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Gait Mechanics Are Different Between Healthy Controls and Patients With Multiple Sclerosis

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Multiple sclerosis (MS) causes severe gait problems in relatively young individuals, yet there have been limited studies to quantitatively identify the specific gait parameters that are affected. The purpose of this study was to define any differences in biomechanical gait parameters between patients with MS and healthy controls. A total of 31 MS patients and 31 healthy controls were evaluated: joint torques and joint powers were calculated at the ankle, knee, and hip during the stance phase of gait. The self-selected walking velocity was used as a covariate in the analysis to ensure that group differences were not due to differences in walking velocity between the MS and healthy control groups. Reduced angular range, less joint torque, and reduced joint power were seen in patients with MS. We also found significant correlations between biomechanical gait parameters and EDSS score, which provides a clinical rating of disease severity. Our findings provide a quantitative assessment of the gait mechanics employed in patients with MS. The altered lower extremity mechanics observed in patients with MS reflect both a neurological and strength deficit compared with healthy controls during walking.

Keywords: joint kinetics, neurological disease, gait velocity, lower extremity

Multiple sclerosis (MS) is an autoimmune disease that causes progressive neurodegeneration and is commonly diagnosed in young adults between 20 and 40 years old.[1] Symptoms vary widely within individuals with MS, but commonly reported symptoms include sensory disturbances, limb weakness, clumsiness, gait ataxia, and cognitive deficits.[2] There is a demonstrated need for a mechanism to identify gait abnormalities in the early stages of the disease, before the onset of a clinical disability, which could provide better classification for MS patients and for targeting more aggressive therapies.[3] The disease severity and the clinical classification of movement disability in MS patients are typically measured using the Kurtzke Disability Scale (EDSS). This scale rates patients on a 20 point scale through a series of functional system tests.[4] While the scale provides the clinician with a general perception of the patient's level of disability, it does not provide information regarding the quality of gait or any other associated movement problems.

Quantitative evaluation of gait in the form of joint mechanics may provide one solution to this problem.[5] This analysis has been used extensively to characterize gait abnormalities in different populations and to guide treatment or to assess outcomes.[6–12] Specifically, evaluation of joint torques and powers allows for the quantification of the relative contribution of muscle groups during a movement, as well as the identification of the specific type of muscular contraction (ie, concentric or eccentric) that is controlling the joint motion. However, in patients with MS, relatively few studies have used such methodology to examine gait characteristics. Previous studies describe the ground reaction force and joint angle patterns[13] and balance control during gait initiation in patients with MS.[14] Recently, Kelleher et al[15] used kinematics, ground reaction forces and EMG in MS patients and reported reduced gait speed, reduced maximum hip and knee extension, ankle plantar flexion angle and propulsive force compared with the control group. Wurdeman and colleagues[16] examined ground reaction forces to describe differences in the frequency component between MS patients and healthy controls. Finally, relationships between gait mechanics and fatigue and quality-of-life measures have been identified.[17] While these studies have successfully used biomechanical data to begin to identify specific gait patterns in MS patients,

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evaluating joint torques and powers to investigate joint muscular responses and their contributions can provide fundamental understanding of the mechanics of walking in individuals with neuromuscular pathologies. This information may allow for a better understanding of how varying symptoms relate to gait problems, how the progression of the disease affects gait, and to a classification of the different neuromuscular adaptations that underlie any alterations in gait mechanics. Thus, this study extends previous work by examining joint torques and powers during gait that allow for specific determination of muscular contributions and responses at each joint. This study also uses walking velocity as a covariate in the analysis to determine whether the gait differences observed between the groups are influenced by differences in walking velocity across the groups. Finally, this study examines the relationship between the gait parameters of MS patients and their clinically defined EDSS scores to determine whether biomechanical gait parameters are related to disease severity.

It was hypothesized that MS patients would exhibit reduced joint torques and powers at the ankle, knee, and hip compared with healthy age-matched controls. This prediction was made based on the previously reported spatial temporal alterations in MS patients where stride lengths were found to be shorter and double support times were found to be longer in MS patients.[3,13,18] Such spatial and temporal alterations are likely related to changes in joint torques and powers of MS patients. In addition, it was hypothesized that there would be significant correlations between gait parameters and EDSS scores since patients with are assigned higher EDSS scores as mobility decreases.[4]

Methods

Subjects

Thirty-one MS patients and 31 healthy controls participated in this study (Table 1). All procedures were approved by the University's Medical Center Institutional Review Board. Participants were recruited through the University Medical Center's Neurology clinic and referrals from private practice neurology clinics. Control subjects were recruited through family members of MS subjects and advertisement in the community to match for age and sex. Exclusion criteria for the MS subjects included inability to give informed consent, an Expanded Disability Status Scale (EDSS) score greater than or equal to 6.5, completion of treatment for relapse less than 30 days before study participation, and any other neurological or vestibular disorder. MS subjects were not excluded for taking any approved disease-modifying therapy for MS[19] but subjects taking the symptom-modifying medication Fampridine were excluded since it has been shown to specifically affect gait.[20]

Data Collection Protocol

Patients were prepared for collection by wearing a form-fitting outfit and obtaining anthropometric data. Reflective markers were placed bilaterally according to anatomical positions using a modified Helen Hayes marker set.[21] The initial starting point for the walking trials was determined so that the subject could strike the force platform, which was in the middle of the walkway, with only one foot. During each trial, the subject walked at self-selected pace over a 10 m walkway while three-dimensional marker trajectories (EvaRT 5.0, Motion Analysis Corp., Santa Rosa, CA, sampling at 60 Hz) and ground reaction forces (Kistler force platform, Model 9281B11; Amherst, NY, sampling at 600 Hz) were simultaneously collected. Subjects rested for a minimum of one minute between each trial. The process was repeated to obtain five good walking trials with the patient's foot landing completely within the force plate without altering the stride, and then the other limb was collected using the same process. Data collection procedures for MS patients and for healthy controls were identical. Joint angle, torque, power variables and walking speed were calculated for each trial and the average value across all 5 trials for each leg was used for analysis. This resulted in one value for each variable for each leg. All subjects walked at self-selected pace to ensure that the natural walking pattern was captured since self-selected pace is the most stable during walking[22] and in MS patients,

Table 1 Demographic information for MS patients and healthy controls

	MS Patients (n = 31)	Healthy Controls (n = 31)
Sex (M/F)	5 male, 26 female	8 male, 23 female
Age	46.2 ± 10.6 y	42.0 ± 12.5 y
EDSS	2.6 ± 0.7 (range 1.0–4.0)	—
Height (cm)	165.3 ± 6.7	170.6 ± 18.8
Mass (kg)	78.1 ± 15.8	77.2 ± 11.04
Walking Velocity (m/s)*	1.06 ± 0.21	1.21 ± 0.22

*$P < .05$, significant difference in walking velocity between MS patients and healthy controls.

walking at a faster than preferred pace results in increased metabolic cost.[23] The ranges of walking speeds were 0.68–1.42 m/s for MS subjects and 0.69–1.55 m/s for controls.

Data Analysis

A low-pass second order Butterworth digital filter with a 7 Hz cutoff was used to smooth the marker trajectories during postprocessing. Inverse dynamics were applied using custom MATLAB programs to calculate the peak flexor and extensor torques for the ankle, knee, and hip joints in the sagittal plane based on algorithms described by Winter.[5] The joint torques and muscle powers (Tables 2–4) were normalized to the subject's body mass. The gait cycle definitions and procedures followed for identifying these parameters have been outlined in other gait studies.[6,24–26]

Statistical Analysis

Linear mixed effects modeling was used to evaluate differences in mean joint angles, torques, and powers between groups. Group, velocity, and the interaction between group and velocity were included in the model as fixed effects. In this way, the model allowed for identification of variables that were different as a result of group, regardless of walking velocity differences between groups. Because both limbs of each subject were used for analysis, random effects were included in the model to account for the correlation between limbs of the same subject. Spearman correlations were performed between each joint angle, torque, and power variable and the MS patient EDSS score. Spearman correlations were used because the distribution of EDSS scores was not normal according to the Shapiro-Wilk test ($P = .027$). Statistical analyses were performed using SAS version 9.2. Statistical significance was set at $\alpha = .05$.

Results

At the ankle, peak plantar flexion angle in early stance (APF_ES) was significantly lower ($F = 10.95$, $P = .016$) and peak plantar flexion angle at toe-off (APF_TO) was significantly lower ($F = 5.51$, $P = .022$) in MS patients. APF_TO significantly correlated ($P < .001$) with EDSS score in MS patients. There were no significant differences between groups found in knee joint motion. However, peak knee flexion angle (KFLX) and knee range of motion (KROM) significantly correlated ($P = .001$; $P = .012$ respectively) with EDSS score in MS patients. At the hip, extension (HEXT) at terminal stance, was also significantly lower ($F = 4.64$, $P = .034$) in MS patients. There was a significant interaction between group and

Table 2 Joint angle results between MS patients (MS) and healthy controls (HC)

	MS Mean (SD)	HC Mean (SD)	Paired Test *P*-Value	MS Correlation with EDSS
Joint Angles (Degrees)				
APF_ES	–5.47 (3.62)	–6.58 (2.37)	0.016*	–0.019
ADF	12.74 (3.52)	11.28 (3.37)	0.142	–0.065
APF_TO	–13.53 (5.56)	–16.88 (4.73)	0.022*	–0.593‡
AROM	18.12 (3.94)	17.78 (3.24)	0.956	–0.077
KFLX	13.94 (5.60)	13.88 (5.67)	0.349	–0.565‡
KEXT	4.97 (4.10)	4.31 (3.84)	0.348	–0.289
KROM	9.33 (3.88)	9.79 (4.19)	0.437	–0.477‡
HFLX	25.18 (4.62)	27.35 (4.57)	0.088	–0.532‡
HEXT	–10.42 (4.53)	–11.91 (4.23)	0.034*†	–0.174
HROM	35.58 (4.91)	39.26 (3.84)	0.001*†	–0.635‡

*$P < .05$, significant difference between MS and HC.

†$P < .05$, significant effect of walking velocity on difference.

‡$P < .05$, significant correlation between gait variable and EDSS score.

APF_ES—peak ankle plantar flexion angle during early stance; ADF—peak ankle dorsiflexion angle during late stance; APF_TO—peak ankle plantar flexion angle at toe-off; AROM—total angle range of motion during stance phase; KFLX—peak knee flexion angle during stance; KEXT—peak knee extension angle during stance; KROM—total knee range of motion during stance phase; HFLX—peak hip flexion angle during early stance; HEXT—peak hip extension angle during late stance; HROM—total hip range of motion during stance phase.

Table 3 Joint torque results between MS patients (MS) and healthy controls (HC)

	MS Mean (SD)	HC Mean (SD)	*P*-Value	MS Correlation with EDSS
Joint Torques (N·m/kg)				
ADT	–0.267 (0.072)	–0.381 (0.163)	0.002*	–0.296
APT	1.195 (0.141)	1.354 (0.219)	<0.001*	–0.297
KET	0.490 (0.203)	0.709 (0.236)	0.009*	–0.580‡
KFT	–0.291 (0.154)	–0.256 (0.232)	0.086	–0.013
HET	0.650 (0.207)	0.789 (0.238)	0.157	0.062
HFT	–0.765 (0.189)	–0.975 (0.277)	0.003*	0.148

*$P < .05$, significant difference between MS and HC.

‡$P < .05$, significant correlation between gait variable and EDSS score.

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.

Table 4 Joint Power results between MS patients (MS) and Healthy Controls (HC).

	MS Mean (SD)	HC Mean (SD)	*P*-Value	MS Correlation with EDSS (ρ)
Joint Powers (W/kg)				
A1	–0.398 (0.196)	–0.601 (0.261)	0.015*	–0.409‡
A2	2.440 (0.668)	3.121 (0.874)	0.008*†	–0.574‡
K1	–0.675 (0.353)	–1.021 (0.430)	0.006*	–0.595‡
K2	0.436 (0.276)	0.533 (0.308)	0.492	–0.398‡
K3	–0.511 (0.217)	–0.857 (0.594)	0.019*†	–0.173
H1	0.460 (0.238)	0.617 (0.298)	0.073	–0.104
H2	–0.651 (0.253)	–0.903 (0.366)	0.016*	–0.497‡
H3	0.495 (0.114)	0.672 (0.321)	0.014*	–0.109

*$P < .05$, significant difference between MS and HC.

†$P < .05$, significant effect of walking velocity on difference.

‡$P < .05$, significant correlation between gait variable and EDSS score.

Note. Negative joint power 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.

velocity for HEXT ($F = 4.35$, $P = .041$), which showed that at slow walking velocities HEXT was significantly lower ($t = 2.01$, $P = .049$) in patients with MS but at faster walking velocities, there was no difference ($t = 0.79$, $P = .434$). Peak hip flexion (HFLX) significantly correlated ($P = .002$) with EDSS score in MS patients. Total hip range of motion (HROM) was significantly lower ($F = 18.08$, $P < .001$) in MS patients. There was a significant interaction between group and velocity for HROM ($F = 14.25$, $P = .003$), which showed that at slow walking velocities HROM was significantly lower ($t = 4.69$, $P = .001$) in patients with MS but at faster walking velocities, there was no difference ($t = 0.04$, $P = .967$). HROM also significantly correlated ($P < .001$) with EDSS score in MS patients such that subjects with higher EDSS scores had lower HROM.

At the ankle, dorsiflexor torque during early stance (ADT) and plantar flexor torque during late stance (APT) were significantly lower ($F = 10.50$, $P = .002$; $F = 13.73$, $P < .001$; respectively) in MS patients (Figure 1, top). The knee extensor torque (KET) during early stance was significantly lower ($F = 7.29$, $P = .009$) and hip flexor torque (HFT) during late stance was significantly lower ($F = 9.35$, $P = .003$) in MS patients (Figure 1, middle and bottom). KET was significantly correlated ($P = .001$) with EDSS score in MS patients. Importantly, none of these joint torque differences were affected by the group difference in walking velocity, which indicates significant differences between groups was not because the groups walked at different speeds (Table 3).

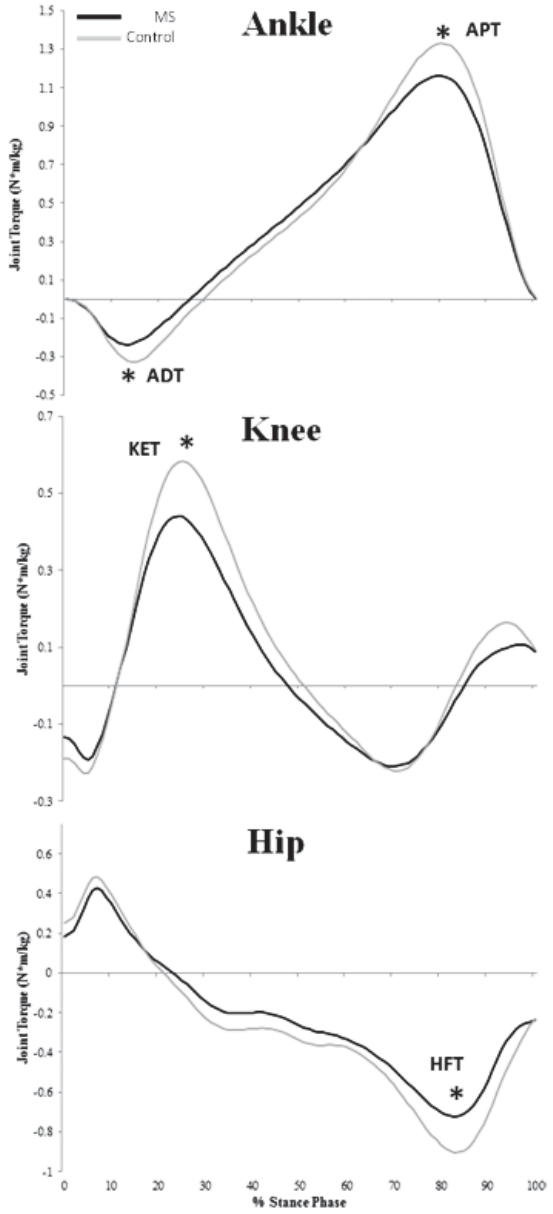

Figure 1 — Joint torque mean ensemble curves for the healthy controls (identified with gray lines) and the MS patients (identified with black lines) at normal walking velocity. Significant differences ($P < .05$) between groups for the parameters selected are identified with an asterisk (*). ADT is peak ankle dorsiflexor torque during early stance. APT is peak ankle plantar flexor torque during late stance. KET is peak knee extensor torque during stance. HFT is peak hip flexor torque during late stance.

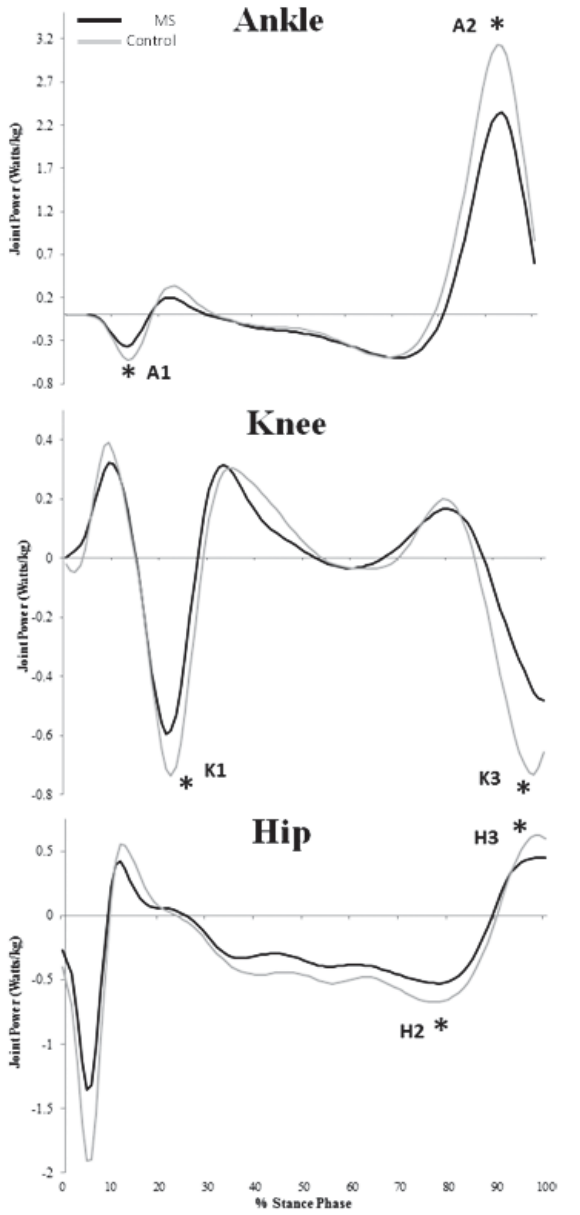

Figure 2 — Joint power mean ensemble curves for the healthy controls (identified with gray lines) and the MS patients (identified with black lines) at normal walking velocity. Significant differences ($P < .05$) between groups for the parameters selected are identified with an asterisk (*). A1 is the ankle power absorption during early stance. A2 is the ankle power generation during late stance. K1 is the knee power absorption during early stance and K3 during late stance. H2 is the hip power absorption at midstance and H3 is the hip power generation during late stance.

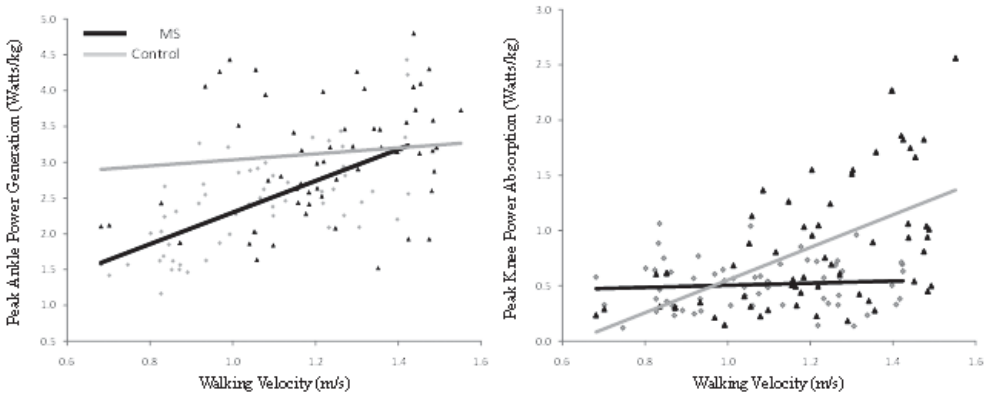

Figure 3 — Scatter plots to represent the significant interactions between specific joint power variables and walking velocity. The gray lines are for the healthy control group and the black lines are for the MS patients group. The left graph illustrates interaction ($P = .03$) between walking velocity and peak ankle power generation during late stance (A2); the right graph illustrates interaction ($P = .003$) between walking velocity and peak knee power absorption during late stance (K3).

The ankle power absorption during early stance (A1) and the ankle power generation during late stance (A2) were significantly lower ($F = 6.22$, $P = .015$; $F = 7.63$, $P = .008$; respectively) in MS patients (Figure 2, top). Both A1 and A2 were significantly correlated ($P = .022$; $P = .001$ respectively) with EDSS score in MS patients. There was a significant interaction between group and velocity for A2 ($F = 4.88$, $P = .03$), which showed that at lower walking velocities, A2 was significantly lower ($t = 3.64$, $P = .006$) in MS patients, but at faster walking velocities, the difference between MS patients and controls was no longer significant ($t = 1.14$, $P = .260$) (Figure 3, left). Knee power absorption during early stance (K1) ($F = 8.27$, $P = .006$) and during late stance (K3) ($F = 5.93$, $P = .019$) were both significantly lower in the MS patients (Figure 2, middle). K1 and K2 (peak knee power generation during midstance) were both significantly correlated ($P < .001$, $P = .027$ respectively) with EDSS score in MS patients. There was a significant interaction between group and velocity for K3 ($F = 9.29$, $P = .004$), which showed that at slower walking velocity there was no difference ($t = 0.87$, $P = .391$) between patients with MS and controls but at faster walking velocities, K3 was significantly lower ($t = 4.04$, $P = .002$) in patients with MS (Figure 3, right). Hip power absorption at midstance (H2) was significantly lower ($F = 6.19$, $P = .16$) and hip power generation during late stance (H3) was significantly lower ($F = 6.58$, $P = .014$) in MS patients (Figure 2, bottom). H2 was significantly correlated ($P = .004$) with EDSS score in MS patients. None of the power differences at the hip were affected by the walking velocity differences between the MS patients and the controls, which indicates the significant differences between groups was not because the groups walked at different speeds.

Discussion

This study demonstrates that joint torques and powers during walking in MS patients are significantly altered compared with healthy controls when walking velocity was considered as a covariate in the analysis and indicates that differences between the two groups were independent of walking velocity differences. Our results agree with the original hypothesis that MS patients would exhibit reduced joint torques and powers compared with controls. In addition, there were several significant relationships identified between gait variables and the EDSS score for MS patients that indicate that the biomechanical gait variables are related to clinical measures of disease severity.

At the ankle, dorsiflexor torque during the loading response (ADT) was reduced in MS patients. This could occur if the resultant ground reaction force vector is closer to the ankle joint center during loading.[5] During early stance, there was a reduced plantar flexion at the beginning of single limb stance and overall significantly reduced hip range of motion. These results indicate that the ground reaction force vector likely remained closer to the ankle joint during weight acceptance. Thus, patients with MS could exhibit a gait pattern with the foot landing on the ground in a less plantar-flexed position. Overall, this joint position could reduce the requirement of the ankle dorsiflexors to control the plantar flexion movement during early stance. This is also evident by the significant reduction in the ankle power absorption (A1) during early stance, which reveals a decreased eccentric contraction by the MS patients. This decreased contraction could be the result of changes in joint geometry at or shortly after touchdown, or it may suggest a decreased neuromuscular ability to control the plantar flexion movement during early stance.

Peak ankle plantar flexor torque was significantly reduced in MS patients during terminal stance, which led to a reduced concentric contraction of the plantar flexors as revealed by the ankle power generation (A2) during preswing (push-off power). While peak dorsiflexion angle during late stance was not different, peak plantar flexion at toe-off was significantly lower in MS patients, so the angular distance traveled by the ankle is lower and the amount of torque generated (APT) was reduced.

The reduction in plantar flexor torque will result in an inability to support forward progression of the trunk and to properly initiate the swing phase of gait.[27] In addition to supporting trunk progression and initiating the swing phase of gait, ankle power generation has been reported as the strongest predictor of step length in elderly subjects[28] and has been found to correlate positively with gait velocity and stride length in older persons.[5] Control subjects walked faster than MS patients, but there was a significant interaction between walking velocity and group (MS vs. control) for ankle power generation (Figure 3, left). This suggests that fast walking MS patients were able to increase ankle power generation and achieve concentric contractions at the ankle. It is possible that the evaluation of ankle power generation could reflect severity of gait disturbance in persons with MS. Such a conclusion is supported by the strong relationship between A2 and EDSS score, which reflects clinically rated neurological impairment. In addition, peak ankle torque at late stance showed no group by velocity interaction. Because ankle joint power was affected by walking velocity but peak ankle torque was not, only angular velocity of the segment changed at different walking speeds. This may indicate that subjects able to walk faster could also increase the angular velocity of the segment, which resulted in increased ankle power at late stance and is a possible indication of better segment control through muscle firing. It seems the interaction observed is likely more neurological in nature than muscular, but this conclusion must be investigated further by identifying any changes in muscle strength through dynamometer use and by examining EMG changes during walking.

At the knee, extensor torque (KET) was significantly reduced during early/midstance and power absorption (K1) was reduced during early stance. During early stance, the knee functions as a shock absorber by flexing and eccentrically absorbing power.[29] The reduction in K1 in patients with MS indicates that while peak knee flexion angle was not different, the neural control of the knee flexors was impaired so the eccentric control of knee flexion was reduced. There was a strong relationship between K1 and EDSS score for MS patients, which could indicate that if K1 is reflective of reduced neural control of the knee flexors, then patients with higher EDSS scores (greater neurological impairment) have reduced neural control of lower extremity muscles. During late stance, the knee extensors again eccentrically control knee flexion and absorb power. However, power absorption during late stance (K3) was significantly reduced in MS patients. The inability to control knee flexion by the extensor muscles may be the result of neuromuscular control deficits in the MS patients since knee flexion and extension angles were unchanged. During late stance, with reduced ankle power generation there is reduced energy transferred up the kinetic chain to the knee. With less energy transferred up to the knee by the ankle plantar flexors, there is less power that needs to be absorbed at the knee; thus K3 is reduced. Such an explanation is also supported by Judge et al,[28] who interpreted power absorption at the knee as a natural consequence of the energy transferred between the ankle and knee.

There was a significant interaction between walking velocity and group for K3, where in the MS group, as walking velocity increased, K3 values did not increase but in controls K3 did increase with velocity (Figure 3, right). The interaction indicates a different neuromuscular strategy for the patients with MS. Interestingly, there was also a significant interaction between walking velocity A2 mentioned earlier. The relationship between A2 and K3 is such that if A2 increased with velocity then K3 should also increase with walking velocity but instead, K3 did not increase with walking velocity in the MS group. This disagreement may be due to an inability of the MS group to actively control (eccentric muscle contraction) knee flexion by the knee extensor muscles as walking velocity increases. This finding suggests that the MS patients who were able to walk faster were still not able to increase power absorption at the knee and achieve the necessary eccentric contractions of the knee extensors. Therefore, knee power absorption (K3) is equally affected regardless of the severity level, since K3 does not increase with increasing walking velocity as it does in controls.

Hip joint motion showed reduced peak extension angle (HEXT) and total range of motion (HROM) in MS patients. Reduced HEXT limits the progression of the trunk over the leg during stance and results in significantly reduced flexion torque (HFT) during late stance. Both HEXT and HROM during stance showed an interaction between group and walking velocity. This effect of velocity at the hip was not seen in joint torque or power measures, which indicates that faster walking MS patients have increased range of motion capability at the hip, but could not activate the hip musculature adequately to transition the leg into swing. In addition to decreased strength during late stance, peak power generation (H3) was lower in MS patients. During the push-off phase of stance, the large burst of power generation is necessary to accelerate the leg.[29] The inability of MS patients to generate sufficient power at the end of stance points to an inability to adequately activate the flexor muscles of the hip. Just after midstance, there is a period of power absorption at the hip that signals the transition from joint flexion into extension.[29] Peak power absorption at this point (H2) is also significantly lower in MS patients. It appears joint motion control must be attenuated in patients with MS since the transition from controlling the joint motion (eccentric muscle action of the hip flexors) to initiating the joint motion (concentric muscle action of the hip extensors) is impeded by reduced neuromuscular control. The moderate relationship between H2 and EDSS score supports the idea that transitioning from controlling to initiating joint motion is impaired in MS patients due to neurological deficits since the EDSS score reflects gross neurological impairment.[4] This finding is also supported by the reduced HROM, which indicates that overall movement at the hip is limited to reduce the power generation and absorption requirements during stance. In studies of elderly, a decrease in ankle plantar flexor

power generation at late stance is compensated with an increase in hip power generation at the same point in the stance phase.[6,24,30] In MS patients this compensation does not occur. It seems that MS patients are unable to adapt any other compensatory strategies to overcome reduced power at one joint by increasing power at another joint.

The slower walking velocity measured in MS patients compared with controls may be explained by the reduced dorsiflexor torque and accompanying reduced power absorption at loading response and by reduced ankle and hip power generation at terminal stance. These variables have been shown to be the most important in predicting performance during walking in both elderly and Parkinson populations.[6,12,31] Like MS, Parkinson's disease (PD) patients also have significantly reduced ankle torque, reduced hip extension, and reduced hip power during late stance.[12,31]

This study does have some limitations. This study did not measure lower extremity spasticity or lower extremity muscle strength. In future studies it would be of great interest to examine how spasticity and muscle strength are related to the gait parameters described in this article. Because of the many factors that can affect gait in persons with MS, this study did not include/exclude or classify subjects based on any specific walking tendencies (ie, drop foot or reported leg weakness). For this reason, the MS group is mildly affected based on the mean EDSS score only. Finally, gait variables were correlated with the gross EDSS score only and not the specific functional system scores. While it would be of interest to examine the relationship between gait variables and, for example, cerebellar and pyramidal signs, the focus of this article is to examine the biomechanical differences in gait only. In the future, it would be of high interest to expand these findings by examining the relationship between gait and related neurological deficits. Finally, torque and power variables were normalized with respect to body mass, but instead could have been normalized with respect to walking speed or leg length, for example. However, this study used walking speed as a covariate in the analysis and because the groups showed no statistical difference in mass or height, it is unlikely that leg length normalization would have altered the results.

Despite limitations, this study found extensive significant differences in joint torques and powers of patients with MS compared with healthy controls. These differences provide a picture of the mechanical gait deficit seen in these patients and indicate the importance of using advanced gait analysis evaluation to classify disability in MS. Importantly, the differences in gait kinetics between MS patients and controls cannot be explained by differences in walking velocity alone since velocity was controlled in our statistical model. Thus, specific neuromuscular mechanisms independent of walking velocity were revealed for this pathology including the inability to eccentrically control muscle activity (power absorption by the ankle dorsiflexors) and to concentrically generate muscle activity (power generation by the ankle plantar flexors and the hip flexors). The conclusion that the interactions between joint powers and walking velocity are likely more neurological in nature than muscular must be investigated further by measuring changes in muscle strength and muscle firing patterns during walking. The significant relationships between EDSS score and gait variables in the MS patients indicate that biomechanical gait variables are related to clinical disability. Thus, gait analysis could be performed in MS patients to support clinical decision making as is the case for patients with cerebral palsy.[10] Gait analysis offers an additional tool to monitor disease status and progression and to determine outcomes from rehabilitation interventions and pharmacological treatment protocols.

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