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Slow and faster post-stroke walkers have a different trunk progression and braking impulse during gait

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ABSTRACT

Background: Braking forces absorbed by the leading paretic limb are greater than expected with regard to gait speed and not correlated with propulsive forces generated by the non-paretic limb in individuals with severe hemiparesis. Altered foot placement due to poor sensorimotor capacities may explain excessive braking forces. **Research question:** The main objective of this study was to determine whether paretic foot placement was related to paretic braking forces in post-stroke individuals with various self-selected walking speeds and motor deficits. **Methods:** In this cross-sectional study, 34 chronic hemiparetic post-stroke individuals, divided into slow (< 0.7 m/s, $n = 17$) and faster ($n = 17$) subgroups, walked at their self-selected speed. Kinematic and kinetic parameters were measured. Braking impulses, peak braking forces, step characteristics and clinical status were compared between groups and limbs, and their correlations were tested using Pearson (or Spearman) correlation tests.

Results: On the paretic side, braking impulses and step length were similar between groups despite the slower walking speed in the slow group. Paretic peak braking forces and step length were correlated in both groups ($r = 0.5$). Paretic braking forces were correlated with walking speed, foot placement ahead of the pelvis, trunk progression (TP) from non-paretic initial contact to paretic initial contact, and better motor function of the paretic limb for the faster walkers ($0.6 < r < 0.7$), but not for the slow walkers. Among the slow walkers, reduced TP ahead of the paretic foot was correlated with a higher paretic impulse ($r = -0.6$).

Significance: Better motor function likely helped the faster walkers to decelerate their center of mass appropriately relative to their walking speed. In the slow hemiparetic walkers, TP ahead of the paretic foot was perturbed. Clinicians should therefore consider vasti and plantar flexor muscle tone and activity that likely restrict TP ahead of the paretic foot and increase braking forces.

1. Introduction

Walking speed is a major determinant for independent mobility about home and community [1]. Consequently, low walking speed after stroke is a negative factor for community ambulation [2,3] and social participation [4]. Among post-stroke individuals with chronic hemiparesis, the reported average walking speed is around 0.7 m/s (*standard deviation: 0.32 m/s*) [5]. One determining factor of slow walking speed after stroke is the propulsive capacity of the paretic lower limb [6]. The paretic propulsive phase characteristics have been extensively analyzed in terms of muscular contribution [7], joint moments [8], angular displacements [9] and its contribution to gait function [6].

During walking, the succession of propulsive and braking phases at each leg contributes to the control of the forward progression of the

body's center of mass (CoM) through modulations of its acceleration [10]. Unlike the propulsive phase, the paretic braking phase and its determinants have not been extensively studied. During normal steady-state gait, the braking forces absorbed by the leading limb are correlated with the trailing limb propulsive forces and both are of similar magnitude [11], whereas in stroke survivors, they are no longer correlated when the paretic limb is braking and the non-paretic limb is propelling during double support [12]. It reflects an altered control of the acceleration of the center of mass (CoM) that can affect the cost of walking and endurance [13].

In individuals with severe hemiparesis, the paretic braking impulse is increased compared to the non-paretic leg [14] and no longer correlated with gait speed [6]. It has been suggested that the increased paretic braking force may be a mechanical response to the paretic leg

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Table 1
Participant characteristics (mean (SD)) and clinical assessment (median [25th percentile–75th percentile] except for gait endurance: mean (SD)) with p-values indicating when groups values were different.

	Slow walker group (n = 17)	p-value	Faster walker group (n = 17)
General data			
Age (years)	62 (12)	< 0.05	52 (14)
Weight (kg)	74.7 (13.1)		75.2 (12.1)
Gender	5 W / 12 M		8 W / 9 M
Side of the hemiparesis	6 R / 11 L		7 R / 10 L
Time post-stroke (months)	83 (108)		55 (32)
Clinical assessment			
Muscle tone, CSI (/16 pts)	6 [4 – 8]		7 [4–10]
Balance, BBS (/56 pts)	47 [44 – 52]	< 0.001	55 [50–56]
Motor function, CMSA (/14 pts)	7 [5 – 8]	< 0.001	10 [8–12]
Gait endurance, 6MWT (m)	247 (99)	< 0.001	430 (113)

W: women; M: men; R: right; L: left. CSI: Composite Spasticity Index (16 = maximal hyperactive response to tendon jerk, a maximal resistance to passive displacement and sustained clonus); BBS: Berg Balance Scale (56 = lowest fall risk and good balance); CMSA: Chedoke McMaster Stroke Assessment (14 = highest stage of motor abilities, based on leg and foot components); 6MWT: Six-Minute Walk Test.

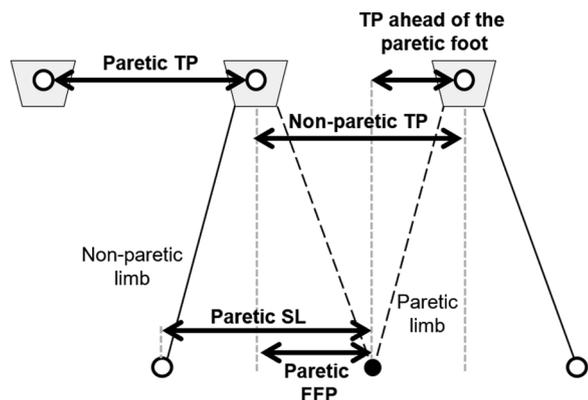


Fig. 1. Description of the step parameters: step length – SL, forward foot placement – FFP, trunk progression – TP and TP ahead of the foot on stance.

taking a longer step [6], since peak braking forces and braking impulses increase with a longer ipsilateral step length (SL) in healthy people [15]. However, SL is determined by trunk progression (TP) and foot forward placement (FFP), and their contribution to SL varies among post-stroke individuals [16]. A better understanding of the contribution of TP and FFP to the paretic braking force would support the development of interventions to restore an efficient relationship between paretic braking forces and non-paretic propulsive forces in individuals with severe hemiparesis, as a complement to training programs focused on paretic propulsion [17,18].

The main objective of this study was to examine the relationships between SL, TP and FFP and the paretic braking forces in post-stroke individuals walking slowly or faster than 0.7 m/s.

2. Methods

Thirty-four participants with chronic hemiparesis (unilateral stroke, 6 months or more, with residual weakness in the affected lower limb), who were able to walk 10 m independently with or without a cane, were included in the study. A clinical assessment was performed (Table 1): spasticity with the Composite Spasticity Index [19], balance with the Berg Balance Scale (BBS [20]), motor function with the Chedoke-McMaster Stroke Assessment (CMSA, lower limb function [21]),

Table 2
Mean (SD) and minimum and maximum [Min–Max] values for walking speed, cadence, braking and step parameters on the paretic and non-paretic side for the hemiparetic slow and faster walkers, with p-values indicating when group or side values were different.

	Slow walker group (n = 17)	p-value	Faster walker group (n = 17)
Walking speed (m/s)			
Walking speed	0.51 (0.15)	***	0.85 (0.17)
[Min–Max]	[0.27 – 0.68]		[0.70 – 1.44]
Cadence (steps/min)			
	83.7 (11.3)		80.1 (10.6)
[Min–Max]	[60.3 – 99.4]		[69.1 – 102.8]
Peak braking (N/kg)			
Paretic stance	–0.88 (0.30)	*	–1.15 (0.35)
[Min–Max]	[–0.39 – –1.46]		[–0.69 – –1.78]
Non-paretic stance	–0.67 (0.44)	**	–1.09 (0.41)
[Min–Max]	[–0.13 – –1.87]		[–0.48 – –1.68]
Braking impulse (N.s/kg)			
Paretic stance	–0.25 (0.08)		–0.25 (0.08)
[Min–Max]	[–0.10 – –0.43]	**	[–0.15 – –0.45]
Non-paretic stance	–0.15 (0.09)	**	–0.24 (0.1)
[Min–Max]	[–0.01 – –0.34]		[–0.12 – –0.50]
Step length (m)			
Paretic step	0.37 (0.09)		0.44 (0.11)
[Min–Max]	[0.16 – 0.52]	**	[0.25 – 0.66]
Non-paretic step	0.28 (0.12)	***	0.42 (0.11)
[Min–Max]	[0.08 – 0.46]		[0.27 – 0.65]
Foot forward placement (m)			
At the paretic initial contact	0.22 (0.04)	***	0.28 (0.05)
[Min–Max]	[0.16 – 0.28]		[0.17 – 0.38]
At the non-paretic initial contact	0.22 (0.07)	***	0.29 (0.05)
[Min–Max]	[0.09 – 0.30]		[0.23 – 0.36]
Trunk progression (m)			
Paretic step	0.37 (0.11)	**	0.47 (0.10)
[Min–Max]	[0.20 – 0.63]	**	[0.33 – 0.67]
Non-paretic step	0.29 (0.08)	***	0.43 (0.10)
[Min–Max]	[0.14 – 0.42]		[0.25 – 0.69]
Trunk progression (TP) ahead of the foot on stance (m)			
Paretic TP ahead of the non-paretic foot	0.15 (0.08)		0.18 (0.07)
[Min–Max]	[0.02 – 0.34]	**	[0.07 – 0.31]
Non-paretic TP ahead of the paretic foot	0.07 (0.06)	**	0.15 (0.06)
[Min–Max]	[–0.02 – 0.19]		[0.06 – 0.31]

*: p < 0.05; **: p < 0.01; ***: p < 0.001.

and gait endurance with the 6-Minute Walk Test (6MWT [22]). The ethics board approved the study, and informed consent was obtained from each participant.

2.1. Protocol

The participants walked on an instrumented walkway equipped with three embedded AMTI force plates. An Optotrak 3020 Motion Capture System (NDI) was used to measure kinematic data.

After a familiarization period, participants were asked to walk at their self-selected speed. Kinematic and kinetic data were collected during five trials. An average of three gait cycles with a similar walking speed was used for analysis. A cut-off value of 0.7 m/s was used to divide the participants into two subgroups: slow and faster walkers [5].

2.2. Data processing

Based on the antero-posterior (AP) component of the ground reaction forces, the negative (braking) phase was identified, and time integrated over the entire stance phase (impulse); its maximum (peak) amplitude was also identified.

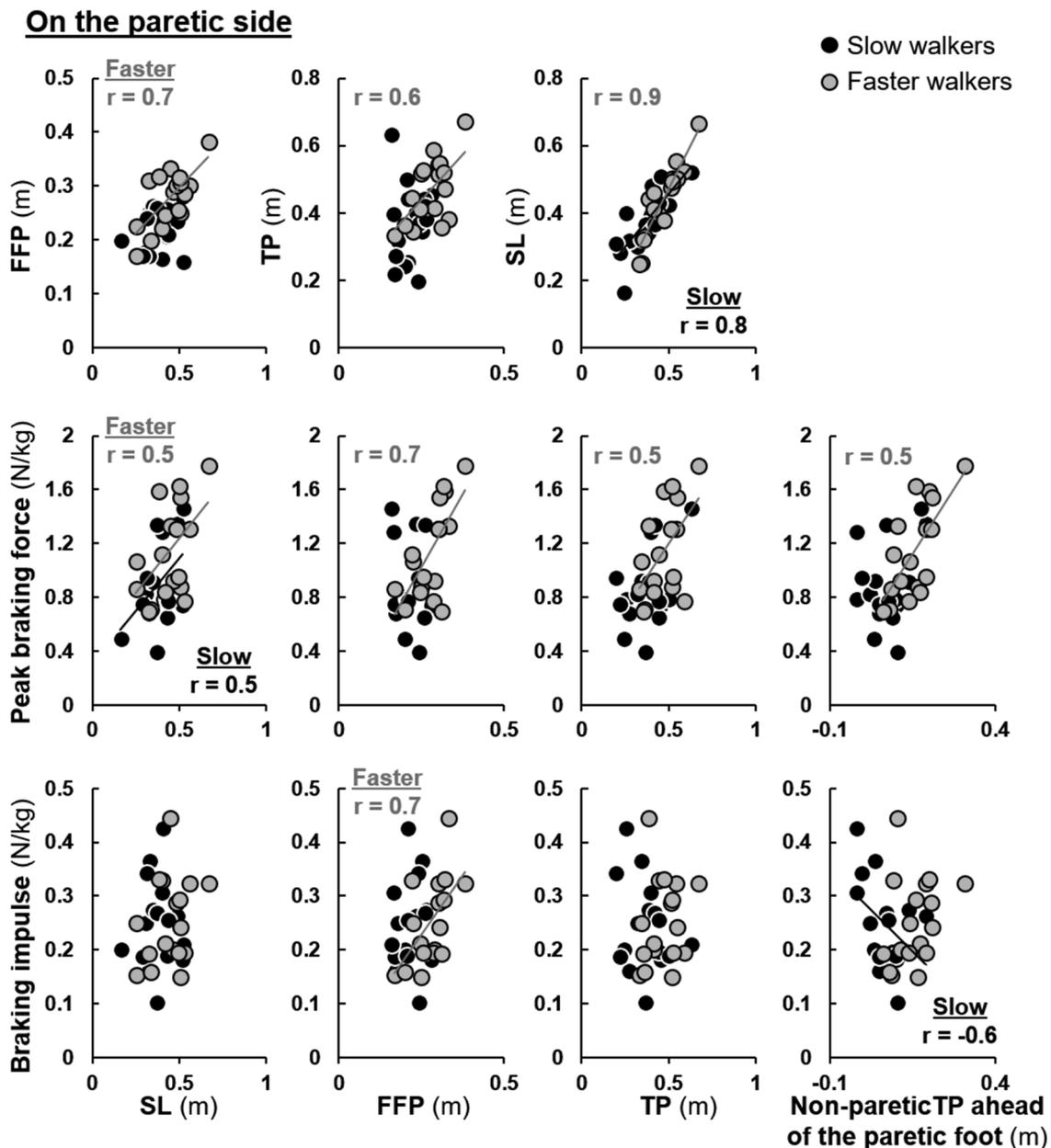


Fig. 2. Scatterplots showing the relationships between step parameters (step length – SL, forward foot placement – FFP, trunk progression – TP and TP ahead of the foot on stance) and braking parameters (peak and impulse) on the paretic side, for the hemiparetic slow (black circle) and faster (grey circle) walkers. Significant relationships for each group are indicated ($p < 0.05$).

The AP position of the pelvis CoM and right and left ankle joint centers were determined based on kinematic and anthropometric data. SL was defined as the distance between the ankle joint centers at two successive initial contacts (IC). At each IC, FFP was calculated as the AP distance between the pelvis CoM and the leading ankle joint center. In addition, paretic TP was defined as the AP distance traveled by the pelvis CoM from the IC of the non-paretic limb to the IC of the paretic limb [16] and vice-versa for non-paretic TP (Fig. 1).

2.3. Statistical analysis

A Shapiro-Wilk test confirmed the normal distribution of data. Participant characteristics were compared between groups with an unpaired student t-test. Braking and step parameters were analyzed using repeated measures two-way (Group \times Side) ANOVAs. For each group, the relationships between braking parameters and (1) ipsilateral step parameters and walking speed were determined using Pearson's

correlation coefficient, and (2) clinical scores were determined using Spearman's rank correlation coefficient. Only significant correlations ($p < 0.05$) with coefficients ≥ 0.5 were reported.

3. Results

Seventeen hemiparetic participants were included in each group (< 0.7 , slow speed; and ≥ 0.7 m/s, faster speed). Groups were similar for general data, except that the slow group was older (mean (SD): 62 (12) vs 52 (14) years; $p < 0.01$; Table 1). The slow walkers had greater balance, motor and locomotor deficits than the faster walkers. No participant used a walking aid during the data collection.

On the paretic side, braking impulses and SL were similar between groups while peak braking force differed (Table 2). For the slow walkers, SL on the paretic side was significantly longer than SL on the non-paretic side. FFP did not differ between sides, but paretic TP was longer than non-paretic TP in both groups. FFP and TP were the shortest

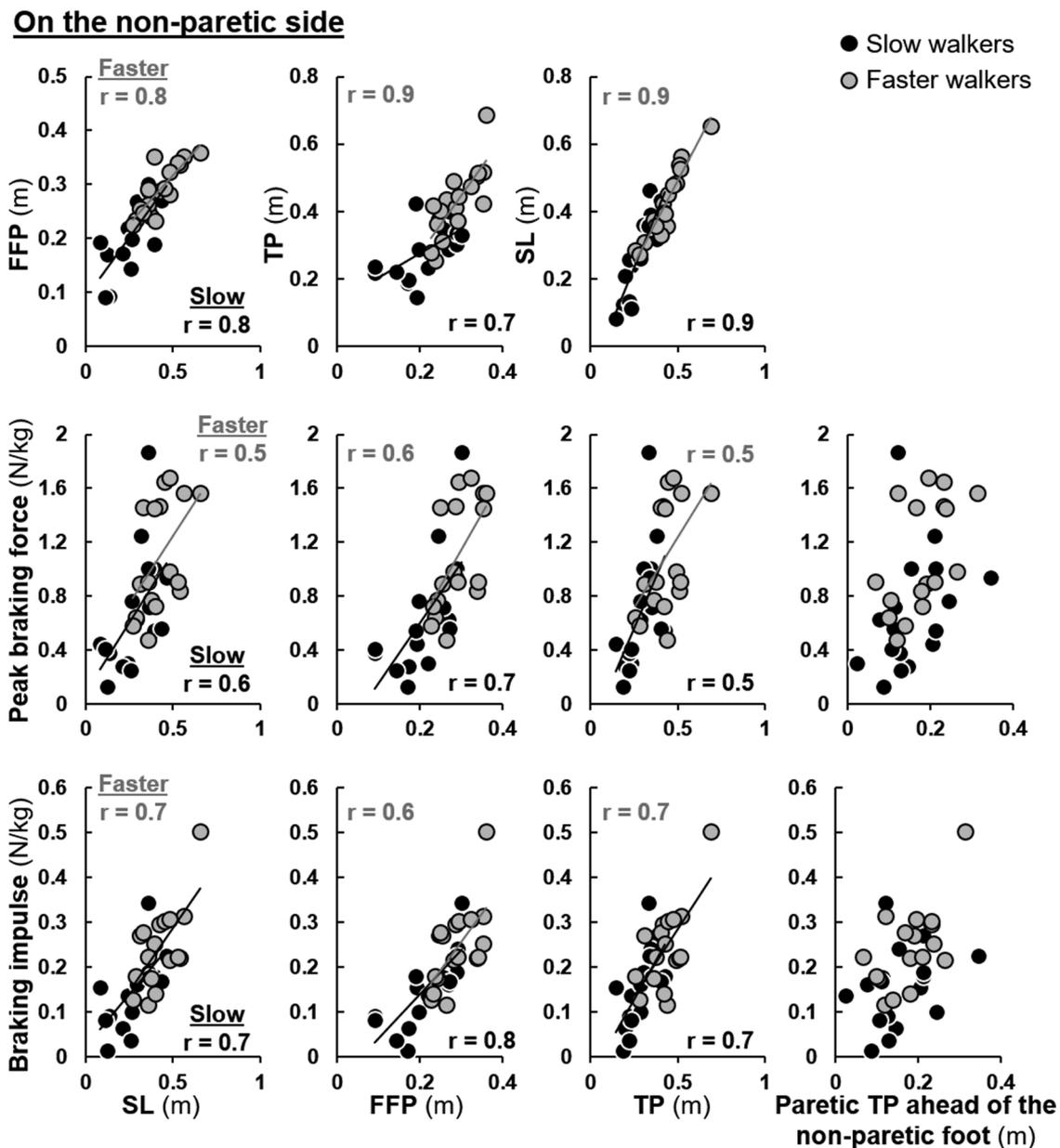


Fig. 3. Scatterplots showing the relationships between step parameters (step length – SL, forward foot placement – FFP, trunk progression – TP and TP ahead of the foot on stance) and braking parameters (peak and impulse) on the non-paretic side, for the hemiparetic slow (black circle) and faster (grey circle) walkers. Significant relationships for each group are indicated ($p < 0.05$).

in slow walkers (Table 2).

In the faster group, SL, FFP and TP were significantly correlated (Fig. 2). Paretic peak braking force increased with longer SL, FFP and TP, with faster gait speed ($r = 0.6$, $p = 0.01$) and better motor function of the paretic limb ($r = 0.7$, $p < 0.01$). In addition, paretic braking impulse was correlated with FFP and motor function ($r = 0.7$, $p < 0.01$).

For the slow group, paretic SL and TP were correlated, but not FFP (Fig. 2). Paretic peak braking force was correlated with SL, but the relationship between paretic braking forces (peak and impulse) and paretic FFP, paretic TP and walking speed did not reach statistical significance ($p > 0.1$).

On the non-paretic side, step parameters and braking forces were significantly correlated in both groups (Fig. 3).

A further analysis of TP (see Fig. 1) showed that non-paretic TP ahead of the paretic foot was significantly and negatively correlated with paretic braking impulse during paretic stance in the slow group (r

$= -0.6$, $p < 0.05$; Fig. 2). This TP ahead of the paretic foot was significantly lower in the slow group compared to the non-paretic side and the faster group (Table 2).

4. Discussion

Severity of post-stroke locomotor impairments affected the relationships between SL, TP and FFP and the paretic braking forces. Our results showed that the peak of braking paretic force was of less magnitude in post-stroke individuals walking under 0.7 m/s than in post-stroke individuals walking faster. It was not the case for the braking impulse that was similar for the two groups. In addition to the magnitude of the force, braking impulse takes into account the time to generate the force; this time was thus longer in slow walkers. Regarding the step characteristics, the associations with the braking force parameters were complex. They varied with the side of the body (paretic and non-paretic) and the group of walking speed (slow vs. faster). Step

characteristics had little association with the braking force parameters on the affected side in the slow group while they were related on the non-paretic side in all groups. Nevertheless, a specific part of the non-paretic TP was related to paretic braking impulse in slow walkers, i.e. the progression of the trunk ahead on the paretic foot.

In the faster walkers, peak braking forces increased as expected with gait speed and with the foot placed further ahead of the trailing foot and ahead of the pelvis, as previously reported for healthy subjects [15]. Better motor function likely helped the faster walkers to generate paretic SL and decelerate their CoM appropriately relative to their walking speed, as revealed by the positive correlations between braking forces, and walking speed and motor function.

In the slow group, the mean peak paretic braking force was of lower magnitude than in the faster group, as expected due to the slower gait speed. However, the paretic impulse was similar between groups, as previously observed [14], instead of also being lower. The magnitude of the braking force therefore seems appropriate for the paretic leading limb at the IC but may be applied over an extended period of time in slow walkers, as underlined by the higher impulse on the paretic side. It can be explained by poor motor function assessed in the slow group, in accordance with Turns and al. who observed abnormal muscle activity of vastus lateralis and biceps femoris in early stance, specifically in individuals with severe hemiparesis [14].

In the slow group, the paretic SL was longer than the non-paretic SL. However, FFP was symmetrical, contrary to what was previously observed [23] in very slow walkers (≤ 0.3 m/s) who likely had to reduce their non-paretic FFP to respond to limited paretic propulsion [16]. No relationship was observed between paretic braking forces and paretic TP and FFP. Thus, in slow walkers, the progression of the trunk when the paretic limb is swinging, and then the placement of the paretic foot in front of the pelvis were not associated with the corresponding braking force. However, a significant and negative relationship was found for the non-paretic TP ahead of the paretic foot on stance on the ground. Thus, the higher the paretic braking forces and deceleration of CoM, the smaller the TP ahead of the paretic foot is. The braking impulse after IC likely restricts progression of the trunk ahead of the paretic foot and decreases non-paretic TP and SL. The excessive braking impulse observed is likely due to increased activity of the vasti muscles that prolongs the deceleration phase of the CoM [14] and the plantar flexor muscles that actively and passively control TP [24,25], during the early and late paretic stance phases.

In conclusion, paretic SL, TP and FFP are not related to excessive paretic braking impulses in slow walkers. Clinicians should consider action of paretic vasti and plantar flexors as they reduce the TP during paretic stance and may cause abnormal braking forces. Studies should continue to assess braking and propulsive phases and consider determining relation with asymmetry parameters.

Declarations of interest

None.

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