).

Conclusions

Individuals with PD appear to walk with muscle activation patterns that are more rhythmic than normal. This suggests that there is more stride-to-stride consistency, and there are fewer postural adjustments and responses to perturbations. We also found that treadmill gait was more rhythmic than overground walking. These findings, although preliminary, challenge the paradigm and current approach to gait retraining of patients with PD.

Keywords:
Surface electromyography (SEMG); gait; central pattern generator; rhythmicity

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Introduction

Parkinson's Disease (PD) is a complex neurodegenerative disorder that affects approximately 0.3% of the population worldwide[1]. Clinical management of PD currently focuses on pharmacological treatment and surgically implanted devices[2]. While these approaches effectively treat some motor symptoms, they are less successful at treating postural instability and gait disturbances, especially as the disease advances [3,4]. There is a growing interest in developing physical activity-based treatments such as tai chi and tango dancing that are designed to retrain balance and locomotion through repetitive movement[5]. However, there is limited evidence that these approaches are effective[6]. The focus of many physical therapy interventions is to promote rhythmic movement through cadenced repetition. We propose that cadenced repetition may contradict the pathophysiology of PD, which has been characterized as having greater-than-normal rhythmicity[7]. Normal gait involves a certain amount of variability, which is believed to optimize dynamic stability [8]. So, we set out to describe the rhythmic characteristic of muscle activation signals in PD compared to a normative sample to provide deeper insight into the neuromuscular behaviour of PD gait disturbances.

Surface electromyography (SEMG) signals can provide a non-invasive representation of neural behaviour downstream from the central nervous system. Previous SEMG studies have identified abnormal motor control features of PD that have helped inform our understanding of the disease[9,10,11]. Ferrarin et al. (2007) identified the following abnormalities in EMG activity during PD gait[12]. There is prolonged activation of Rectus Femoris (RF) and Semimembranosus (SM) muscles throughout the gait cycle. Recruitment of the RF at heel contact and the SM in late swing are reduced. There is also reduced recruitment of the Gastrocnemius Medialis (GM) at push-off, and reduced recruitment of the Tibialis Anterior (TA) at heel contact. Some of these effects can be partially ameliorated with levodopa[13]. Premature activation of the TA and GM is seen prior to freezing of gait[14]. Miller et al. (1996) reported

that variability in the gastrocnemius muscle was greater than normal in PD gait[15]. Taken together, these findings present a detailed but obscure picture of the pathological control of gait in PD.

There are a number of characteristic gait disturbances associated with PD, such as reduced arm swing, slow walking speed, short step length, and difficulty turning[6,16]. PD gait is also characterized by increased variability of walking speed, step length, step width, and stride duration[17,18]. Fractal analyses have shown that the kinematics of PD gait are less ordered, and more complex and random-like compared to normal gait[16,19]. All of these gait features negatively impact mobility and balance. They also contribute to postural instability and risk of falling, which can lead to further injury, disability and death[4]. In PD, the relative contribution to gait of supraspinal centres is altered[20]. It is not clear how or to what extent the gait disturbances are caused by impaired supraspinal input. The present study is one of the first attempts to analyze and interpret multiple muscle SEMG during PD gait.

The aim of this study was to describe rhythmic patterns of peripheral muscle activation in PD and identify differences from normative behaviour. The foundation of our analysis is the idea that spinal locomotor control consists of a set of modular burst generators[21,22]. The idea that human gait is controlled by an efficient, minimal set of movement primitives, or muscle synergies, located in the spinal cord has been advanced for many years [23,24]. Muscle synergy analysis can be used by clinicians to draw inferences about neural structures underlying motor behaviours and guide rehabilitation decisions [25]. There is some opposition, however, to the muscle synergy hypothesis in the scientific literature [26]. Physiological interpretations based on the idea that reducible patterns in peripheral muscle activity reflect simplicity within the central nervous system should be made with caution.

Methods

Participants

Eight (n=8) individuals with idiopathic, mild to moderate PD (Hoehn-Yahr scale 1 to 3)[27], and a convenience sample of ten (n=10) individuals with no history of neurological disorders (Able-bodied group) participated in this study. PD participants were recruited from an outpatient clinic of the Movement Disorders and Neurorehabilitation Center at the Methodist Neurological Institute in Houston, TX. All PD participants used Levodopa and performed all tasks while in the "ON" state. The study procedures were approved by the University of Houston's Committee for Protection of Human Subjects, and all participants provided informed written consent. Descriptive statistics of the participants are provided in Table 1.

Experimental Protocol

Each participant performed two walking trials over ground (OG), followed by two walking trials on a treadmill (TM). Between each trial, participants sat and rested for 5 minutes. The OG trials consisted of the participants walking along a straight 10m track on a hardwood floor in a large, open gymnasium. They started walking 2m before the track and continued walking for at least 2m after the track, such that the trial did not include step initiation or stopping. Participants were instructed to "walk as though you are going to mail a letter." Their self-selected walking speed was calculated as 20m divided by the time it took to complete both OG trials (i.e., average speed of both trials). During the TM trials, the treadmill speed was set to the participant's self-selected walking speed. Each TM trial was 60s in duration at a constant speed. An equal number of gait cycles were analysed under both conditions for each subject.

Table 1. Summary statistics of participants

Group	N	Age (years)	Sex	H&Y*
PD	8	71±12	6M/2F	2.2±0.7
AB	10	29±10	6M/4F	–

* Hoehn & Yahr scale [19]

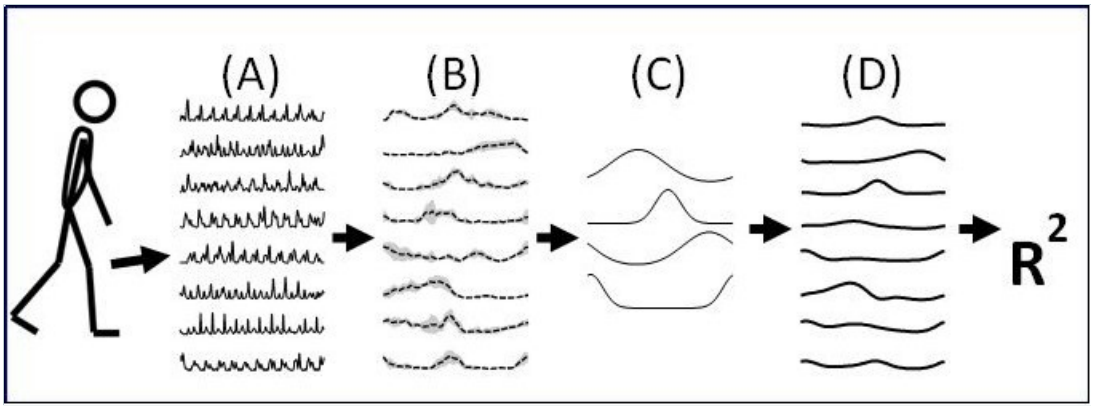


Figure 1. Classification procedure. Processed SEMG signals (A) were separated into time-normalized gait cycles (B), and coded as four synergistic patterns of muscle activity during the gait cycle (C). Signals were reconstructed (D), and the amount of information retained was evaluated by the statistic, R^2.

Instrumentation

Participants' lower extremities were instrumented with an 8-channel SEMG system (DataLOG MWX8, Biometrics Ltd, Ladysmith, VA, USA). Dry reusable surface electrodes (10mm diameter discs with 20mm between centres) were carefully placed over the muscle belly of the following muscles bilaterally: vastus lateralis (VL), long head of biceps femoris (BF), tibialis anterior (TA) and gastrocnemius lateralis (LG). The SENIAM recommendations for sensor location were used[28]. These particular muscles were selected as a representative set of the major actuators during gait[29]. The skin was cleaned and lightly abraded before the electrodes were attached with double-sided adhesive tape. SEMG signals were detected via differential electrodes, pre-amplified (1000 gain), filtered (bandpass: 20 – 450 Hz), and A/D converted at a sampling rate of 2000 Hz. The beginning and end of each gait cycle were determined by initial contact of the right foot, which was detected by the activation of a foot switch placed on the sole of the right shoe directly under the heel.

Data Analysis

After recording, SEMG signals were rectified and filtered using a zero-lag, low-pass Butterworth filter with a cut-off frequency of 10Hz, which is considered sufficient for noise removal without loss of signal[30]. Signals were then coded by fuzzy sets according to a classification procedure previously published[31]. Figure 1 illustrates the procedure. This method is designed to represent multiple muscle activation signals as a recurrent sequence of four basic burst patterns in the manner of a spinal locomotor circuit[21]. Four was shown to be the optimal number of bursts[31]. Pearson's correlation coefficient, R, was determined using the empirical SEMG data versus the model estimation across all channels and gait cycles. The relative amount of SEMG signal that fits this model can be quantified using the statistic, R^2, which is a measure of goodness of fit between the empirical data (Figure 2B) and the model-fitted signals (Figure 2D). R^2 incorporated every individual gait cycle, and was calculated for each individual trial. We interpreted R^2 as an index of rhythmicity, because it represents the proportion of the muscle activation signals that recur with observed regularity. Any stride-to-stride variability in muscle activation would not be represented by the model and would therefore decrease the index of rhythmicity.

A two-way analysis of variance (ANOVA) was performed with R^2 as the dependent variable. We tested for main effects of one between-subject factor (PD versus Able-bodied), and one within-subject factor (OG versus TM). We also tested for an interaction effect of these two factors. The difference in self-selected walking speed between groups was tested using Student's t-Test for independent means. A level of significance of $p < 0.05$ was used. All data processing and statistical analyses were performed using custom-written software in the MATLAB programming language (The Mathworks, Inc., Natick, MA, USA).

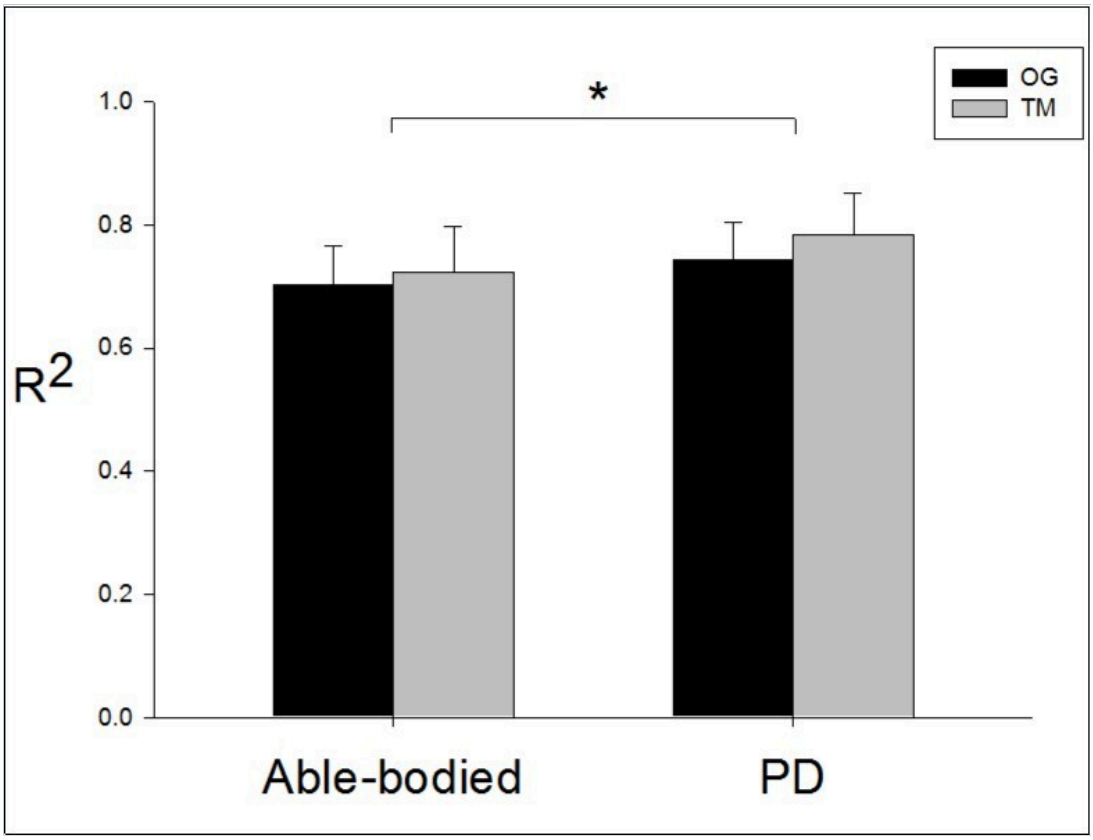


Figure 2. R2 for both groups, both walking conditions. Index of rhythmicity of muscle activation patterns for both groups and both conditions. Mean values for each group shown. Error bars represent standard deviations. Significant differences between groups and between walking conditions were observed

Results

All participants successfully completed the protocol without difficulty. SEMG signal quality was good in all cases. The classification procedure yielded values of R^2 from 0.582 to 0.866. The means and standard deviations for the different groups and walking conditions are illustrated in Figure 2. There was a significant group effect on the index of rhythmicity, R^2 (p = .0458). The mean (± standard deviation) index of rhythmicity was 0.7754 ± 0.0574 for the PD patients, and 0.7138 ± 0.0683 for the able-bodied subjects. There was also a significant difference between OG walking and TM walking (p = .0097). The mean (± standard deviation) index of rhythmicity for both groups of participants was 0.7264 ± 0.0625 for OG walking, and 0.7559 ± 0.0757 for TM walking. There was no significant interaction between factors (p = .3671).

There was a significant difference between PD and the able-bodied group in terms of self-selected walking speed (p = .0024). The mean (± standard deviation) walking speed was 1.29 ± 0.18 m/s for the able-bodied subjects, and 0.87 ± 0.31 for the PD patients.

Discussion

Healthy, mature motor skills are governed by neurological processes that allow for an optimal amount of movement variability[32]. Systems that produce more than optimal variability are noisy and unstable, while systems that produce less than optimal variability tend to be rigid and unfit to cope with perturbations. Pathological conditions can result in either case.

The main finding of this study is that the rhythmicity of muscle activation signals in the lower extremities of elderly individuals with PD is higher than in young, able-bodied individuals. In other words, the muscle activation patterns in PD exhibit greater regularity and are more consistent with the four-burst model of spinal locomotor control. It also means that there was less stride-to-stride variability in terms of

SEMG in PD patients than in able-bodied individuals. In normal gait, the majority of the SEMG signals (70% OG, 72% TM) were explained by the four-burst CPG model. The parts of the signal that were not explained by the model would be made up of non-rhythmic elements such as anticipatory postural adjustments, feedback response to irregular perturbations, and aberrant neurological commands (i.e., noise). In PD gait, a significantly greater portion of the SEMG signals is represented by the CPG model (76% OG, 80% TM). This is a rather unexpected result. We presumed that PD gait would involve more aberrant neurological commands than normal, based on reports that there is higher variability in PD gait [15,16,33]. However, the overall amount of non-rhythmic elements in SEMG was reduced in PD gait, suggesting that there are fewer stride-to-stride adjustments and responses to errors.

The idea that human locomotion is driven by oscillating neural circuits located in the spinal cord has been advanced for decades[34]. These circuits, known as the Central Pattern Generator (CPG), provide rhythmic bursts of muscle activation signals that form the basis of locomotor control[35,36,37]. Rhythmic patterns can be identified using a statistical model of CPG[31]. By this approach, we are able to estimate the relative contribution of the CPG to muscle activation signals. Furthermore, we can estimate how much of the variability in gait is due to sources other than the spinal CPG circuits (e.g., feedforward adjustments from supraspinal centers).

If confirmed by a larger study, these findings may lead to significant changes in current clinical approaches to gait retraining of patients with PD. The traditional physical therapy approach of retraining locomotion through rhythmic activities such as treadmill training, tandem bicycling, and OG walking may be inconsistent with the fact that rhythmic muscle activation patterns are already more pronounced in PD[5,6]. These findings may support the development of new research protocols for assessing the role of conventional therapies, such as dopamine replacement therapy and DBS, on neurological control of gait.

One of our secondary findings was that there is a significant difference between OG walking and TM walking in both groups. Muscle activation signals during TM were more rhythmic than during OG walking. This result was expected. TM walking involves a more stable, controlled environment with fewer irregularities to perturb the body. Therefore, there is less need for anticipatory adjustments from the supraspinal control centres, and the spinal CPG output will be more represented in the muscle activation signals.

The variability of PD gait has been reported broadly in the scientific literature. Most analyses have focused on the variability of kinematic features of gait, i.e., stride length, stride interval, gait speed, etc. [17,18]. These studies have all concluded that the variability of movement in PD gait is greater than in normal gait. However, none of them address the cause of the movement variability. Our findings suggest that there is reduced variability in the control signals that are sent to the skeletal muscles. This does not contradict the previous observations of increased movement variability.

It should be noted that comparisons between the PD group and the young, able-bodied controls could be confounded by age. It is not possible to determine from our data whether between-group differences are due to age, disease or something else. The Able-bodied data is provided as a reference of normative behavior.

The present study is one of the few analyses that deal with variability of multiple muscle activation signals using SEMG. Some studies have focused on individual SEMG signals. Previously, it was reported that the variability of SEMG in the GM muscle is greater in PD than in able-bodied individuals[12]. However, when we looked at multiple muscle SEMG signals, we found that they were much more representative of basic CPG function. There is no inconsistency between these two different findings. Our methods represent a broader paradigm which is more consistent with the complexity of CNS control of gait.

Conclusion

Individuals with PD appear to walk with muscle activation patterns that are more rhythmic than those seen in young, able-bodied individuals. Specifically, their muscle activation patterns exhibit more stride-to-stride consistency and are easier to describe with a basic burst model of recurring muscle activation. According to the optimal variability concept[32], this behaviour is characteristic of a rigid

control system, which is less able to cope with perturbations than a more flexible system. One possible explanation for this is that PD gait is more heavily influenced by spinal locomotor control with diminished control elements from supraspinal centres.

Competing Interests

The authors have declared that no competing interests exist.

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