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Persons With Multiple Sclerosis Show Altered Joint Kinetics During Walking After Participating in Elliptical Exercise

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Patients with multiple sclerosis (MS) experience abnormal gait patterns and reduced physical activity. The purpose of this study was to determine if an elliptical exercise intervention for patients with MS would change joint kinetics during gait toward healthy control values. Gait analysis was performed on patients with MS (n = 24) before and after completion of 15 sessions of supervised exercise. Joint torques and powers were calculated, while also using walking velocity as a covariate, to determine the effects of elliptical exercise on lower extremity joint kinetics during gait. Results show that elliptical exercise significantly altered joint torques at the ankle and hip and joint powers at the ankle during stance. The change in joint power at the ankle indicates that, after training, patients with MS employed a walking strategy that is more similar to that of healthy young adults. These results support the use of elliptical exercise as a gait training tool for patients with MS.

Keywords: exercise, gait analysis, kinetics, rehabilitation

Multiple sclerosis (MS) is a progressive neurological disease that affects over 400,000 Americans (National Multiple Sclerosis Society). In contrast to an acute injury event such as stroke or traumatic brain injury, patients with MS face a chronic worsening of symptoms but are often treated only during the transient relapses. As a result of axon demyelination in the brain and spinal cord, nerve fiber function is compromised and axonal conduction velocity is slowed in patients with MS (White & Dressendorfer, 2004). The lower axonal conduction velocity would result in slow voluntary muscle activation and slower rate of force development, particularly in the lower limb muscles, of patients with MS (Sharma, Kent-Braun, Mynhier, Weiner, & Miller, 1995), which causes significant problems during gait. Due to the progressive pattern of the disease, longitudinal studies using clinical-based measures reveal that 75–80% of all patients with MS (relapsing-remitting or primary progressive) will eventually require an assistive device to walk (Confavreux, Vukusic, Moreau, & Adeleine, 2000; Weinshenker et al., 1989). These studies indicate a critical need to maintain walking function of patients with MS. Unfortunately, none of the currently available disease-modifying medications have been shown to either stop or reverse gait disability in patients with MS (Lo & Triche, 2008). Ampyra (dalfampridine, formerly known as Fampridine-SR), from Acorda Therapeutics, is a symptom-modifying drug approved by the FDA in January of 2010 for its ability to improve walking speed in people with any type of MS. Ampyra is currently available by prescription (Goodman et al., 2009) but has not been shown to improve gait mechanics in patients with MS. There is also evidence, however, that patients with MS benefit from structured exercise (Dalgas, Stenager, & Ingemann-Hansen, 2008) and that walking, specifically, can be improved with exercise training (Snook & Motl, 2009). These findings indicate that exercise training may be used to help improve and to maintain walking function in patients with MS.

An elliptical exercise machine offers comparable aerobic benefits as a standard treadmill (Mercer, Dufek, & Bates, 2001), while also incorporating a closed kinetic chain movement pattern that may be more easily employed in persons with lower extremity disability. Elliptical exercise has recently become a treatment and rehabilitation device for patients who have chronic stroke (Jackson, Merriman, & Campbell, 2010), patellar tendonectomy (Shelbourne, Henne, & Gray, 2006), and patients with patellofemoral pain syndrome (Ganley, Gaugles, & Moroz, 2006) or diabetes (Cuff et al., 2003). Elliptical training requires both aerobic effort and strength because the machine is propelled by the user in a lower extremity movement pattern that is similar to normal walking (Burnfield, Shu, Buster, & Taylor, 2010). In gait

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training, an important consideration is that the limitation of voluntary control of individual muscle groups predicts overall capacity to recover walking (Beres-Jones & Harkema, 2004). For patients with MS who experience miscommunication between muscle activation and the commands provided by the brain, gait training may help to improve walking by increasing voluntary control of muscle groups. Because an exercise like elliptical training requires constant increased muscle activation to perform the activity, it could provide an alternative option to improve walking for persons with MS. In addition, elliptical training allows for use by those who are unable to safely perform treadmill walking for longer periods of time than would be necessary in a similar treadmill exercise intervention.

The purpose of this study was to evaluate the effectiveness of a six-week elliptical exercise training to alter joint kinetics during walking in patients with MS toward joint kinetics values as those found in healthy controls. The measured gait variables were joint torques and powers. Changes in joint kinetics would result from alteration in the relative contribution of individual muscle groups to the total output of the limb (DeVita & Hortobagyi, 2000). It was hypothesized that joint torques and powers would change in patients with MS following elliptical training since training could cause an increase in strength or increased activation of lower extremity muscle groups during gait. It was also hypothesized that after training, joint torque and power variables would be closer to normative values from healthy controls.

Methods

Subjects

A total of 24 patients with MS and 24 age-, height-, and weight-matched healthy controls participated in this study (Table 1). All procedures were approved by the University's Medical Center Institutional Review Board.

Inclusion criteria for the patients with MS included cognitive competency to give informed consent, as determined by an the MS clinical care professional (author MF), age ranging from 19 years to 65 years, an Expanded Disability Status Scale (EDSS; Kurtzke, 1983) score of 1 to 6.0, where all subjects were able to walk 25 feet without aid. Healthy controls were in the same age range and were free from any other neurological or orthopedic conditions that could affect their gait or balance. Patients were excluded if they had any other comorbid conditions that would make participation in exercise unsafe.

Data Collection Protocol

For all gait data collections, after providing informed consent, subjects were prepared for data collection and reflective markers were placed bilaterally according to anatomical position (Houck, Yack, & Cuddeford, 2004). Marker placement was applied by the same person (author JH) for all data collections. During each walking trial, the subject was directed to walk using a self-selected pace over a 10 m walkway while three-dimensional marker trajectories and ground reaction force data were simultaneously collected. During the stance phase (heel contact to toe-off), marker trajectories were captured with an eight camera, high-speed real-time system (EvaRT 5.0, Motion Analysis Corp., Santa Rosa, CA) sampling at 60 Hz while ground reaction force data were acquired with a Kistler force platform (Model: 9281-B11; Amherst, NY), amplified by a Kistler amplifier (Model: 9865; Amherst, NY), sampling at 600 Hz. Walking velocity was calculated directly from the marker trajectories during each walking trial. Subjects were not given feedback

Table 1 Demographic information, means (*SD*), for patients with MS and healthy controls (HC); *p*-values are listed for the independent (pretraining vs. healthy control, posttraining vs. healthy control) or paired (pre- vs. posttraining) *t* tests.

	Patients with MS (n = 24)	Healthy Controls (n = 24)	*P*-Value
Sex	5 male, 19 female	8 male, 16 female	—
Age	45.1 (10.8) years	43.7 (12.8) years	0.680
EDSS	2.5 (0.7)	—	—
Height (cm)	166.2 (6.7)	170.0 (12.4)	0.203
Mass (kg)	79.9 (15.9)	77.9 (20.5)	0.706
Walking Velocity (m/s)	1.09 (0.20) pre	1.14 (0.19)	0.176 pre vs. HC
	1.12 (0.21) post		0.503 pre vs. post
			0.549 post vs. HC

Note. EDSS = Expanded Disability Status Scale.

regarding their walking velocity and the success of a trial was not determined by the subjects walking velocity. This procedure allowed us to avoid any fatigue effects for the patients with MS. However, walking velocity was controlled post hoc as indicated below in the statistical analysis. Because only one force platform was available at the laboratory, we collected one limb at a time. Once the walkover was completed, the patient rested for a minimum of 1 min. The same process was repeated to obtain five good walkovers with each limb, in which the patient's foot landed completely within the force plate without altering the stride. The data collection procedure for the patients with MS and for the healthy controls was identical. After the elliptical training was complete, patients with MS underwent a repeat gait evaluation with an identical data collection procedure.

Data Analysis

A low-pass second-order Butterworth digital filter with a 7 Hz cutoff frequency was used to smooth the marker trajectories during postprocessing. A custom MatLab program was used to calculate the joint kinetics and kinematics of each subject. Inverse dynamics using linear and angular Newtonian equations of motion were used to calculate the joint torques (D.A. Winter, 2005). Joint power is calculated as the product of the net torque of force at a joint (M_j) and the relative joint angular velocity (ω_j), or $P_j = M_j \times \omega_j$ (joules per second or watts). Positive power indicates that energy is being generated (concentric contraction) and negative power that energy is being absorbed (eccentric contraction). Joint torques and powers were normalized with respect to the subject's body mass (DeVita & Hortobagyi, 2000). All normalization occurred after the peak values were determined so that the normalized values were used in the statistical analysis. The peak value was determined for each trial and the average peak value from five trials on each leg was used for analysis. Gait variables were identified for the ankle, knee, and hip joints during the stance phase according to other gait studies on joint kinetics (DeVita & Hortobagyi, 2000; Graf, Judge, Ounpuu, & Thelen, 2005; Kerrigan et al., 2000; D.A. Winter, Patla, Frank, & Walt, 1990). The specific joint torque variables of interest were peak ankle dorsiflexor torque during early stance (ADT), peak ankle plantar flexor torque during late stance (APT), peak knee flexor torque during stance (KFT), peak knee extensor torque during stance (KET), peak hip flexor torque during late stance (HFT), and peak hip extensor torque during early stance (HET). The specific joint power variables of interest were peak ankle power absorption in early stance (A1), peak ankle power generation in late stance (A2), peak knee power absorption in early stance (K1), peak knee power generation in midstance (K2), peak knee power absorption in late stance (K3), peak hip power generation in early stance (H1), peak hip power absorption in late midstance (H2), peak hip power generation in late stance (H3).

Exercise Training Protocol

Twenty-four patients with MS completed 15 sessions of elliptical exercise training. All patients with MS were naïve to exercise with the elliptical machine and none of the subjects were involved in any structured exercise program in the three months preceding the study. The elliptical machines (Precor EFX 546 Elliptical trainer) were housed in a section of the cardio workout room in the recreation facility on the University of Nebraska at Omaha campus. Every patient was supervised for each training session on the elliptical machine and heart rate was monitored via a commercially available heart rate monitor (Polar Electro Inc., Lake Success, NY). The exercise supervisor made sure each patient used the machine without leaning on the side rails for support but no other specific instructions were provided regarding how the patients needed to perform the exercise. Each session consisted of 30 min of training on the elliptical trainer. This time period for this exercise intervention (15 sessions, 2–3 times per week over 6 weeks) was within the range of time periods previously used for resistance and aerobic training in patients with MS (Gutierrez et al., 2005; Newman et al., 2007; Oken et al., 2004; Petajan et al., 1996).

At the first exercise session, patients initiated exercise at their own self-selected pace with the instruction that they would need to complete 30 min of exercise and could take as many breaks as necessary. The cadence or rotations per minute (RPMs) for the first session was based on the patients' self-selected pace. All patients started the exercise session with the elliptical set at an incline of zero (no incline) and a resistance of 1 (lowest possible resistance). All initial starting values (RPM, heart rate) were recorded as a baseline picture of subject fitness. Progression of exercise intensity was achieved by increasing the resistance level of the elliptical machine and/or by increasing the patient's RPM (stepping speed was controlled by the patient and not the elliptical machine). Initiating the progression of exercise intensity was based on the patient's age-predicted heart rate maximum (HRM) measured with the heart rate monitor (Polar Electro Inc., Lake Success, NY). The first exercise session's intensity was dictated by the patient's level of motivation, ability to perform, spasticity, and fitness level. The heart rate during exercise session was continually monitored in a log and tracked as a percentage of the age-predicted HRM to ensure that the patient was progressing in exercise intensity as fitness level improved. Increases in intensity (either machine resistance or machine RPM) were made approximately every three exercise sessions.

Statistical Analysis

Linear mixed effects modeling was used to evaluate differences in mean gait parameters between groups (control, MS at baseline, and MS after intervention). Because changes in velocity will have an impact on joint kinetics, both Group and Velocity effects, as well as the interaction between Group and Velocity, were included

in the model as fixed effects. A significant Group effect indicates a significant difference in the variable value according to group (control vs. MS at baseline vs. MS after intervention). If a significant Group effect was found, a post hoc paired test was performed. By treating velocity as a fixed effect in the model, we were able to identify changes that were specific to group (MS pre vs. post vs. controls) and changes that were the result of velocity differences between the groups. Random effects were included in the model to account for the correlation due to limbs (within subject) and for correlation between baseline and post intervention conditions within subjects with MS. A compound symmetry covariance structure was used to model the random components. Tukey's method was used to adjust for multiple comparisons and statistical significance was set at the 0.05 alpha level. Statistical analyses were performed using SAS version 9.2.

Results

Twenty-four subject with MS (45.1 ± 10.8 years) completed the training protocol and were matched with 24 healthy control subjects (43.7 ± 12.8) according to age ($p = .680$), height ($p = .203$), and weight ($p = .706$). Walking velocity was not different between subjects with MS and healthy controls before ($p = .176$) or after ($p = .549$) subjects with MS completed the training protocol (Table 1).

For all joint torque and joint power variables, differences listed as significant indicate that the p-value for the comparison was less than 0.05 before and after the Tukey correction for multiple comparisons was applied. Differences listed as marginally significant refer to the p-value after the Tukey correction being close to 0.05 but not less than 0.05. Before Tukey's correction, the marginally significant variables had a p-value of less than 0.05.

There was a significant Group effect ($F = 3.81_{2, 53.8}$; $p = .028$) for peak ankle dorsiflexor torque (ADT). Paired tests showed that both pretraining ($t = 2.66$, $p = .032$) and posttraining ($t = 2.75$, $p = .026$), ADT for subjects with MS was significantly lower compared with controls (Table 2; Figure 1). There was a significant ($F = 7.19_{2, 80.7}$; $p = .001$) Group effect for peak ankle plantar flexor torque (APT). Paired tests showed that pretraining, APT in the subjects with MS was significantly ($t = 3.71$, $p = .001$) lower than controls and pre- to posttraining, there was a marginally significant ($t = 2.06$, $p = .103$) increase in APT in the subjects with MS. Posttraining, the APT for the subjects with MS was marginally significantly ($t = 2.35$, $p = .058$) lower than controls (Table 2; Figure 1, top). There was a significant ($F = 5.39_{2, 80.4}$; $p = .006$) Group effect for peak knee extensor torque (KET). Paired tests showed both pretraining ($t = 3.26$, $p = .006$) and posttraining ($t = 3.11$, $p = .008$), KET in subjects with MS was significantly lower than controls (Table 2; Figure 1, center). There was a significant ($F = 3.80_{2, 77.9}$; $p = .027$) Group effect for peak hip extensor torque (HET). Paired tests showed pretraining, HET in the subjects with MS was marginally significantly ($t = 2.13$, $p = .095$) lower than controls and pre- to posttraining, there was a marginally significant ($t = 2.27$, $p = .065$) increase in HET in the subjects with MS. Posttraining, HET in the subjects with MS was not significantly different from controls. There was a significant ($F = 4.44_{2, 76.7}$; $p = .015$) Group effect for peak hip flexor torque during late stance (HFT). Paired tests showed pretraining ($t = 2.36$, $p = .056$) and posttraining ($t = 2.98$, $p = .012$), HFT in subjects with MS was marginally significantly lower than controls (Table 2; Figure 1, bottom).

There was a significant Group effect ($F = 3.98_{2,75.5}$; $p = .023$) for peak ankle power absorption during early stance (A1). Paired tests showed pretraining, A1 in the subjects with MS was significantly ($t = 2.01$, $p = .012$) lower than controls and pre- to posttraining, there

Table 2 Mean (*SD*) joint torque values (N·m/kg) for patients with MS pre- and posttraining and healthy controls (HC)

					***P*-Value**		
	MS Pretraining	**MS Posttraining**	**Healthy Controls**	***P*-Value (Group Effect)**	**MS Pre vs. HC**	**MS Pre vs. MS Post**	**MS Post vs. HC**
ADT	–0.277 (0.069)	–0.282 (0.078)	–0.350 (0.113)	**0.028§**	**0.032***	0.977	**0.026***
APT	1.215 (0.137)	1.265 (0.123)	1.354 (0.153)	**0.001§**	**0.001***	**0.103****	**0.058****
KET	0.514 (0.190)	0.534 (0.209)	0.697 (0.207)	**0.006§**	**0.006***	0.933	**0.008***
KFT	–0.269 (0.131)	–0.282 (0.162)	–0.218 (0.136)	0.214	0.326	0.822	0.188
HET	0.627 (0.186)	0.689 (0.141)	0.739 (0.207)	**0.027§**	**0.095****	**0.065****	0.600
HFT	–0.776 (0.186)	–0.459 (0.184)	–0.899 (0.238)	**0.015§**	**0.056****	0.697	**0.012***

§Significant group effect, $p < .05$.

*Significant group difference, $p < .05$, after Tukey's adjustment for multiple comparisons.

**Marginally significant group difference, $p < .05$, after Tukey's adjustment for multiple comparisons.

Note. ADT—peak ankle dorsiflexor torque during early stance; APT—peak ankle plantar flexor torque during late stance; KFT—peak knee flexor torque during stance; KET—peak knee extensor torque during stance; HFT—peak hip flexor torque during late stance; HET—peak hip extensor torque during early stance.

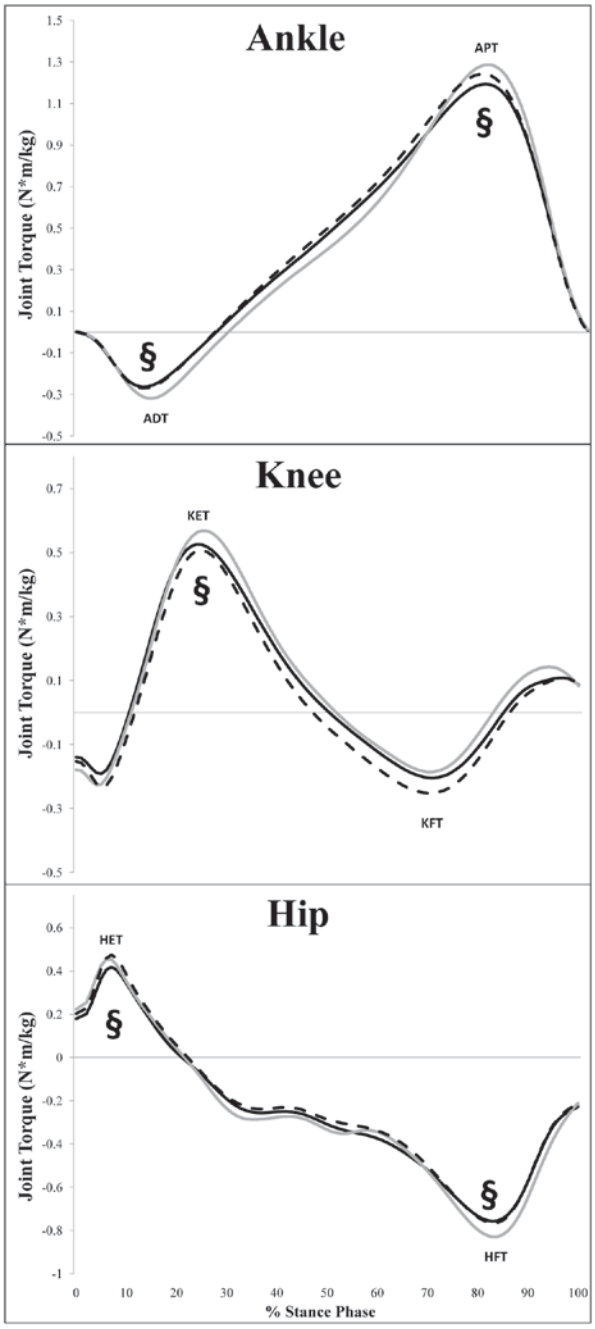

Figure 1 — Joint torque mean ensemble curves for healthy controls (gray line), pretraining MS patients (black line), and posttraining MS patients (dashed black line) at normal walking velocity (Table 3). §Significant group effect, $p < 0.05$.

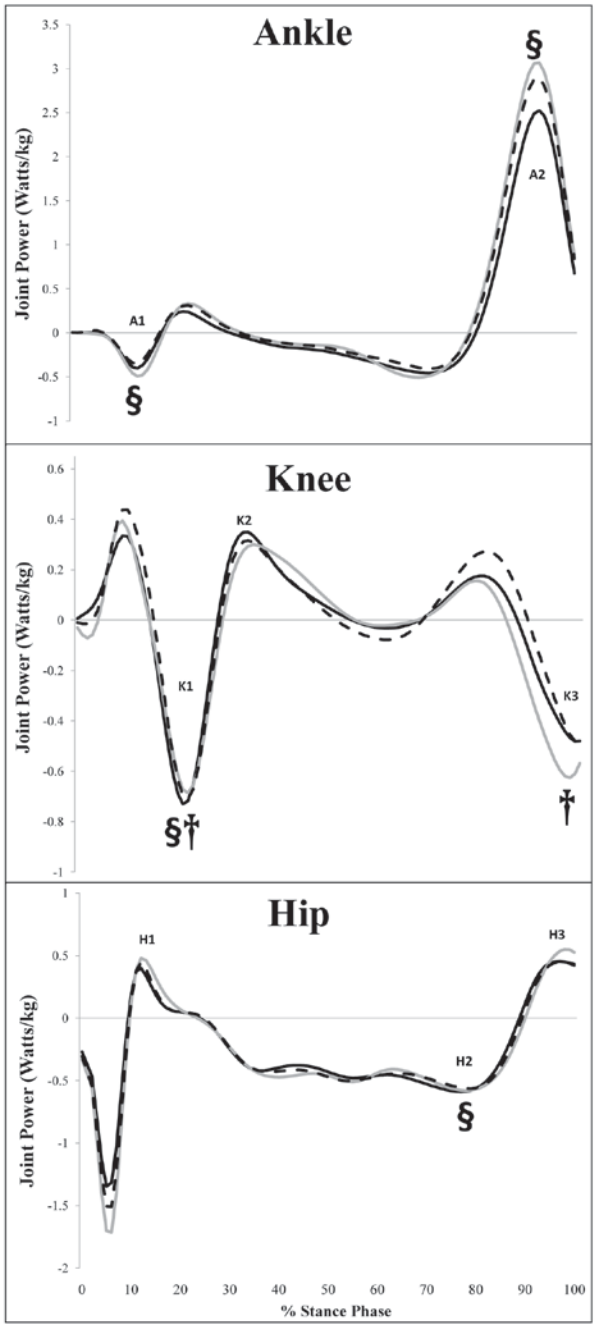

Figure 2 — Joint power mean ensemble curves for healthy controls (gray line), pretraining MS patients (black line), and posttraining MS patients (dashed black line) at normal walking velocity (Table 3). §Significant group effect, $p < 0.05$. †Significant interaction: group × velocity, $p < 0.05$.

was a significant (t = 2.38, p = .050) increase in A1 for subjects with MS. Posttraining A1 in the subjects with MS was not different from controls. There was a significant Group effect ($F = 4.72_{2,\ 76.6}$; p = .023) for peak ankle power generation during late stance (A2). Paired tests showed pretraining, A2 in the subjects with MS was marginally significantly (t = 2.39, p = .054) lower than controls and pre- to posttraining, there was a significant (t = 2.41, p = .047) increase in A2 for subjects with MS. Posttraining, A2 in the subjects with MS was not different from controls (Table 3; Figure 2, top).

Table 3 Mean (*SD*) joint power values (W/kg) for patients with MS pre- and posttraining and healthy controls (HC)

					P-Value		
	MS Pretraining	MS Posttraining	Healthy Control	*P*-Value (Group Effect)	MS Pre vs. HC	MS Pre vs. MS Post	MS Post vs. HC
A1	–0.410 (0.204)	–0.484 (0.233)	–0.553 (0.262)	**0.023§**	**0.012***	**0.050***	0.559
A2	2.534 (0.695)	2.786 (0.750)	3.10 (0.922)	**0.012§**	**0.054***	**0.047***	0.334
K1	–0.713 (0.337)	–0.798 (0.459)	–0.938 (0.388)	**0.018§†**	**0.035*** (slow) 0.105 (med) 0.888 (fast)	0.721 (slow) 0.494 (med) 0.114 (fast)	**0.015*** (slow) 0.294 (med) 0.818 (fast)
K2	0.444 (0.276)	0.438 (0.284)	0.501 (0.295)	0.705	0.854	0.869	0.726
K3	–0.505 (0.211)	–0.479 (0.235)	–0.702 (0.455)	**0.112†**	0.904 (slow) 0.152 (med) **0.018*** (fast)	0.931 (slow) 0.526 (med) 0.241 (fast)	0.852 (slow) 0.071 (med) **0.003*** (fast)
H1	0.435 (0.250)	0.445 (0.184)	0.547 (0.242)	0.247	0.230	0.995	0.246
H2	–0.682 (0.232)	–0.656 (0.240)	–0.784 (0.211)	**0.037§**	0.185	0.591	**0.028***
H3	0.491 (0.141)	0.475 (0.155)	0.585 (0.280)	0.1489	0.327	0.547	0.166

§Significant group effect, $p < .05$.

†Significant group × velocity interaction, $p < .05$; *p*-values are listed for each group comparison based on slow, medium, or fast walking velocity. The three velocities were defined based on the range of walking velocities exhibited in both patients with MS and healthy controls.

*Significant group difference, $p < .05$ after Tukey's adjustment for multiple comparisons.

**Marginally significant group difference, $p < .05$ after Tukey's adjustment for multiple comparisons.

Note. Negative values indicate power absorption and positive values indicate power generation. A1—peak ankle power absorption in early stance; A2—peak ankle power generation in late stance; K1—peak knee power absorption in early stance; K2—peak knee power generation in midstance; K3—peak knee power absorption in late stance; H1—peak hip power generation in early stance; H2—peak hip power absorption in late midstance; H3—peak hip power generation in late stance.

There was a significant ($F = 4.35_{2, 92.9}$; $p = .018$) Group effect for knee power absorption during early stance (K1) and a significant ($F = 4.17_{2, 93.4}$; $p = .018$) Group by Velocity interaction for K1. Paired tests showed that pretraining K1 was significantly ($t = 2.56$, $p = .035$) lower in subjects with MS at slow walking velocity only. Posttraining, K1 was still significantly ($t = 2.88$, $p = .015$) lower in subjects with MS at slow walking velocities only. There was no Group effect for knee power absorption during late stance (K3) but there was a significant Group by Velocity interaction ($F = 3.47_{2, 64.4}$; $p = .037$) for K3. Paired tests showed pretraining the K3 was significantly ($t = 2.85$, $p = .018$) lower in subjects with MS at fast walking velocity only. Posttraining, K3 was still significantly ($t = 3.52$, $p = .003$) lower in subjects with MS at fast walking velocities only (Table 3; Figure 2, center).

There was a significant ($F = 3.43_{2, 82.5}$; $p = .037$) Group effect for hip power absorption during midstance (H2). Paired tests showed pretraining, H2 in the subjects with MS was not different from controls and posttraining H2 in the subjects with MS was still significantly ($t = 2.63$, $p = .028$) lower than controls (Table 3; Figure, bottom).

Discussion

The purpose of this study was to evaluate the effectiveness of a six-week elliptical exercise training to alter joint kinetics during walking in patients with MS toward joint kinetics values as those found in healthy controls. The measured gait variables were joint torques and powers. Changes in joint kinetics would result from alteration in the relative contribution of individual muscle groups to the total output of the limb (DeVita & Hortobagyi, 2000). It was hypothesized that joint torques and powers would change in patients with MS following elliptical training since training could cause an increase in strength or an increase muscle activation of lower extremity muscle groups during gait. It was also hypothesized that posttraining, joint torque and power variables would be closer

to normative values from healthy controls. No other studies have identified changes in joint torques and powers as a result of any exercise training in subjects with MS. Joint torques and powers were evaluated while subjects with MS walked at their self-selected pace, pre- and posttraining, and compared with those of healthy, age-, height-, and weight-matched control subjects. The results indicated that both hypotheses were supported since significant differences were found in the joint torques at the hip and joint powers at the ankle from pre- to posttraining. Thus, MS subjects who participated in our elliptical exercise training significantly altered specific joint torques and powers such that there were no longer differences compared with healthy controls. The evaluation of joint kinetics in this study allows for more specific determination of the effects of elliptical training on gait compared with evaluating only spatial and temporal gait parameters.

After training, power absorption (A1) and generation (A2) at the ankle increased to the level of healthy controls. Ankle plantar flexor torque (APT) marginally increased ($p = .103$) at the ankle. Torque during early stance (ADT) did not improve which may be because the foot is flat on the foot plate of the machine for the duration of the exercise; thus, there is no period of eccentric lowering of the foot to the ground that is seen during gait. These changes indicate that overall there was limited change in muscle force provided during push-off, indicated by APT, at late stance. The increase in A1 and A2, however, indicate the voluntary control of the ankle plantar flexors and dorsiflexors changed such that both eccentric and concentric muscle activity was increased as reflected by the increase in power absorption and generation, respectively. Overall, the increase in ankle power generation during late stance will help to support forward progression of the trunk and to properly initiate the swing phase of gait (Neptune, Kautz, & Zajac, 2001). Clinicians refer to gait powered by the ankle push-off as using an *ankle strategy*, which is thought to be the preferred walking strategy in healthy young adults (Kerrigan, Todd, Della Croce, Lipsitz, & Collins, 1998; Mueller, Sinacore, Hoogstrate, & Daly, 1994). After training, subjects with MS appear to adopt a gait pattern that is primarily supported by increased ankle power. In healthy controls, the plantar flexors provide compensatory mechanisms for musculoskeletal deficits that could not be provided for by compensatory action of other muscle groups; thus, it is suggested that rehabilitation or preventative exercise programs should consider focusing on increasing or maintaining plantar flexor strength (Goldberg & Neptune, 2007).

Peak hip extensor torque (HET) increased as a result of elliptical training in subjects with MS, so there was no longer a difference as compared with healthy control values. The increase in hip extensor torque is likely due to the increased activation of the hip extensors (Burnfield et al., 2010) and increased peak hip flexor and extensor torques (Lu, Chien, & Chen, 2007) seen during elliptical exercise training. This increased muscle activation and redistribution of joint torques during elliptical exercise indicates that the hip musculature is providing more work while using the machine. Overall, the increase in HET indicates an improvement in the ability of the hip extensor to move the hip from peak flexion at the beginning of stance toward extension. Increased peak torque also indicates a muscular strength increase of the hip extensors as a result of elliptical training. Interestingly, in elderly individuals and ACL reconstruction patients, reduced ankle and knee torque, as seen in subjects with MS pretraining, are compensated for with increased hip extensor torque (DeVita, Hortobagyi, & Barrier, 1998; DeVita & Hortobagyi, 2000). This compensation was not seen in subjects with MS pretraining. Posttraining, however, HET was significantly increased in subjects with MS. Ankle and knee torques were unchanged posttraining, so the HET increase may indicate a compensatory strategy that the subjects with MS were able to adopt posttraining.

The deficit in knee extensor torque (KET) and knee power absorption during early (K1) and late (K3) stance compared with healthy controls were maintained after training indicating that elliptical exercise did not affect gait mechanics at the knee. Whereas Burnfield et al. (2010) reported that peak activation and duration of activation of the knee flexors and extensors were higher during elliptical exercise compared with walking and Lu et al. (2007) reported that knee torques were significantly greater during elliptical exercise than during walking, there was no component of negative work at the knee during elliptical exercise (Lu et al., 2007). Thus, the activation of the knee extensors is during elliptical exercise appears to be concentric muscle activation only. The lack of changes in eccentric knee extensor activity (KET and K3) indicates a lack of specificity in the elliptical training since no negative work/power absorption occurs at the knee.

An advantage of using an elliptical trainer for gait-simulating exercise in subjects with MS is that the machine allows for variations in training settings and avoids over-habituation of sensory information during training. Cai et al. (2006) suggested that the functional connectivity that is responsible for standing and stepping is related with the overall assembly of synapses, wherein the probability of these synaptic events is not deterministic. The authors recommended that motor training for a specific task should also incorporate variability since having a fixed training modality does not accommodate for variability that is intrinsic to neural circuits and may cause over-habituation of sensory information, making the individual less adaptable to unanticipated disturbances to the system (Cai et al., 2006). The specific improvements at the ankle can also be explained according to suggested need for variability during training. According to research by Burnfield et al. (2010), in which similarity of joint kinematics between four different elliptical exercise machines and normal walking were evaluated, the kinematics at the ankle showed the lowest coefficient of multiple correlations between walking and the elliptical machines. Interestingly, the specific ankle angle at initial contact, peak plantar flexion, peak dorsiflexion, and

midswing showed higher percent standard deviation for each of the elliptical trainers than either the hip or knee joint angles at the same points in the stance phase. The increased range of variability at the ankle, regardless of the specific elliptical machine used, is in line with the suggestion by Cai et al. (2006) that motor training for a specific task should also incorporate variability. Lack of variability in training at the other joints may be the reason for a lack of significant change in joint torque and power at the knee and hip. The altered neuromuscular control of the ankle is reflected in the increased eccentric muscle control of the ankle dorsiflexors during early stance and increased concentric muscle control of the ankle plantar flexors during late stance/preswing.

This study showed that as a result of a brief (6 weeks) exercise intervention using an elliptical exercise machine, subjects with MS showed significant changes in joint kinetics at the hip and ankle toward values obtained from healthy controls. However, there were some limitations associated with the study. The first limitation is the lack of an MS control group, but the use of a control group would not address the issue of intervention effects because individuals within the control group would also have individual patterns of disease progression that may or may not have matched those of the experimental group. However, because all of the MS subjects were naïve to any exercise programs within the 3 months preceding enrollment in the study, the elliptical exercise training is the only physical activity change that could account for the change in joint kinetics. In addition, since all of the subjects were mildly affected, all were relatively active outside of the home, so the changes in joint kinetics should not be attributed to increased day-to-day physical activity. The second limitation is that only mildly affected subjects with MS were included in the study, so these results are only applicable to individuals who are able to walk independently. To use the elliptical safely, subjects need to be able to support their own body weight independently, so it was not feasible to safely perform the intervention if the patient could not stand independently.

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