Step length asymmetry is representative of compensatory mechanisms used in post-stroke hemiparetic walking

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ABSTRACT

Post-stroke hemiparetic subjects walk with asymmetrical step lengths that are highly variable between subjects and may be indicative of the underlying impairments and compensatory mechanisms used. The goal of this study was to determine if post-stroke hemiparetic subjects grouped by step length asymmetry have similar abnormal walking biomechanics compared to non-impaired walkers. Kinematic and ground reaction force data were recorded from 55 hemiparetic subjects walking at their self-selected speed and 21 age and speed-matched non-impaired control subjects. Hemiparetic subjects were grouped by paretic step ratio, which was calculated as the paretic step-length divided by the sum of paretic and nonparetic step-lengths, into high (>0.535), symmetric ($0.535–0.465$) and low (<0.465) groups. Non-parametric Wilcoxin signed-rank tests were used to test for differences in joint kinetic measures between hemiparetic groups and speed-matched control subjects during late single-leg stance and pre-swing. The paretic leg ankle moment impulse was reduced in all hemiparetic subjects regardless of their paretic step ratio. The high group had increased nonparetic leg ankle plantarflexor and knee extensor moment impulses, the symmetric group had increased hip flexor moment impulses on both the paretic and nonparetic leg and the low group had no additional significant differences in joint moment impulses. These results suggest that the direction of asymmetry can be used to identify both the degree of paretic plantarflexor impairment and the compensatory mechanisms used by post-stroke hemiparetic subjects.

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1. Introduction

Post-stroke hemiparetic subjects walk more slowly than non-impaired walkers, and self-selected walking speed is regularly used as a measure of rehabilitation outcomes because it is related to functional status and quality of life [1]. However, since compensatory action by the nonparetic leg can result in more effective self-selected walking speeds, speed alone is not a reliable measure of hemiparetic walking ability. While step length asymmetry has also been used as a measure of walking ability, it is weakly related to walking speed such that post-stroke hemiparetic patients walking at similar speeds may exhibit different step length asymmetries [2–4]. Since this between subject variability may result from the use of different walking mechanisms, step length asymmetry may be more indicative of the underlying impairments and compensatory mechanisms used than self-selected walking speed.

An important factor in generating step length is forward propulsion, which is generated through the anterior-posterior ground reaction force (A/P GRF) of the stance leg that enables the trunk to progress forward while the contralateral leg is in swing [5,6]. A recent study found step length asymmetry of post-stroke hemiparetic subjects negatively correlates with propulsion asymmetry [3], with those subjects unable to generate sufficient propulsion during paretic leg stance showing decreased nonparetic leg step lengths compared to the paretic leg. Balasubramanian et al. [3] suggest that subjects who generate shorter nonparetic step lengths may have an active reduction in paretic leg propulsion generation due to ankle plantarflexor muscle impairment. The plantarflexors are important contributors to propulsion in non-impaired walking [7,8] and hemiparetic subjects often exhibit paretic leg plantarflexor impairment [9,10]. As a result, several studies have found step length and step length asymmetry to correlate with paretic leg plantarflexor impairment in post-stroke hemiparetic subjects [4,11].

Conversely, subjects walking asymmetrically with shorter paretic steps generated a higher percentage of total propulsion by the paretic leg [3]. While one explanation for this phenomenon could be that the paretic plantarflexors are unimpaired, it is more

likely that these subjects are unable to generate sufficient pre-swing energy to the paretic leg (which would reduce the paretic leg swing time, thus decreasing paretic step length). Previous studies of hemiparetic walking have found the paretic hip flexors are also important for walking performance [12,13] and often compensate for plantarflexor impairment. During late stance the uniarticular hip flexors pull the leg upwards and forwards, acting to advance the leg further before its subsequent heel strike [13], which contributes to swing initiation [14]. Therefore it is possible that hemiparetic subjects with shorter paretic steps have reduced paretic leg advancement during swing due to impaired paretic leg hip flexor activity in pre-swing.

If step length asymmetry is a good indicator of walking impairment in post-stroke hemiparetic subjects, it may be helpful in developing and monitoring rehabilitation programs as a simple surrogate for more complex biomechanical measures to identify neuromotor impairments. While step length asymmetry has been studied previously [e.g. 3,4,11], its potential to classify subjects into groups with similar walking mechanics has not been examined. Therefore, the goal of this study was to determine if post-stroke hemiparetic subjects grouped by step length asymmetry have similar walking biomechanics, as measured by joint kinetics from mid through late stance (when propulsion and pre-swing occur in non-impaired subjects), compared to non-impaired walkers. We hypothesized that compared to non-impaired walkers at a similar speed, those subjects who walk symmetrically will have similar joint moments, those who walk with longer paretic than nonparetic steps will have reduced paretic leg plantarflexor moment and those subjects who walk with shorter paretic than nonparetic steps will have a reduced paretic leg hip flexor moment.

2. Methods

2.1. Subjects

Fifty-five subjects with chronic hemiparesis (28 left hemiparesis, 35 men; age 61.2 ± 11.4 years; 5.3 ± 5.7 years post stroke) and twenty-one age-matched non-impaired controls (4 men; age 65.2 ± 9.6 years) were recruited at the VA Brain Rehabilitation Research Center (Gainesville, FL). Inclusion criteria for the hemiparetic subjects included hemiparesis secondary to a single unilateral stroke, absence of significant lower extremity joint pain, limb contractures, or major sensory deficits, ability to walk independently with an assistive device over 10 m on a level surface, walk on a daily basis in the home, and absence of cardiovascular impairments contraindicative to walking. Subjects were excluded from the study if they had orthopedic or additional neurologic conditions. All subjects signed informed consent and protocol was approved by the Institutional Review Boards of the University of Florida and the University of Texas at Austin.

2.2. Experimental set-up and procedure

Each hemiparetic subject completed three 30 s walking trials on an instrumented split-belt treadmill (Tecmachine, Andrézieux Boutheon, France) at their self-selected treadmill walking speed without use of an assistive device. Each control subject completed trials at 0.3, 0.6 and 0.9 m/s for speed-matched comparisons. All subjects walked approximately 10 s prior to each data collection to ensure a steady-state walking pattern had been reached. A safety harness mounted to the laboratory ceiling protected the subjects in the event of a loss of balance. One or more practice trials were performed to ensure subjects' comfort and safety. Retro-reflective markers were recorded using a twelve-camera system (Vicon Motion Systems, Oxford, UK) to assess bilateral 3D kinematics at 100 Hz. A modified Helen Hayes marker set that included additional marker triads attached to rigid plates located on each foot, shank and thigh segment was used to define each body segment and reduce measurement error. Bilateral GRFs were captured at 2000 Hz.

2.3. Data analysis

Data were processed using Visual3D (C-Motion, Inc., Germantown, MD). Marker and GRF data were low-pass filtered using a 4th-order Butterworth filter with cutoff frequencies of 6 Hz and 20 Hz, respectively. Intersegmental joint moments (normalized by subject body weight) were calculated using standard inverse dynamics analysis. Data were time normalized to 100% of the gait cycle (paretic leg for hemiparetic subjects and right leg for controls). We calculated joint kinetics in a region approximately corresponding to the usual propulsive phase for each leg. This region was divided into late single-leg stance (i.e. second 50% of single-leg stance)

and pre-swing (i.e. double support phase preceding swing). Within late single-leg stance and pre-swing, joint kinetics were calculated as the joint moment impulses (i.e. time integral of the joint moment) at the hip (flexor positive), knee (extensor positive) and ankle (plantarflexor positive). To quantify propulsion, the A/P GRF impulses (AP impulses) during late single-leg stance and pre-swing were calculated. For the control subjects, the right and left leg values were averaged.

2.4. Statistical analyses

Hemiparetic subjects were grouped by PSR (calculated as the paretic step length divided by the sum of paretic and nonparetic step length) into high (PSR > 0.535), symmetric (0.535 ≥ PSR ≥ 0.465), and low (PSR < 0.465) groups. This grouping criterion was derived from the range of PSR values from the non-impaired controls who were considered to walk symmetrically [15]. All statistics were performed using Matlab (MathWorks, Natick, MA) with significance level set at 0.05. For each PSR group we used non-parametric Wilcoxin signed-rank tests to test for differences in joint moment and AP impulses in late single-leg stance and pre-swing between each hemiparetic subject in the group and the average data of the control subjects walking at the matched speed of that subject (paretic leg vs. control, nonparetic leg vs. control) based on functional walking status (Table 1). For variables that were significantly different from controls in all PSR groups, we used a nonparametric Kruskal–Wallis test with rank sums tests post hoc analyses to test for differences between each PSR group and their speed matched controls.

3. Results

Nine hemiparetic subjects were in the low PSR group (i.e., shorter paretic than non-paretic steps), 17 subjects walked with a symmetric PSR, and 29 subjects were in the high PSR group (i.e., longer paretic than non-paretic steps).

All PSR groups had significantly different paretic leg AP impulses compared to speed-matched controls during paretic leg stance (Fig. 1). The high PSR group was most unable to generate propulsion from the paretic leg; during paretic late single-leg stance both the high and symmetric PSR groups generated less ($p = 0.000$, 0.031) and the low PSR group generated more ($p = 0.013$) paretic leg AP impulse compared to speed-matched controls. During paretic pre-swing all groups had significantly less AP impulse compared to controls ($p = 0.000$, 0.000, 0.002). During both paretic late single-leg stance and pre-swing the high PSR group had a significantly larger deviation between paretic leg AP impulse and the controls than both the low ($p = 0.001$, 0.005) and symmetric ($p = 0.000$, 0.000) groups. During nonparetic late single-leg stance the low PSR group had less ($p = 0.004$) and the high PSR group had more ($p = 0.041$) nonparetic leg AP impulse than controls. During nonparetic pre-swing only the low PSR group had a significantly different nonparetic leg AP impulse compared to controls (reduced, $p = 0.002$).

All PSR groups had less paretic leg plantarflexor moment impulse compared to controls walking at similar speed. In the high PSR group this difference was significant in both paretic late single-leg stance ($p < 0.001$) and pre-swing ($p < 0.001$). In the symmetric PSR group this difference was significant in paretic late single-leg stance ($p = 0.015$) and approached significance in paretic pre-swing ($p = 0.068$). In the low PSR group this difference approached significance in both paretic late single-leg stance ($p = 0.055$) and pre-swing ($p = 0.055$). The deviation from controls was greater in the high PSR group than both the low and

Table 1
For all statistical analyses, household hemiparetic walkers were matched with controls walking at 0.3 m/s, limited community walkers with controls walking at 0.6 m/s, and community walkers with controls walking at 0.9 m/s.

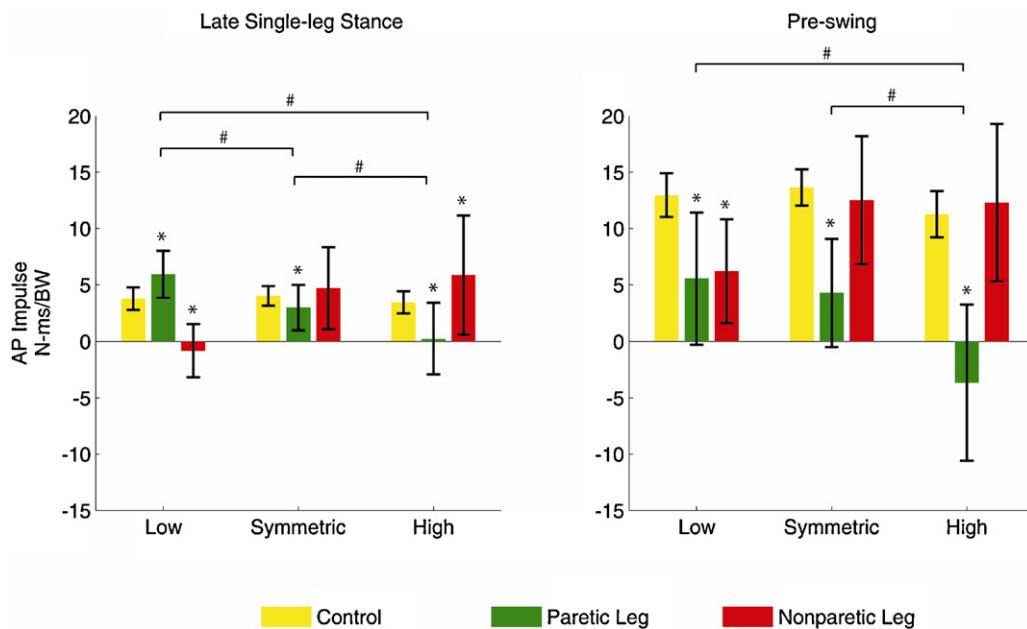| Functional walking status	Hemiparetic speed (m/s)	Matched control speed (m/s)	Total number of hemiparetic subjects (low, symmetric, high PSR)
Household	<0.4	0.3	29 (4, 4, 21)
Limited community	0.4–0.8	0.6	19 (4, 11, 4)
Community	>0.8	0.9	6 (1, 2, 3)

Fig. 1. AP impulse for control (yellow, average of right and left legs), paretic leg (green) and nonparetic leg (red) in late single-leg stance and pre-swing phases. Hemiparetic subgroups were determined by the paretic step ratio (PSR) (low: PSR < 0.465; symmetric: 0.465 ≤ PSR ≤ 0.535; high: PSR > 0.535). * denotes a significant difference relative to the controls, # denotes a significant difference between PSR groups; all $p < 0.05$.

symmetric group ($p = 0.038$ and 0.004) during late single-leg stance (see Fig. 2). Therefore the hypothesis that hemiparetic subjects with longer paretic than nonparetic steps would show reduced paretic leg plantarflexor moment was supported. In addition, compared to controls walking at a similar speed the high PSR group had significantly more nonparetic leg plantarflexor moment impulse ($p = 0.007$) and hip flexor moment impulse ($p = 0.006$) during late single-leg stance as well as a knee extensor versus flexor moment impulse that approached significance ($p = 0.065$) during late single-leg stance and was significant ($p = 0.027$) during pre-swing.

There were several significant joint moment impulse differences in the symmetric PSR group compared to speed-matched controls (see Fig. 2). Thus the hypothesis that subjects with symmetric steps would have similar joint moment impulses as speed-matched controls was not supported. In addition to a reduced paretic leg plantarflexor moment impulse, the symmetric PSR group had a significantly larger paretic leg hip flexor moment impulse during both paretic late single-leg stance and pre-swing ($p = 0.017$, 0.003), a larger nonparetic leg plantarflexor moment impulse ($p = 0.025$) and hip flexor moment impulse ($p = 0.017$) during nonparetic late single-leg stance, and an increase in nonparetic leg hip flexor moment impulse that approached significance ($p = 0.055$) during nonparetic pre-swing.

The low PSR group had no other significant joint moment impulse differences compared to speed-matched controls. Therefore the hypothesis that subjects with shorter paretic than nonparetic steps would have reduced paretic leg hip flexor moment impulses compared to speed-matched controls was not supported.

4. Discussion

The overall goal of this study was to determine if step length asymmetry in post-stroke hemiparetic subjects is indicative of underlying differences in joint moments during walking to gain insight into the observed impairments and compensatory strategies. All hemiparetic subjects were found to have paretic leg plantarflexor impairment (i.e., reduced plantarflexor moment

impulse), with the degree of plantarflexor impairment being related to the asymmetry group. In addition, PSR groups used different compensatory strategies to overcome this impairment: the high PSR group relied on the nonparetic leg and the symmetric group relied on a bilateral hip flexor strategy.

4.1. Paretic leg plantarflexor impairment is consistent across PSR groups

Paretic leg plantarflexor impairment is common among post-stroke hemiparetic subjects [9,10,16,17] and our study suggests that this impairment occurs in all step length asymmetry groups. The level of impairment, however, depends on step length asymmetry as the high PSR group had a greater paretic leg plantarflexor moment reduction compared to speed-matched controls than both the symmetric and low PSR groups. Consistent with previous studies that found the plantarflexors to be important for forward propulsion [7,8,18,19], the reduction was accompanied by a decrease in propulsion compared to speed-matched controls. Previous studies examining step length asymmetry have found both reduced paretic leg plantarflexor strength [11] and peak torque [4] result in a greater asymmetry. These studies defined step length asymmetry using an absolute value of the deviation from symmetry and were unable to differentiate between directions of asymmetry. However, most subjects walked with longer paretic than nonparetic steps (i.e. high PSR). Therefore, their results are consistent with ours that show a greater paretic leg plantarflexor moment impulse reduction in high PSR subjects than symmetric subjects. To our knowledge, no other study has found a difference in paretic leg plantarflexor impairment between subjects who walk with different directions of step length asymmetry. This is likely because studies examining step length asymmetry in the hemiparetic subjects include fewer subjects who walk with shorter paretic than nonparetic steps (i.e. low PSR) or did not differentiate subjects based on PSR. Our study shows that paretic leg plantarflexor impairment occurs across all asymmetry groups and the degree of impairment depends on the direction of step length asymmetry.
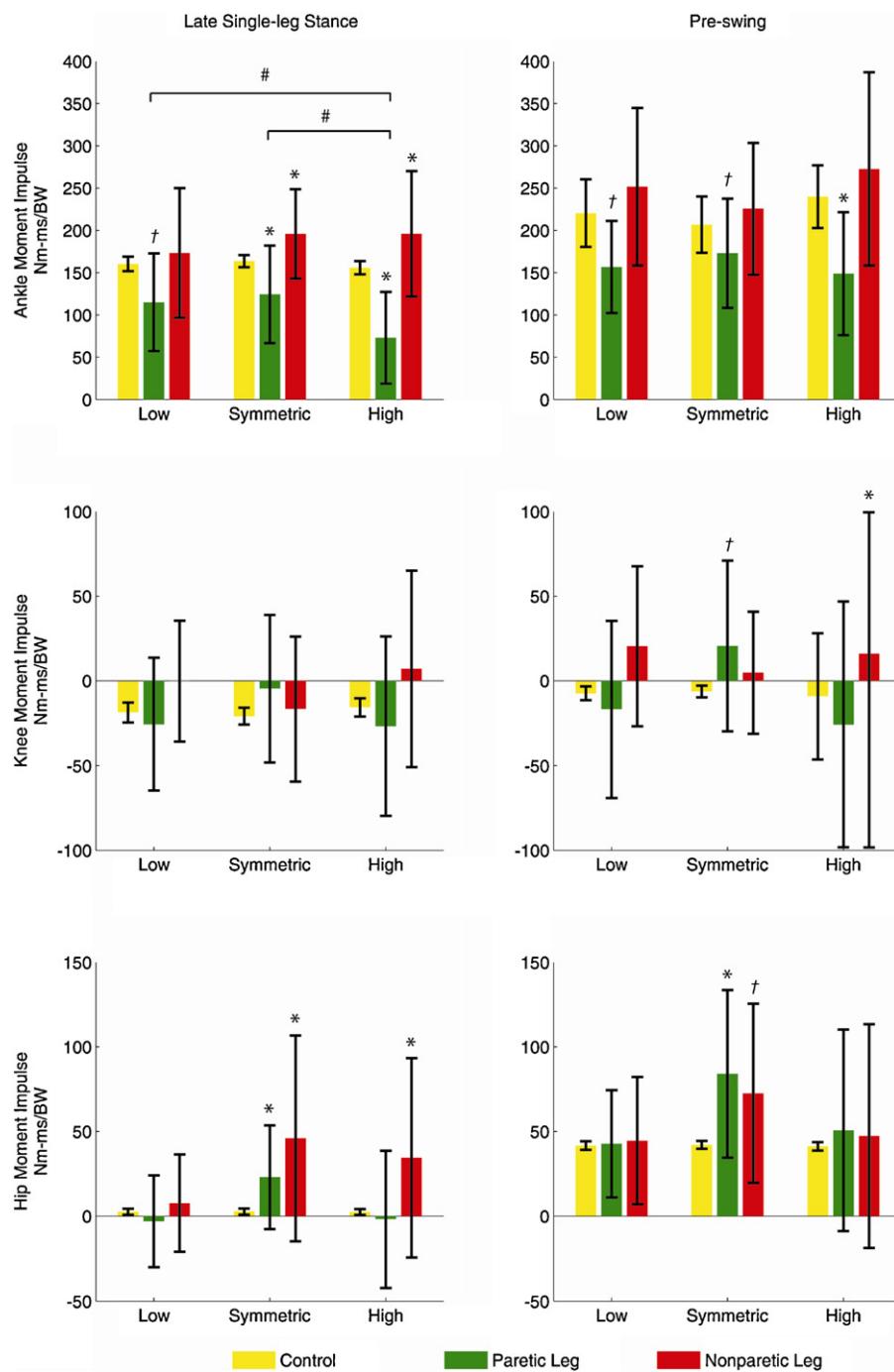
Fig. 2. Ankle (plantarflexor positive), knee (extensor positive), and hip (flexor positive) joint moment impulses for control (yellow, average of right and left legs), paretic leg (green), and nonparetic leg (red) in their respective late single-leg stance and pre-swing phases. Hemiparetic subgroups were determined by the paretic step ratio (PSR) (low: PSR < 0.465; symmetric: 0.465 ≤ PSR ≤ 0.535; high: PSR > 0.465). * and † denote statistical differences relative to the controls, $p < 0.05$ and $p < 0.1$; # denotes a significant difference between PSR groups, $p < 0.05$.

4.2. PSR is an indicator of compensatory mechanisms

To compensate for the impaired paretic leg plantarflexors, the high PSR group relied primarily on the nonparetic leg. Compared to speed-matched controls, the nonparetic leg plantarflexor moment was significantly increased during late single-leg stance. This was accompanied by increased nonparetic leg AP impulse, which is consistent with the plantarflexors as main contributors to forward propulsion [7,8,18]. By using the nonparetic leg plantarflexors to increase forward propulsion, the center-of-mass moves further forward while the paretic leg is in swing, thus increasing the

paretic step length [20]. In addition, the high PSR group had a knee extensor moment during both nonparetic pre-swing and late single-leg stance. The knee extensors have previously been shown to contribute to propulsion in both mid to late-stance [21] and pre-swing [22]. Similar to the nonparetic leg plantarflexors, the nonparetic leg knee extensors likely increase the paretic step length due to increased propulsion. These results are consistent with Hsu et al. [4] who found the strength of the nonparetic leg knee extensors correlates positively with the paretic step length.

To compensate for the impaired paretic plantarflexors, the symmetric PSR group altered other joint moments in order to walk

symmetrically. Therefore, the hypothesis that this group would exhibit similar joint moments as non-impaired controls was not supported. Instead, symmetric walkers appeared to utilize a bilateral hip strategy to compensate for the impaired paretic plantarflexors. This strategy utilizes the hip flexor muscles to accelerate the leg forward during pre-swing and swing [14] and is commonly used to overcome plantarflexor weakness [13,23–25]. While increased hip flexor moments act to increase swing initiation, a consequence is a reduction in propulsion as it acts to offload the leg and decrease the AP GRF impulse [19]. However, there was no significant reduction in nonparetic leg propulsion, which was likely due to the increased nonparetic leg plantarflexor output. Paretic leg propulsion, on the other hand, was decreased which is consistent with both the reduction in paretic leg plantarflexor output and increase in hip flexor output. Based on these results, symmetric PSR subjects seem limited primarily by paretic leg plantarflexor impairment.

The AP impulses found in the low PSR group were consistent with our suggestion that these subjects are unable to generate sufficient pre-swing energy to the leg due to impaired paretic plantarflexors and hip flexors. The reduced paretic AP impulse seen during pre-swing, which is a result of the reduced paretic plantarflexor moment impulse, would act to decrease the energy delivered to the leg and result in shorter paretic swing time and step length. This shorter swing time would also explain the significantly reduced nonparetic leg AP impulse. Unfortunately, no other significant differences in joint moment impulses consistent with this hypothesis were found. This is likely due to the smaller number of low PSR subjects combined with the high inter-subject variability. Further study including additional low PSR subjects is needed to elucidate any underlying joint compensation patterns in this group.

4.3. Limitations

A limitation of this study is that data were collected as subjects walked on a treadmill and previous studies have shown differences in treadmill and overground walking stride lengths [26,27]. However, rehabilitation using treadmill supported modalities [e.g. 28,29] is common and thus understanding walking biomechanics on a treadmill is directly applicable to these rehabilitation methods. In addition, a recent study found no fundamental difference in AP impulses between overground and treadmill walking [30] and we recently suggested that treadmill walking reveals the same motor control impairments in hemiparetic walking [15]. Therefore, we expect the treadmill to have a minimal effect on our results. Another limitation was that data were collected from subjects while walking without an assistive device regardless of whether they normally used one. However, if the overall goal is to improve walking ability such that the patient no longer has to walk with an assistive device, then understanding the impairments that are present when walking without an assistive device is important in developing rehabilitation strategies.

5. Conclusions

Paretic leg plantarflexor impairment (i.e., reduced net plantarflexor moment impulse during walking) consistently occurs in all post-stroke hemiparetic subjects regardless of step length asymmetry. Thus, clinicians should focus on improving the paretic leg plantarflexor output in all subjects during gait rehabilitation. The direction of asymmetry can be used to understand both the degree of paretic leg plantarflexor impairment and the compensatory mechanism used. Those subjects who walk with symmetric steps are able to compensate for plantarflexor impairment using similar output from both legs. Those subjects who walk with

longer paretic than nonparetic steps, on the other hand, typically rely on the nonparetic leg, and thus improving paretic leg output in these subjects may improve their walking symmetry.

Conflict of interest statement

None.

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