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ORIGINAL RESEARCH ARTICLE

Effects of Treadmill Training with Load on Gait in Parkinson Disease

A Randomized Controlled Clinical Trial

ABSTRACT

Trigueiro LCL, Gama GL, Simão CR, Sousa AVC, Godeiro Júnior CO, Lindquist AR: Effects of treadmill training with load on gait in Parkinson disease: a randomized controlled clinical trial. Am J Phys Med Rehabil 2015;94:830–837.

Objective: The aim of this study was to assess the effects of 3% of body weight loads (0%, 5%, and 10%) on treadmill gait training in subjects with Parkinson disease.

Design: This study used a randomized controlled single-blind trial.

Results: Spatiotemporal variables demonstrated significant intragroup alterations in all three groups at pretraining and posttraining, with an increase in speed (m/sec; $F = 4.73$, $P = 0.04$), stride length (m; $F = 12.00$, $P = 0.002$), and step length (m; $F = 16.16$, $P = 0.001$) and a decrease in the double-stance phase (%; $F = 6.59$, $P = 0.02$) and stance phase (%; $F = 4.77$, $P = 0.04$). Intergroup pretraining and posttraining did not exhibit significant differences ($F < 2.18$, $P > 0.14$). Angular variables showed significant intragroup alterations, with an increase in knee range of motion ($F = 5.18$, $P = 0.03$), and intergroup comparison revealed no significant changes ($F = 1.87$, $P = 0.17$).

Conclusions: Posttraining improvements in speed, stride length, step length, double-stance, stance phase, and knee range of motion were observed in all groups, where no load (0%, 5%, or 10%) had any significant effect, suggesting that the influence of load did not make one experimental condition better than another. All participants benefitted from treadmill gait training, irrespective of the use of load.

Key Words: Parkinson Disease, Physical Therapy Modalities, Treadmill Test, Gait Disorders

Although the causes of Parkinson disease (PD) are still being investigated, its neurochemical aspects define it as a neurodegenerative syndrome caused by the progressive neuronal depletion of ventrolateral cells from the compact part of the substantia nigra in the mesencephalon, responsible for dopamine production. Dopamine deficiency interferes with neural conduction between the substantia nigra and the striate body, inducing alterations in motor coordination, cognition, and autonomic function.[1] A reduction in dopamine provokes changes in the execution and regulation of voluntary and automatic movements such as gait, one of the most incapacitating symptoms in PD, reducing muscle strength.[2]

Many subjects with PD develop motor complications that cannot be adequately controlled by medication. These complications arise during the off-phase of antiparkinson medication, with dyskenesis and worsened gait, which promote reduced mobility and increased risk for falls.[3] Evidence shows that the use of external cues (visual, auditory, and/or proprioceptive) associated with gait training improves walking in subjects with PD, optimizing learning capacity and motor retention.[4] Activation by external cues of some intact neuronal circuits in the premotor cortex compensates for the dopamine deficiency in the pathways between the basal nuclei and supplementary motor area, thereby maintaining gait automaticity.[5]

Treadmill walking training can function as an external cue, imposing rhythmicity and requiring the individual's attention.[6] Treadmill training with partial body weight support has proven to be an efficient technique in subjects affected by stroke[7] and spinal cord lesion.[8] However, a study by Dietz et al.[9] showed that partial body weight support in PD causes a reduction in electromyographic activity in the gastrocnemius and a decrease in ankle range of motion (ROM), owing to the decline in proprioceptive stimuli from foot contact on the treadmill. Furthermore, the sensitivity threshold of leg extensor muscle receptors is lower in PD and is directly affected by partial body weight support. These impairments decrease propulsion, stride length, and walking speed.[9]

Studies with healthy subjects[10,11] investigated the effects of treadmill training with increased load on the sensitivity threshold of these receptors. The results demonstrate increased reflex and electromyographic activity in the leg extensor muscles, indicating that additional load is related to maintaining stance. In this regard, Toole et al.[12] assessed the effects of treadmill walking with 5% of body weight load on parkinsonian subjects, observing gains in motor function, walking speed, stance time in the gait cycle, knee extension, and dorsiflexion. Similarly, Filippin et al.[13] compared treadmill walking with 10% of body weight load to conventional group therapy, obtaining improved motor function and nonmotor aspects related to quality of life in subjects with PD after treadmill intervention with the load. Treadmill with additional load leads to gains and can benefit gait functionality in these subjects; however, only one study[12] evaluated gait kinematic variables.

There is no consensus in the literature as to ideal load percentage for this type of training in PD subjects, and it is not known whether there is a real difference in the effects of different load percentages (5% and 10%) because the studies[12,13] used different methods. Toole et al.[12] and Filippin et al.[13] investigated the effects of different loads associated with treadmill training, based on the results obtained by Dietz et al.[9] with PD patients and in studies using healthy subjects[10,11] suggesting that the use of load in PD improves proprioception and gait biomechanics.

The use of 5% and 10% of loads produced positive results, but it is important to determine which of the following experimental conditions is more effective: treadmill alone, treadmill walking with 5% of load, or treadmill walking with 10% of body weight load. The authors hypothesized that 10% of body weight load would promote gains in a larger number of spatiotemporal, angular, and motor function variables than the other two conditions (0% and 5% of load).

METHODS

Design

This is a randomized controlled single-blind trial involving subjects with PD, diagnosed by a neurologist specialized in movement disorders, according to U.K. Parkinson's Disease Brain Bank Criteria.

Participants

Thirty-five subjects with PD were enrolled in the trial based on the following inclusion criteria: idiopathic PD; men or women aged between 40 and 75 yrs; between stages 2 and 3, according to the Modified Hoehn and Yahr Scale; regular use of antiparkinson drugs; ability to walk independently without any type of orthosis; absence of other types of neurologic, musculoskeletal, cardiovascular, and/or respiratory disorders; absence of visual and/or auditory deficits that could pose a risk and interfere with training; absence of serious cognitive impairments (Brazilian Mini Mental State Examination)[14]—scores greater than 13 for subjects with no schooling, 18 for low/medium educational levels (4–8 yrs), and 26 for

high educational levels (more than 8 yrs); and no previous stereotaxic surgery. All the participants gave their informed consent.

The exclusion criteria were as follows: subjects reporting hypertensive peaks with values greater than the systolic and diastolic (200 and 110 mm Hg, respectively), before and/or after training[15]; heart rate (HR) greater than submaximal values (75% of HR_{max}), calculated by the formula [$HR_{submax} = 0.75 \times (220 - age)$][16]; alterations in the dosage and/or type of antiparkinson drug during the study period; and presence of marked pain and/or muscle fatigue that would impede exercise.

The study adhered to recommendations established by Consolidated Standards of Reporting Trials (CONSORT), and sample distribution will be presented later (supplement table, http://links.lww.com/PHM/A90).

Randomization

The sample was randomly divided into three groups: treadmill alone (control group [CG]), treadmill with 5% of load (experimental group 5% [$EG_{5\%}$]), and treadmill with 10% of load (experimental group 10% [$EG_{10\%}$]). During the intervention, eight subjects dropped out because of problems with transport. Twenty-seven subjects (CG, 9; $EG_{5\%}$, 9; and $EG_{10\%}$, 9) concluded the training period and were analyzed. Participants were allocated to groups, using a simple draw and allocation concealment. The first researcher was responsible for random selection, the second for was responsible training participants, and the third was responsible for statistical analysis. Ethical principles with respect to clinical research involving human beings adhered to those proposed by the Declaration of Helsinki, and the study was approved by the institutional ethics committee. All participants gave their written informed consent.

Measurement Instruments

Participants were assessed in the pretraining and posttraining phases during the on-phase of antiparkinson medication. Initially, clinical (age, sex, disease duration, current history, previous treatment, pharmacologic treatment, etc.), demographic (life habits, schooling, etc.), and anthropometric (height and weight) data were collected, and the following instruments were applied: the Modified Hoehn and Yahr Scale, Mini Mental State Examination, and activities of daily living and motor examination domains from the Unified Parkinson's Disease Rating Scale (UPDRS). The standard criterion measures of PD severity included both the Hoehn and Yahr and the Movement Disorders Society UPDRS. Higher scores in the Modified Hoehn and Yahr Scale and UPDRS indicate significant impairment. The most affected lower limb was determined by testing rigidity and leg agility (both belonging to the motor examination domain of UPDRS), bilaterally.

Gait Analysis

Kinematic analysis was obtained using the Qualisys Motion Capture Systems 3-dimensional computerized motion analyzer (Qualisys Medical AB, Gothenburg, Sweden), and eight cameras were used in the study (Qualisys Oqus 300), with capture frequency of 120 Hz. Two types of passive markers were used: anatomic and tracking markers (rigid rectangular-based clusters). Prediction error and maximum residual parameters were determined at 15 and 5 mm, according to marker diameter.

Anatomic markers identified segment length and location of articular axes. These were placed on the highest portion of the iliac crest, greater trochanter of the femur, medial and lateral condyle of the femur, medial and lateral malleolus of the ankle, calcaneus, and head of the first and fifth metatarsal bones. The rigid rectangular-based clusters identify the trajectory of each segment during movement (pelvis, thigh, and leg), and each cluster contained four tracking markers. The pelvis cluster was placed at the base of the sacrum between the posterior-superior illiac spines; the thigh and leg clusters were fixed to the mid-third of the lateral surface of each of these segments. In the ankle-foot complex, three reference markers were used: lateral malleolus, calcaneus, and fifth metatarsus.[17]

After this procedure, a static collection was made to identify segments. Calibration was performed, and participants remained in an upright (orthostatic) position for 3 secs. Next, anatomic markers were removed, but tracking markers remained for dynamic collections, where participants were asked to walk along an 8-m-long walkway at self-determined speed, that is, at a comfortable and safe pace,[18,19] during ten repetitions. All participants wore their usual footwear.

Study Protocol and Training Procedure

Participants were followed for 4 consecutive wks (three 30-min sessions per week) during the on-phase of antiparkinson medication, as in the assessment protocol. The groups with added load were fitted with a belt containing weight pockets (Seasub, Brazil) around their waist because of the proximity to the body's center of mass.[12,13] The load consisting of 1-kg lead weights was calculated according to percentage body weight (5% for $EG_{5\%}$ and 10% for $EG_{10\%}$). Participants were not allowed to participate in other rehabilitation protocols or use the treadmill as physical activity during the training period.

Gait training was carried out on the GaitTrainer System 2 (Biodex Medical Systems Inc, Shirley, NY). In the first session, participants were allowed a 5-min adaptation period to familiarize themselves with the equipment. After adaptation and at the start of each new session, an attempt was made to gradually increase speed. Participants were instructed to walk at their maximum tolerated speed,[13,18,19] and the physiotherapist in charge monitored posture, body alignment, presence of fatigue, and increased submaximal heart rate, that is, 75% of HR_{max} [$HR_{submax} = 0.75 \times (220 - \text{age})$]. If necessary, manual and verbal feedback were given for correction purposes, and participants were allowed to use upper extremity support during gait training. Posttraining assessment was performed the day after the last training session, by applying the UPDRS and analyzing kinematic gait.

Data Reduction

Data were captured with Qualisys Track Manager 2.6 software and exported to Visual3D system (Visual 3D Standard; C-Motion). This system is responsible for constructing 3-dimensional representations of the biomechanic model and assessment of kinematic gait variables (spatiotemporal and angular variation). A low-pass filter with a cutoff frequency of 6 Hz was applied to eliminate noise from marker trajectories.[20] Each angular displacement is obtained by associating segments with a system of coordinates that uses the Cardan angle sequence. The reference or orthostatic position adopted in this study was considered the neutral position.

Hip, knee, and ankle angulation curves were presented in percentages, during the gait cycle (0%–100%). To delimit the start and end of the cycle, two consecutive initial contact events of the most affected lower limb were identified. The events were defined based on the graphic representations of these markers on the y-axis.[21] The following spatial and temporal gait variables were investigated: speed (m/sec), stride length (m), most affected step length (m), doublestance (%), total stance (%), and total swing (%) phases. Angular variables investigated were related to the most affected lower limb displacements in the sagittal plane. The authors evaluated hip, knee, and ankle ROM (degrees).

Statistical Analysis

Analysis was performed with Statistical Package for the Social Sciences 17.0 (SPSS Inc, Chicago, IL) at 5% of significance level. Data normality of all study variables was determined by the Shapiro-Wilk test. Next, a one-way analysis of variance (ANOVA) was applied to clinical, demographic, and anthropometric variables to evaluate whether there were any imbalances in the distribution of baseline characteristics between the three groups, and Levene test was used to verify intergroup homogeneity of variance. Mixed effects ANOVA (3×2) with repeated measures was used to identify intergroup differences in spatiotemporal, angular, activities of daily living, and motor function domains (presented in the UPDRS) at pretraining and posttraining conditions. The post hoc Bonferroni test was applied to detect which pairs of groups differ.

RESULTS

Characteristics of the Subjects

The sample was composed of 27 subjects (18 men and 9 women), aged between 41 and 75 yrs (62.26 ± 9.07), and disease duration was between 2 and 9 yrs (4.56 ± 2.42). It was found that the right lower limb was the most affected in 74% of the sample. Table 1 shows the characteristics of each group in the training condition. No statistically significant intergroup differences were observed ($P > 0.60$) in the pretraining phase (see Table 1).

The speed (m/sec) adopted by the participants during the training period showed no intergroup differences ($P > 0.32$) in the initial, intermediate, and final phases of training (see Table 2).

The clinical-functional assessment of participants showed significant intragroup differences with respect to activities of daily living ($F = 5.49$, $P = 0.03$), showing a decrease in all groups (CG, 18.22 ± 7.48 *vs*. 17.00 ± 6.75; $EG_{5\%}$, 16.56 ± 7.05 *vs*. 15.89 ± 7.46; $EG_{10\%}$, 15.78 ± 5.89 *vs*. 15.67 ± 5.12); however, no intergroup differences were observed ($F = 1.27$, $P = 0.30$). All groups showed significant intragroup differences ($F = 11.13$, $P = 0.003$) with a reduction in the motor examination domain (CG, 18.67 ± 12.66 *vs*. 13.11 ± 8.99; $EG_{5\%}$, 25.89 ± 14.95 *vs*. 21.00 ± 14.02; $EG_{10\%}$, 16.22 ± 12.62 *vs*. 11.11 ± 6.29), but no intergroup differences were found ($F = 0.02$, $P = 0.98$).

Spatiotemporal Variables

In regard to spatiotemporal variables (see Table 3), significant intragroup differences were observed in all three groups at pretraining and posttraining with an increase in speed (m/sec; $F = 4.73$, $P = 0.04$), stride length (m; $F = 12.00$, $P = 0.002$), and step length (m; $F = 16.16$, $P = 0.001$) and a decrease in the double-stance phase (%; $F = 6.59$, $P = 0.02$) and total stance phase (%; $F = 4.77$, $P = 0.04$). Intergroup pretraining and posttraining showed no significant differences in speed (m; $F = 0.25$, $P = 0.78$), stride length (m; $F = 0.57$, $P = 0.57$), step length (m; $F = 2.18$, $P = 0.14$), double-stance phase (%; $F = 0.23$, $P = 0.80$), and total

TABLE 1 Baseline clinical and demographic characteristics of the subjects

Variables	Treadmill (Control)	Treadmill + 5% (Experimental 1)	Treadmill + 10% (Experimental 2)	*P*
Age, yrs	61.89 ± 6.79	61.44 ± 11.91	63.44 ± 8.79	0.89
Height, cm	161.33 ± 10.49	158.56 ± 7.45	168.44 ± 7.33	0.06
Weight, kg	61.44 ± 10.83	61.89 ± 11.89	68.00 ± 11.78	0.42
Duration of PD, yrs	4.78 ± 2.59	4.44 ± 2.83	4.44 ± 2.07	0.95
HY	2.61 ± 0.33	2.56 ± 0.46	2.56 ± 0.39	0.94
MMSE	25.78 ± 2.99	27.56 ± 2.51	26.00 ± 4.82	0.53
$UPDRS_{II}$	18.22 ± 7.48	16.56 ± 7.06	15.78 ± 5.89	0.74
$UPDRS_{III}$	18.67 ± 12.66	25.89 ± 14.94	16.22 ± 12.62	0.30

Values are expressed as mean ± standard deviation.

HY, Hoehn and Yahr; MMSE, Mini Mental State Examination; $UPDRS_{II}$, UPDRS-activities of daily living domain; $UPDRS_{III}$, UPDRS-motor examination domain.

stance phase (%; $F = 0.81$, $P = 0.46$). None of the three groups exhibited significant intragroup or intergroup changes in total swing (%; $F = 3.67$, $P = 0.07$ and $F = 12.46$, $P = 0.64$, respectively).

Angular Variables

Assessment of angular variables (see Table 3) demonstrated significant intragroup alterations in knee ROM (degrees; $F = 5.18$, $P = 0.03$) in all three groups. The other variables showed no significant difference (hip ROM [degrees], $F = 3.87$, $P = 0.07$; ankle ROM [degrees], $F = 0.002$, $P = 0.72$). Intergroup comparison between the pretraining and posttraining phases revealed no significant changes in hip ROM (degrees; $F = 0.52$, $P = 0.59$), knee ROM (degrees; $F = 1.87$, $P = 0.17$), or ankle ROM (degrees; $F = 0.34$, $P = 0.72$).

Figure 1 exhibits mean angular displacements of the hip (degrees), knee (degrees), and ankle (degrees) in all the groups, before and after training, with respect to the most affected lower limb.

DISCUSSION

The results of this study demonstrate that treadmill training, irrespective of the use of load, improved activities of daily living and motor function in all participants. Studies have reported[12,13] that treadmill gait training leads to a reduction in UPDRS scores—considered the gold standard for measuring the severity of PD symptoms—which represents an improvement in motor function and mobility. However, the beneficial effects of the treadmill for subjects with PD are irrespective of the use or not of load but rather to an increase in cortical excitability, reinforcing the idea that the treadmill can stimulate neuroplasticity of the central nervous system.[22]

The treadmill, as repetitive and task-specific gait training, is able to improve locomotor pattern, as shown by the findings of this study, which observed motor gains in all groups assessed after treadmill training. It is suggested that the treadmill acts as a neuroprotective element that causes positive changes in gait pattern, mobility, and quality of life,[2] thereby enhancing motor function and activities of daily living and not merely providing a simple relief of symptoms.

Spatiotemporal and Angular Variables

Assessment of spatiotemporal variables demonstrated intragroup variations in all three conditions (CG, $EG_{5\%}$, and $EG_{10\%}$) with increased speed,

TABLE 2 Analysis of the behavior of the treadmill speed in the first (S_1), the sixth (S_6), and the last (S_{12}) sessions

	Intervention				
Variable	Treadmill (Control)	Treadmill + 5% (Experimental 1)	Treadmill + 10% (Experimental 2)	*P*	*F*
S_1, m/sec	0.37 ± 0.24	0.27 ± 0.12	0.31 ± 0.19	0.57	0.57
S_6, m/sec	0.58 ± 0.31	0.41 ± 0.22	0.57 ± 0.25	0.32	1.19
S_{12}, m/sec	0.71 ± 0.35	0.57 ± 0.24	0.82 ± 0.44	0.34	1.14

Values are expressed as mean ± standard deviation.

S_1, speed of the first session (initial phase of training); S_6, speed of the sixth session (intermediate phase of training); S_{12}, speed of the 12th session (final phase of training).

TABLE 3 Behaviour analysis of spatiotemporal and angular variables in pretraining and posttraining phases

Variables	Treadmill (Control)		Treadmill + 5% (Experimental 1)		Treadmill + 10% (Experimental 2)		95% Confidence Interval
	Pretraining	Posttraining	Pretraining	Posttraining	Pretraining	Posttraining	Lower Bound–Upper Bound
Speed, m/sec	0.99 ± 0.13	1.05 ± 0.09[a,b]	0.76 ± 0.17	0.85 ± 0.20[a,b]	0.85 ± 0.20	1.04 ± 0.16[a,b]	0.90–1.00
Stride length, m	1.11 ± 0.11	1.15 ± 0.09[b,c]	0.93 ± 0.18	1.01 ± 0.17[b,c]	1.19 ± 0.04	1.24 ± 0.12[b,c]	1.06–1.15
MA step length, m	0.56 ± 0.04	0.57 ± 0.06[b,c]	0.45 ± 0.09	0.51 ± 0.08[b,c]	0.58 ± 0.03	0.61 ± 0.07[b,c]	0.53–0.57
Double-stance, %	15.99 ± 1.77	14.66 ± 1.73[a,b]	17.62 ± 1.91	16.44 ± 2.04[a,b]	15.62 ± 2.51	14.95 ± 1.86[a,b]	15.21–16.54
Total stance, %	66.09 ± 1.53	64.91 ± 2.05[a,b]	68.45 ± 1.71	66.27 ± 1.57[a,b]	70.11 ± 11.13	65.08 ± 2.62[a,b]	65.40–68.23
Total swing, %	33.91 ± 1.53	34.98 ± 1.98	31.55 ± 1.71	33.73 ± 1.57	30.89 ± 1.86	34.92 ± 2.62	31.96–34.70
ROM of hip, degrees	41.65 ± 4.47	42.20 ± 6.04	30.23 ± 5.54	31.74 ± 5.64	37.36 ± 3.55	39.82 ± 5.65	35.25–39.09
ROM of knee, degrees	58.06 ± 3.46	58.85 ± 4.05[a,b]	51.72 ± 9.70	55.41 ± 12.91[a,b]	59.49 ± 4.85	60.04 ± 2.10[a,b]	54.48–60.05
ROM of ankle, degrees	22.27 ± 4.07	21.74 ± 5.14	18.56 ± 4.58	18.46 ± 3.46	20.75 ± 3.81	21.46 ± 1.77	19.11–21.97

Values are expressed as mean ± standard deviation.
MA, most affected.

stride length, and step length and a decrease in the double-stance and stance phases. Thus, treadmill training, with or without load, contributed to all participants achieving the primary goals of gait rehabilitation in PD, that is, to improve walking speed and stride length.[23] These results are similar to those of Toole et al.,[12] who obtained gains in speed and stride length as well as reduced left lower limb stance.

The changes promoted by treadmill training depend much more on alterations in central pattern generator function and/or efferent commands than on the subject's attention during training or local peripheral sensory stimulation (spinal cord) or superior centers.[24] In this study, treadmill training with additional load apparently did not influence posttraining improvement in spatiotemporal variables, because control group participants also obtained gains, suggesting that neuromuscular regulation in PD can be stimulated by the treadmill alone,[12] with no need for additional load. It is noteworthy that, during treadmill exercise, subjects with PD tend to expend around 20% more metabolic energy than their healthy counterparts.[25] Thus, the addition of this type of load would require training with greater physical effort, without being able, however, to promote a significant improvement in motor function, activities of daily living, or mobility.

Step and stride length are correlate measures, which may justify the improvement in these two variables in all groups. Treadmill walking, compared with overground, accentuates hip extension, thereby lengthening step. Subjects develop the ability to progressively adapt to the treadmill, according to the increased demand established, contributing to motor control automaticity.[2,19] Furthermore, the treadmill facilitates the stretching of hip flexors and ankle dorsiflexors at the end of the stance phase, resulting in an increase in stride length, reduction in total stance time, and increase in swing time, thereby augmenting impulsion.[26]

The treadmill speed used during the intervention did not vary between groups, and all individuals exhibited an improvement in ground speed after the treatment protocol, regardless of the use or not of load. During overground walking, the individual experiences speed fluctuations during the entire training period. However, in treadmill training, subjects must keep their feet on a movable belt, and walking speed must follow treadmill speed, that is, individuals must constantly modify their pace. As a consequence, gait speed becomes more uniform and regular.[2] The mechanism of the treadmill alone would explain why all the individuals in the present study increased their speed after training.

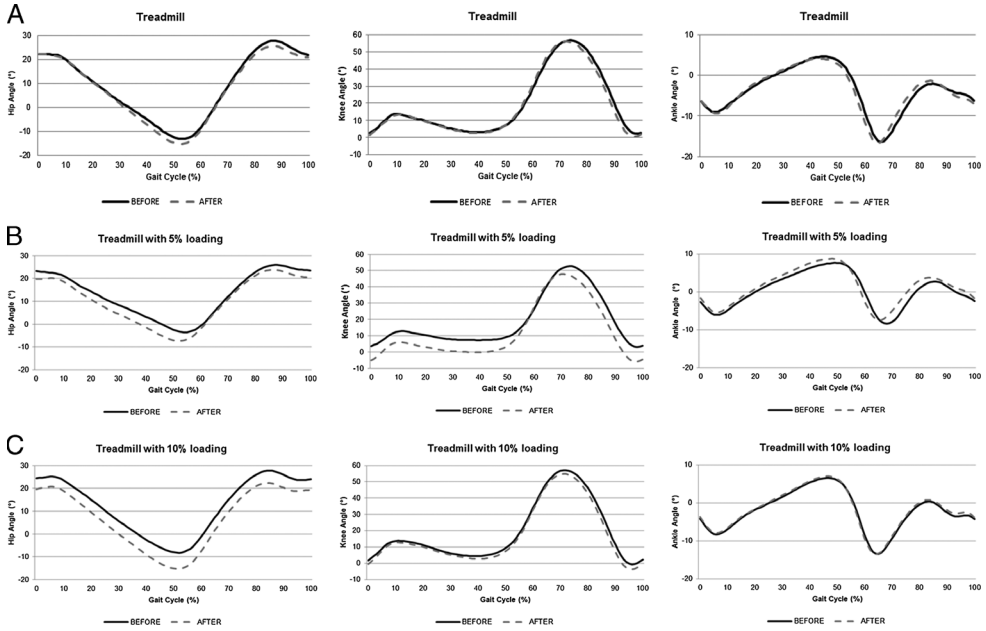

FIGURE 1 *Positive signs indicate hip and knee flexion and ankle dorsiflexion, and negative signs indicate hip and knee extension and ankle plantar flexion. Hip angle (A), knee angle (B), and ankle angle (C) in all groups—treadmill, treadmill with 5% of load, and treadmill with 10% of load.*

Rhythmicity and speed, as well as the repeated movement of the treadmill, may also increase stride length. The ability to achieve a normal stride length pattern seems not to be completely lost in PD and, in contrast to other neurologic pathologies, can be restored through the use of external cues.[27] Schlick et al.[28] reported that the use of external markers such as a rehabilitation strategy may decrease the need for internal planning and organization of movements, reducing cognitive overload. In this respect, the treadmill functions as an external marker, such that it imposes rhythmicity and requires the individual to concentrate during the execution of complex motor tasks such as gait.

Analysis of angular displacements showed intragroup alterations in all three groups in knee ROM only. Similarly, Toole et al.[12] observed improvements in only two variables (ankle dorsiflexion and knee extension, both with respect to the left lower limb) of the ten parameters selected for kinematic analysis, after 6 wks of treadmill training. The gait gain observed may simply be related to that the treadmill demands the subject's attention while walking, irrespective of the use of load or partial body weight support. Another more likely cause for the gain obtained seems to be related with aspects of motor control that are not based on attention.

Treadmill training is characterized by intense, repetitive, task-specific physical activity that provides continuous feedback. The transition of gains obtained on the treadmill to the ground in this study confirms one of the concepts of motor learning, in which learning and retention of a given task are favored if it is performed repeatedly, enhancing automatic motor control. One of the principles that guide the rehabilitation of neurologic patients is that they must practice a skill to improve it.[29]

The hypothesis proposed in the present study was not confirmed, because participants from the three groups obtained positive responses in kinematic variables as well as in activities of daily living and motor function domains at the end of training. Total training time (4 wks) or frequency of training sessions might account for these findings. However, the authors emphasize that, in both studies by Toole et al.[12] and Filippin et al.,[13] subjects underwent three sessions per week. It is important to underscore that the parameters used in the present study comply with the criteria proposed in the meta-analysis performed by Mehrholz et al.,[30] which suggests a mean of 4–8 wks of treadmill training, with one-to-four sessions per week, where each session lasts from 30 to 45 mins.

Other limitations were the small sample size and lack of kinematic data on the contralateral limb. It is suggested that future studies be conducted using a prolonged intervention and follow-up to monitor the possible gains retained after the end of treatment.

CONCLUSIONS

In the present study, treadmill gait training improved activities of daily living, motor function, and spatiotemporal (speed, stride length, step length, double-stance and stance phases) and angular (knee ROM) variables in all groups, where none of the loads (0%, 5%, and 10%) had any significant effect, suggesting that the influence of load was not enough to make one experimental condition stand out from another. These findings simplify gait training in PD patients, because walking, not load, causes improvement.

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