Gait & Posture xxx (2008) xxx–xxx

Contents lists available at ScienceDirect

Gait & Posture

journal homepage: www.elsevier.com/locate/gaitpost

Capacity to increase walking speed is limited by impaired hip and ankle power generation in lower functioning persons post-stroke

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ARTICLE INFO

Article history:
Received 17 April 2007
Received in revised form 14 July 2008
Accepted 23 July 2008

Keywords:
Stroke
Gait
Kinematics
Power
Biomechanics

ABSTRACT

It is well known that stroke patients walk with reduced speed, but their potential to increase walking speed can also be impaired and has not been thoroughly investigated. We hypothesized that failure to effectively recruit both hip flexor and ankle plantarflexor muscles of the paretic side limits the potential to increase walking speed in lower functioning hemiparetic subjects. To test this hypothesis, we measured gait kinematics and kinetics of 12 persons with hemiparesis following stroke at self-selected and fast walking conditions. Two groups were identified: (1) lower functioning subjects ($n = 6$) who increased normalized walking speed from 0.52 leg lengths/s (ll/s, SEM: 0.04) to 0.72 ll/s (SEM: 0.03) and (2) higher functioning subjects ($n = 6$) who increased walking speed from 0.88 ll/s (SEM: 0.04) to 1.4 ll/s (SEM 0.03). Changes in spatiotemporal parameters, joint kinematics and kinetics between self-selected and fast walking were compared to control subjects examined at matched walking speeds (0.35 ll/s (SEM: 0.03), 0.63 ll/s (SEM: 0.03), 0.92 ll/s (SEM: 0.04) and 1.4 ll/s (SEM: 0.04)). Similar to speed-matched controls, the higher functioning hemiparetic subjects increased paretic limb hip flexion power and ankle plantarflexion power to increase walking speed. The lower functioning hemiparetic subjects did not increase power generation at the hip or ankle to increase walking speed. This observation suggests that impaired ankle power generation combined with saturation of hip power generation limits the potential to increase walking speed in lower functioning hemiparetic subjects.

1. Introduction

Gait in persons post-stroke is typically slower compared to non-disabled individuals. Several studies have related impaired walking speed in post-stroke hemiparesis to muscle weakness, spasticity, and impaired balance and sensation [1–9]. Comparison of gait performance in fast and self-selected speeds using biomechanical analysis has the potential to further delineate factors limiting gait performance in hemiparetic persons. Testing the ability to increase gait speed may reveal impairments of locomotor function other than the impairments reflected by reduced walking speed alone.

In non-disabled persons, strategies employed to change from slow to free and fast walking conditions have been documented in terms of changes in joint angles, moments and power, as well as muscle coordination [10–14]. The combination of increased ankle power generation and increased hip power generation has been proposed as an important mechanism in increasing walking speed [10,12–14]. Requiao et al. [15] analyzed muscle utilization ratio and concluded that an increased contribution of both ankle plantarflexors and hip flexors is associated with increased walking speed in control subjects. Nadeau et al. [8] used the muscle utilization ratio with hemiparetic subjects and determined that additional recruitment of the hip flexor muscles was required to achieve faster walking speeds in the presence of plantarflexor weakness. Milot et al. [16] reported a shift towards similar muscular utilization levels of ankle plantarflexors and hip flexors at higher speeds. These studies focused on hemiparetic persons able to walk at relatively normal walking speeds. Little is known about the mechanisms used to increase walking speed in hemiparetic persons with significantly decreased locomotor function.

Our study compared the mechanisms used to increase walking speed in two groups of hemiparetic persons demonstrating higher and lower levels of locomotor function as classified according to walking speed in self-selected and fast conditions. We evaluated the changes in joint kinematics and joint powers that occurred

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doi:10.1016/j.gaitpost.2008.07.010

between self-selected and fast walking in these subjects and compared these observations to data collected from control subjects walking at comparable speeds.

Based on previous findings [8,16], we hypothesized that higher functioning hemiparetic subjects would use the same mechanisms to increase speed as control subjects, but lower functioning hemiparetic subjects would fail to effectively recruit both the hip flexors and ankle plantarflexor muscles of the paretic side. This failure in lower functioning hemiparetic subjects would not only limit the potential to increase walking speed but would also introduce compensations on the non-paretic side.

2. Subjects and methods

The study sample included 12 persons with post-stroke hemiparesis who were able to walk at least 10 m without an ankle foot orthosis or walking aid. Subject characteristics and demographics are presented in Table 1. Control data were collected from a group of 10 subjects (six males and four females) with average age of 43 years (S.D.: 11.6) and no major orthopedic or neurologic pathology affecting their gait performance. All procedures were approved by the Stanford University panels on human subjects in research, and all subjects gave informed consent.

Instrumented gait analysis was undertaken using a seven camera motion capture system (Qualysis, Inc., Goteborg, Sweden, 240 Hz) with three synchronized force plates (AMTI, Watertown, MA, USA and Bertec, Columbus, OH, USA, 100 Hz). A modified Cleveland Clinic marker placement protocol was used (38 markers). Subjects wore their usual footwear. For each condition, a minimum of three valid trials were collected for the paretic and non-paretic limbs.

Control subjects were initially studied at their self-selected walking speed. They were then asked to reduce their walking speed progressively to slow, slower and very slow. This procedure produced walking speeds that averaged 1.41 m/s (SEM 0.03), 0.97 m/s (SEM 0.04), 0.63 m/s (SEM 0.01) and 0.38 m/s (SEM 0.02) for the four conditions, respectively.

Hemiparetic subjects were tested in two walking conditions: self-selected speed (SS) and maximal speed without compromising safety (FAST). Hemiparetic subjects were subsequently classified in two sub-groups based on walking speed normalized with respect to leg length. The first group, "higher functioning hemiparetic" (HFH) subjects ($n = 6$), walked at a self-selected normalized walking speed greater than half of the normalized self-selected walking speed of the control subjects. They were able to increase walking speed to the level of self-selected walking speed in controls. Average walking speed of this HFH group ranged from 0.78 m/s (SEM 0.04) for self-selected speed to 1.25 m/s (SEM 0.12) for the fast speed. The second group, "lower functioning hemiparetic" (LFH) subjects ($n = 6$), walked at a self-selected normalized walking speed of less than half of the normalized self-selected walking speed of the control subjects. They were unable to reach the level of self-selected walking speed in controls. Average walking speed in this LFH group ranged from 0.45 m/s (SEM 0.03) for the self-selected speed to 0.62 m/s (SEM 0.03) for the fast speed.

2.1. Data analysis

To facilitate statistical comparisons between different sized subjects, walking speed parameters were divided by subject's leg length and expressed as leg lengths/s (ll/s). The average leg length in the patient population was 0.87 m (SEM 0.01 m), in the control population the average leg length was 0.91 m (SEM 0.01 m). Joint kinematics were calculated using Visual 3D (C-Motion, Inc., Rockville, MD, USA) and expressed with respect to the gait cycle. Joint powers were divided by body mass.

We determined the maximal and minimal values of pelvic rotation, hip flexion-extension, knee flexion-extension and ankle plantar-dorsiflexion at initial contact (IC) and toe off (TO) and during loading response (LR), single stance (SST), pre-swing (PS) and swing (S). These data for the hemiparetic subjects are illustrated at both self-selected and fast speed in conjunction with the speed-matched data obtained from the control subjects (Figs. 1 and 2). Maximal and minimal values of the joint powers at the hip, knee and ankle were extracted and related to specific features of the joint power profiles:

Hip:

H1: Hip extensor power generation (concentric action).
H2: Hip flexor power absorption (eccentric action).
H3: Hip flexor power generation (concentric action).

Knee:

K3: Knee extensor power absorption (eccentric action).

Ankle:

A1: Ankle plantarflexor power absorption (eccentric action)
A2: Ankle plantarflexor power generation (concentric action).

Table 1
Overview of demographics of the hemiparetic stroke subjects and Fugl-Meyer Motor Assessment scores (lower extremity portion)

	Gender	Side stroke	Age (years)	Time post-stroke (months)	FM – Total (/100)
HFH-subjects					
1	F	L	45	15	90
2	M	R	66	22	90
3	M	R	75	15	91
4	M	R	54	16	87
5	M	L	61	14	87
6	M	L	63	13	90
Average (S.D.)			60.7 (10.3)	15.8 (3.2)	89.2 (1.7)
LFH-subjects					
1	F	L	52	16	86
2	M	R	56	17	86
3	M	R	69	6	84
4	M	L	45	8	80
5	F	R	59	9	80
6	M	L	60	8	79
Average (S.D.)			57.8 (8.6)	9.6 (4.3)	82.5 (3.2)

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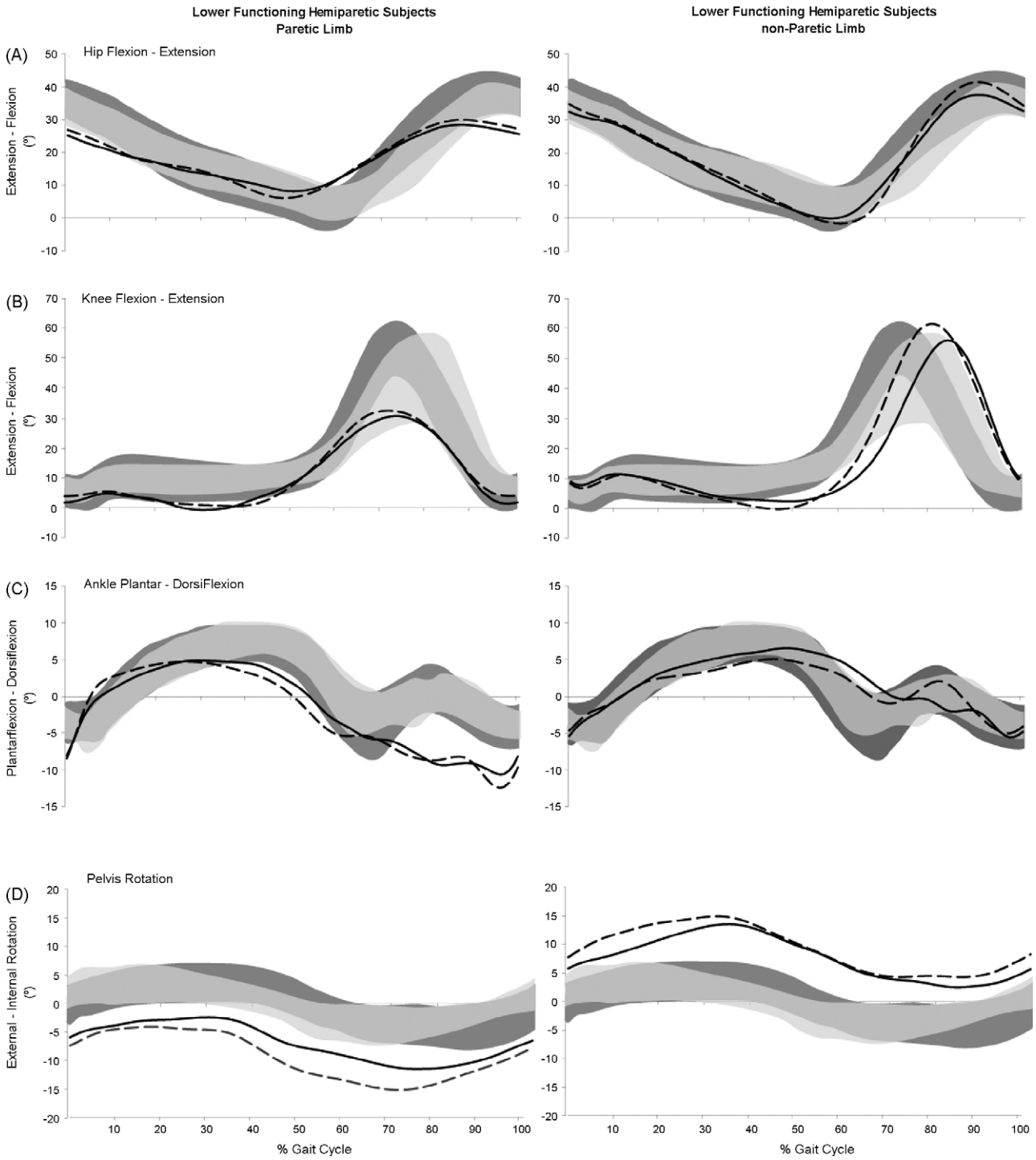

Fig. 1. Sagittal plane joint kinematics of hip, knee and ankle as well as pelvis rotation for the lower functioning stroke subjects walking at self-selected (solid) and fast speed (dashed) with superposition of the relevant speed related controls (average ± S.D., slow speed is indicated in light gray and fast speed in dark gray).

Figs. 3 and 4 present the average joint power profiles at hip and ankle for the hemiparetic subjects and speed-matched control subjects at self-selected and fast speeds in conjunction with speed-matched data obtained from control subjects.

2.2. Statistical analysis

Changes due to increased walking speed of control subjects and hemiparetic subjects were compared using a Wilcoxon Signed-rank statistic for paired data sets. To test for differences between hemiparetic subjects and speed-matched controls, a Kruskall–Wallis test was used. For the higher functioning hemiparetic group, data at self-selected and fast walking speed were compared to control subjects walking at 66% of self-selected and self-selected walking speed, respectively. For the lower functioning hemiparetic group, data from the self-selected and fast walking conditions were compared to control subjects walking at 25% of self-selected walking speed and 45% of the self-selected walking speed, respectively. Statistical significance is reported rounded to $p < 0.1$ (^), $p < 0.05$ (*) and $p < 0.01$ (**).

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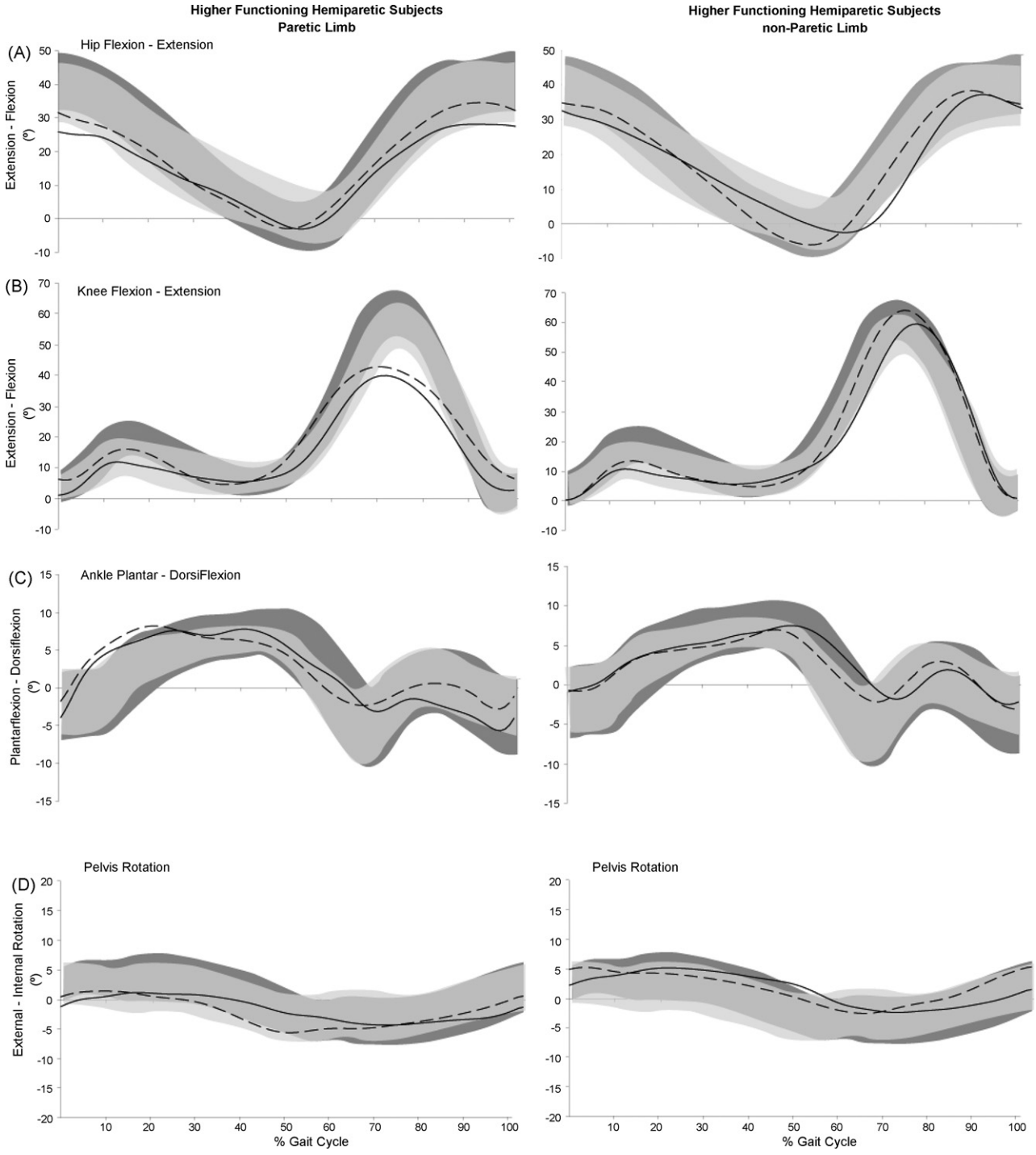

Fig. 2. Sagittal plane joint kinematics of hip, knee and ankle as well as pelvis rotation for the higher functioning stroke subjects walking at self-selected (solid) and fast speed (dashed) with superposition of the relevant speed related controls (average ± S.D., slow speed is indicated in light gray and fast speed in dark gray).

3. Results

Age and mean time since stroke were similar between the hemiparetic subjects (Table 1). LFH-subjects had significantly lower scores on the lower extremity portion of the Fugl-Meyer Motor Assessment ($p < 0.05$).

3.1. Spatiotemporal parameters

Between self-selected and fast walking conditions, the average walking speed increased from 0.88 ll/s (SEM: 0.04) to 1.4 ll/s (SEM: 0.03) in HFH-subjects and from 0.52 ll/s (SEM: 0.04) to 0.72 ll/s (SEM: 0.03) in LFH-subjects. These values corresponded to 25%, 45% and 66% and 100% of SS walking speed of the control subjects (Tables 2A and 2B). The average walking speed in both self-selected and fast conditions differed statistically between LFH and HFH ($p < 0.01$) distinguishing these groups on the basis of biomechanical function.

To increase walking speed, control subjects decreased stride duration and increased stride length. Decreased stride duration results from reduced total stance duration and especially reduced duration of single limb stance phase. Increases in both step and

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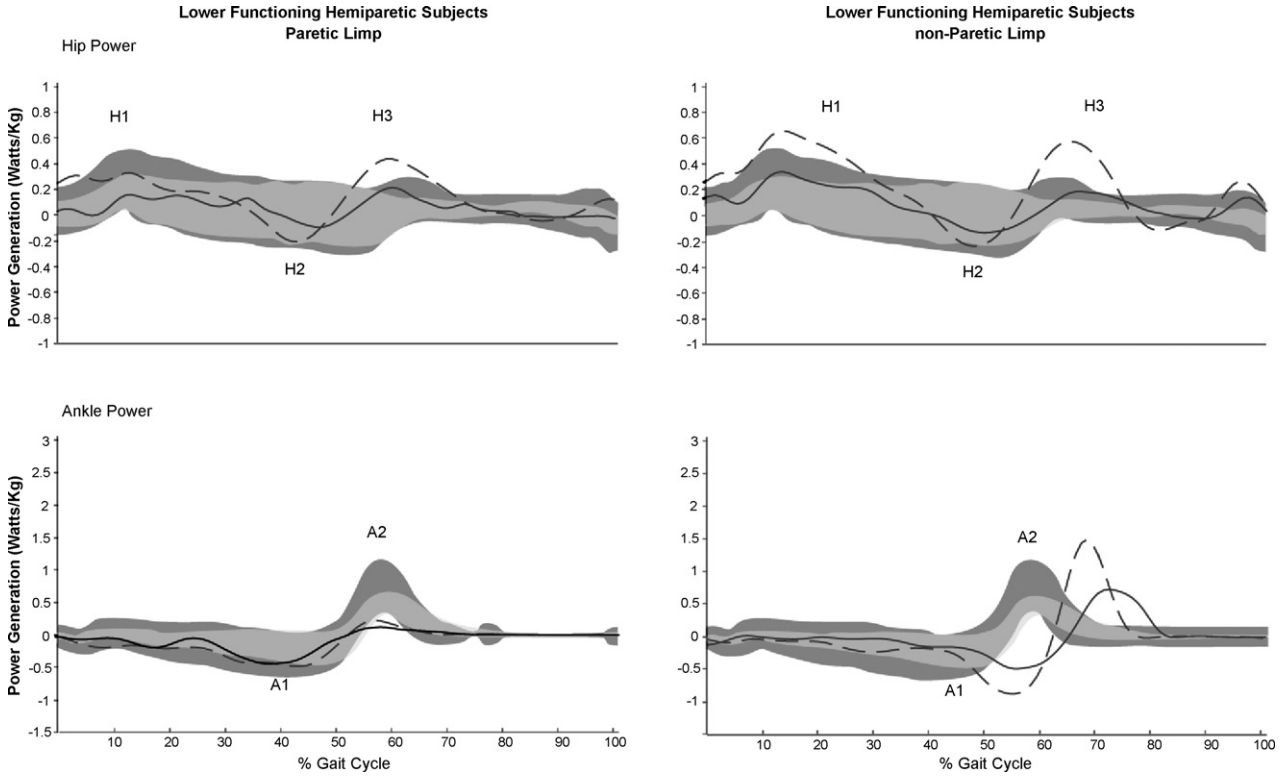

Fig. 3. Joint power at hip and ankle for the lower functioning hemiparetic subjects walking at self-selected (solid) and fast speed (dashed) with superposition of the relevant speed related controls (average ± S.D., slow speed is indicated in light gray and fast speed in dark gray).

swing length contributed to increased normalized stride length (Tables 2A and 2B). Neither hemiparetic subject group shortened the duration of the paretic limb single limb stance phase or increased their swing length with increased walking speed.

3.2. Kinematics

With increased walking speed, control subjects demonstrated increased range of motion in the sagittal plane: At the hip, flexion

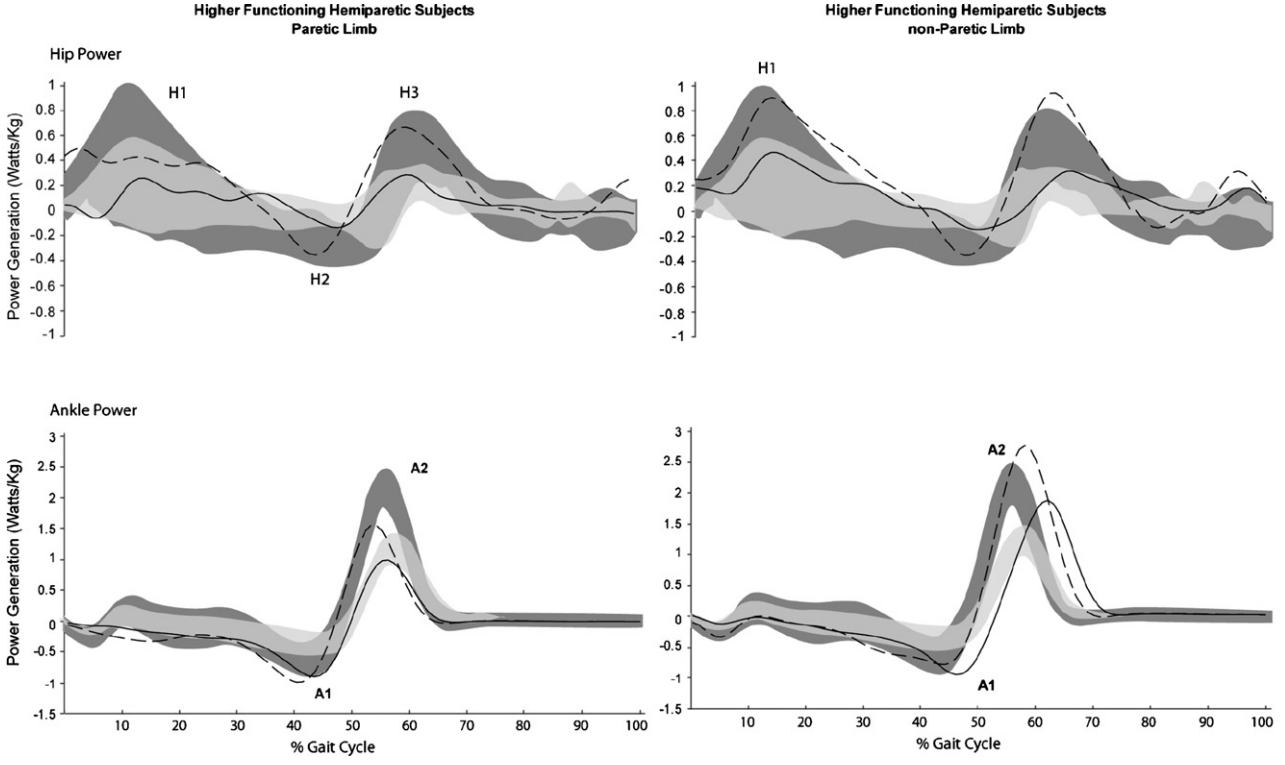

Fig. 4. Joint power at hip and ankle for the higher functioning hemiparetic subjects walking at self-selected (solid) and fast speed (dashed) with superposition of the relevant speed related controls (average ± S.D., slow speed is indicated in light gray and fast speed in dark gray).

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Table 2A
Average (SEM) of walking velocity, stride duration and stride length in hemiparetic subjects and speed-matched control, walking at self-selected and fast condition

	Self-selected condition	Fast condition	Sign
Walking velocity (ll/s)			
Higher functioning hemiparetic subjects	0.88 (± 0.04)	1.4 (± 0.03)	**
Speed-matched control subjects	0.92 (± 0.04)	1.4 (± 0.04)	*
Lower functioning hemiparetic subjects	0.52 (± 0.04)	0.72 (± 0.03)	**
Speed-matched control subjects	0.35 (± 0.03)	0.63 (± 0.02)	*
Stride length (/ll)			
Higher functioning hemiparetic subjects	1.17 (± 0.03)	1.42 (± 0.04)	**
Speed-matched control subjects	1.37 (± 0.04)	1.59 (± 0.04)	*
Lower functioning hemiparetic subjects	0.81 (± 0.05)	0.93 (± 0.06)	**
Speed-matched control subjects	1.0 (± 0.04)	1.25 (± 0.03)	*
Stride duration (s)			
Higher functioning hemiparetic subjects	1.34 (± 0.04)	1.04 (± 0.07)	**
Speed-matched control subjects	1.53 (± 0.04)	1.17 (± 0.02)	*
Lower functioning hemiparetic subjects	1.56 (± 0.07)	1.28 (± 0.05)	**
Speed-matched control subjects	2.91 (± 0.15)	1.97 (± 0.06)	*

Significance is tested between self-selected and fast as well as between stroke subjects and speed-matched controls with (^) $p < 0.1$, (*) $p < 0.05$ and (**) $p < 0.01$.

Table 2B
Average (SEM) of selected spatiotemporal parameters in hemiparetic subjects and speed-matched control subjects, walking at self-selected and fast condition

	Self-selected condition	Fast condition	Sign
Stance duration (s)			
Higher functioning hemiparetic subjects – paretic limb	0.83 (± 0.03)	0.61 (± 0.04)	**
Higher functioning hemiparetic subjects – non-paretic limb	0.92 (± 0.03)	0.69 (± 0.05)	*
Speed-matched control subjects	1.0 (± 0.03)	0.72 (± 0.01)	*
Lower functioning hemiparetic subjects – paretic limb	1.02 (± 0.07)	0.82 (± 0.05)	**
Lower functioning hemiparetic subjects – non-paretic limb	1.22 (± 0.07)	0.94 (± 0.04)	*
Speed-matched control subjects	2.15 (± 0.14)	1.3 (± 0.04)	*
Single stance duration (s)			
Higher functioning hemiparetic subjects – paretic limb	0.41 (±0.02)	0.36 (± 0.02)	ns
Higher functioning hemiparetic subjects – non-paretic limb	0.51 (± 0.02)	0.44 (± 0.01)	*
Speed-matched control subjects	0.53 (±0.01)	0.43 (± 0.03)	**
Lower functioning hemiparetic subjects – paretic limb	0.37 (±0.03)	0.34 (± 0.02)	ns
Lower functioning hemiparetic subjects – non-paretic limb	0.55 (± 0.03)	0.48 (± 0.03)	*
Speed-matched control subjects	0.76 (± 0.02)	0.67 (± 0.03)	**
Cadence (steps/min)			
Higher functioning hemiparetic subjects – paretic limb	203 (± 52)	338 (± 112)	*
Higher functioning hemiparetic subjects – non-paretic limb	171 (± 38)	248 (± 60)	*
Speed-matched control subjects	169 (± 28)	266 (± 50)	**
Lower functioning hemiparetic subjects – paretic limb	139 (± 32)	187 (± 40)	*
Lower functioning hemiparetic subjects – non-paretic limb	129 (± 30)	165 (± 38)	*
Speed-matched control subjects	71.31 (± 13)	129 (± 21)	**
Step length (ll)			
Higher functioning hemiparetic subjects – paretic limb	0.59 (± 0.02)	0.71 (± 0.03)	*
Higher functioning hemiparetic subjects – non-paretic limb	0.62 (± 0.01)	0.74 (± 0.02)	*
Speed-matched control subjects	0.71 (± 0.02)	0.82 (± 0.02)	**
Lower functioning hemiparetic subjects – paretic limb	0.45 (± 0.02)	0.51 (± 0.02)	*
Lower functioning hemiparetic subjects – non-paretic limb	0.5 (± 0.04)	0.54 (± 0.04)	ns
Speed-matched control subjects	0.52 (± 0.01)	0.63 (± 0.01)	**
Swing length (ll)			
Higher functioning hemiparetic subjects – paretic limb	1.11 (± 0.1)	1.35 (± 0.06)	ns
Higher functioning hemiparetic subjects – non-paretic limb	1.01 (± 0.03)	1.21 (± 0.04)	*
Speed-matched control subjects	0.9 (± 0.09)	1.17 (± 0.03)	**
Lower functioning hemiparetic subjects – paretic limb	0.82 (± 0.19)	1.16 (± 0.15)	ns
Lower functioning hemiparetic subjects – non-paretic limb	0.67 (± 0.04)	0.77 (± 0.04)	*
Speed-matched control subjects	0.78 (± 0.03)	0.99 (± 0.03)	**

Significance is tested between self-selected and fast as well as between stroke subjects (paretic and non paretic limb) and speed-matched controls with (^) $p < 0.1$, (*) $p < 0.05$ and (**) $p < 0.01$.

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Table 3
Average (SEM) of hip, ankle and knee power in hemiparetic subjects and speed-matched control subjects, walking at self-selected and fast condition

	Self-selected condition	Fast condition	Sign
H1 (W/kg)			
Higher functioning hemiparetic subjects – paretic limb	0.37 (± 0.08)	0.67 (± 0.27)	ns
Higher functioning hemiparetic subjects – non-paretic limb	0.58 (± 0.04)	0.96 (± 0.23)	ns
Speed-matched control subjects	0.37 (± 0.08)	0.59 (± 0.12)	**
Lower functioning hemiparetic subjects – paretic limb	0.20 (± 0.04)	0.34 (± 0.11)	ns
Lower functioning hemiparetic subjects – non-paretic limb	0.43 (± 0.06)	0.51 (± 0.08)	ns
Speed-matched control subjects	0.22 (± 0.03)	0.34 (± 0.05)	**
H2 (W/kg)			
Higher functioning hemiparetic subjects – paretic limb	−0.25 (± 0.06)	−0.43 (± 0.1)	ns
Higher functioning hemiparetic subjects – non-paretic limb	−0.09 (± 0.02)	−0.27 (± 0.13)	ns
Speed-natched control subjects	−0.17 (± 0.03)	−0.31 (± 0.04)	**
Lower functioning hemiparetic subjects – paretic limb	−0.11 (± 0.04)	−0.14 (± 0.04)	ns
Lower functioning hemiparetic subjects – non-paretic limb	−0.11 (± 0.02)	−0.18 (± 0.08)	ns
Speed-matched control subjects	−0.06 (± 0.01)	−0.09 (± 0.01)	^
H3 – PS (W/kg)			
Higher functioning hemiparetic subjects – paretic limb	0.41 (± 0.05)	0.71 (± 0.25)	^
Higher functioning hemiparetic subjects – non-paretic limb	0.44 (± 0.05)	0.87 (± 0.27)	ns
Speed-matched control subjects	0.3 (± 0.04)	0.56 (± 0.06)	**
Lower functioning hemiparetic subjects – paretic limb	0.33 (± 0.07)	0.36 (± 0.06)	ns
Lower functioning hemiparetic subjects – non-paretic limb	0.31 (± 0.07)	0.44 (± 0.09)	ns
Speed-matched control subjects	0.13 (± 0.01)	0.21 (± 0.02)	**
H3 – SW (W/kg)			
Higher functioning hemiparetic subjects – paretic limb	0.24 (± 0.07)	0.72 (±0.25)	*
Higher functioning hemiparetic subjects – non-paretic limb	0.31 (± 0.03)	0.85 (± 0.25)	^
Speed-matched control subjects	0.33 (± 0.07)	0.63 (± 0.06)	**
Lower functioning hemiparetic subjects – paretic limb	0.21 (± 0.07)	0.26 (± 0.04)	ns
Lower functioning hemiparetic subjects – non-paretic limb	0.25 (± 0.03)	0.31 (± 0.03)	^
Speed-matched control subjects	0.12 (± 0.01)	0.16 (± 0.03)	^
A1 (W/kg)			
Higher functioning hemiparetic subjects – paretic limb	−1.03 (± 0.17)	−1.176 (± 0.31)	ns
Higher functioning hemiparetic subjects – non-paretic limb	−1.18 (± 0.11)	−0.96 (± 0.16)	ns
Speed-matched control subjects	−0.63 (± 0.06)	−0.78 (± 0.06)	**
Lower functioning hemiparetic subjects – paretic limb	−0.6 (± 0.04)	−0.99 (± 0.08)	ns
Lower functioning hemiparetic subjects – non-paretic limb	−0.65 (± 0.11)	−1.03 (± 0.06)	*
Speed-matched control subjects	−0.38 (± 0.03)	−0.48 (± 0.05)	**
A2 (W/kg)			
Higher functioning hemiparetic subjects – paretic limb	1.22 (± 0.3)	1.78 (± 0.51)	^
Higher functioning hemiparetic subjects – non-paretic limb	2.22 (± 0.36)	2.57 (± 0.55)	ns
Speed-matched control subjects	1.46 (± 0.11)	2.41 (± 0.13)	**
Lower functioning hemiparetic subjects – paretic limb	0.29 (± 0.07)	0.44 (± 0.08)	ns
Lower functioning hemiparetic subjects – non-paretic limb	1.42 (± 0.24)	0.86 (± 0.29)	ns
Speed-matched control subjects	0.59 (± 0.07)	0.89 (± 0.07)	**
K3 (W/kg)			
Higher functioning hemiparetic subjects – paretic limb	−0.29 (± 0.09)	−0.73 (± 0.22)	*
Higher functioning hemiparetic subjects – non-paretic limb	−0.36 (± 0.06)	−0.9 (± 0.24)	^
Speed-matched control subjects	−0.35 (± 0.04)	−0.34 (± 0.09)	ns
Lower functioning hemiparetic subjects – paretic limb	−0.19 (± 0.03)	−0.3 (± 0.06)	ns
Lower functioning hemiparetic subjects – non-paretic limb	−0.31 (± 0.05)	−0.3 (± 0.05)	ns
Speed-matched control subjects	−0.17 (± 0.02)	−0.3 (± 0.04)	**

Significance is tested between self-selected and fast as well as between stroke subjects (paretic and non paretic) and speed-matched controls with (^) $p < 0.1$, (*) $p < 0.05$ and (**) $p < 0.01$.

increased at IC, during LR, SST, at TO and during swing, whereas extension increased during SST and during PS. Peak knee flexion increased during swing. Knee flexion during LR and SST increased. Ankle plantarflexion decreased during LR. Internal pelvic rotation increased at IC and LR while external pelvic rotation increased during PS. The stance phase increase in internal pelvic rotation reached statistical significance only during the slow walking condition (Figs. 1 and 2).

With increased walking speed, *HFH-subjects* used a strategy similar to control subjects with increased hip flexion at IC, during LR and swing and increased hip extension during SST. The increase in paretic limb hip extension during PS was less than in controls. At the knee, *HFH-subjects* increased peak flexion during swing but failed to increase knee flexion during LR and SST. At the ankle, no changes in kinematics with walking speed were detected. No changes in pelvic kinematics were observed.

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With increased walking speed, *LFH-subjects* failed to increase hip flexion at IC, during LR and swing. The increased hip extension during SST observed in control subjects was preserved. At the knee, none of the changes seen in the control subjects were observed. In contrast, *LFH-subjects* demonstrated increased knee flexion of the paretic limb at IC. Decreased ankle dorsiflexion of the paretic ankle was observed during PS. At the pelvis, LFH-subjects demonstrated increased external rotation of the paretic hemipelvis during LR and SST at fast walking speeds.

3.3. Joint powers

With increased walking speed, *control subjects* produced increased ankle plantarflexor power (A2) during PS in conjunction with increased hip flexor power during PS, at TO and during SW (H3). Furthermore, hip extensor power generation was increased during LR (H1) and persisted into SST at the fastest speed (Table 3; Figs. 3 and 4).

HFH subjects showed a similar strategy to increase walking speed as observed in control subjects. Plantarflexor power generation was increased during pre-swing (A2) of the paretic limb. For the non-paretic limb, a similar trend was found. Furthermore, hip flexor power generation during pre-swing and swing (H3) increased for the paretic and non-paretic limb. However, HFH-subjects failed to sufficiently increase hip power generation (H1) during LR for either the paretic or non-paretic limbs.

LFH subjects failed to adopt a similar strategy to increase walking speed as speed-matched controls. They failed to increase paretic limb ankle plantarflexor power generation (A2) during PS or hip flexor power generation during PS, at TO and Swing (H3). Hip flexor power generation during PS, at TO and Swing (H3) was increased only in the non-paretic limb. Furthermore, LFH-subjects failed to sufficiently increase hip power generation (H1) during LR in either the paretic or non-paretic limbs.

With increased walking speed, *control subjects* increased hip flexor power absorption during SST and PS (H2). At the slowest walking speed, knee power absorption during PS (K3) and at TO also increased with increasing speed. Plantarflexor power absorption during SST (A1) increased for both speeds (Table 3; Figs. 3 and 4).

Neither group of hemiparetic subjects increased power absorption (H2) during PS. In contrast to the control subjects, *LFH-subjects* did not demonstrate increased knee extensor power absorption (K3) during PS or at TO. *HFH-subjects* demonstrated increased knee extensor power absorption (K3) during PS. Neither group of hemiparetic subjects demonstrated increased plantarflexor power absorption in the paretic limb during SST (A1).

4. Discussion

This study analyzed biomechanical mechanisms contributing to gait speed modulation between self-selected and faster walking speeds in hemiparetic persons and compared these strategies to non-disabled control subjects walking over comparable speed ranges. We found that higher functioning hemiparetic subjects and control subjects increased both ankle plantarflexion power and hip flexor power to increase walking speed, whereas lower functioning hemiparetic subjects failed to demonstrate this mechanism and had more limited ability to modulate walking speed. Differentiation between hemiparetic subjects and comparison to normal data advances understanding of gait dysfunction after stroke and elucidates whether gait deviations result from pathology, functional compensation or simply result from walking more slowly than normal. These distinctions enable identification of specific gait impairments and can help identify targets for rehabilitation.

Gait speed is often used to characterize hemiparetic severity [9,19]. Subjects characterized as lower or higher functioning, based on self-selected and fast walking speed, also demonstrated differences in their clinical examination (lower extremity Fugl-Meyer Motor scores) and gait kinetics. Inclusion of lower functioning hemiparetic subjects in the present study extended understanding of hemiparetic gait dysfunction to more severely affected individuals. We reported data from hemiparetic subjects at markedly lower walking speeds compared to previous studies [8] (average walking speed of 0.47 m/s compared to 0.73 m/s). Despite the small sample size, this study identified and differentiated biomechanical mechanisms underlying gait dysfunction in these two groups of hemiparetic subjects.

4.1. Gait speed modulation

The potential for hemiparetic persons to modulate gait speed has been limited to analysis of changes in the spatiotemporal parameters [17]. Comparison of gait speed modulation in two functionally distinct groups of hemiparetic subjects, and comparison to control subjects over a comparable range of walking speeds offered an opportunity to determine the extent to which increased walking speed relied on normal or compensatory mechanisms.

Our observations suggest that impaired gait speed modulation in hemiparetic subjects results from inability to modify the duration of the different phases of gait including failure to decrease the duration of single stance and inability to increase swing length of the paretic limb. These limitations were revealed in both the higher and lower functioning hemiparetic subjects.

In comparison to controls, higher functioning hemiparetic subjects were hindered in increasing stride length as a result of limitations in both hemipelvis rotational range of motion and hip extension during pre-swing. Moreover, higher functioning hemiparetic subjects were unable to use additional knee flexion to increase shock absorption during LR and SST. In the lower functioning hemiparetic group gait speed modulation was further compromised by increased retraction of the paretic hemipelvis during the first half of stance. Additional limitations to forward progression included decreased ankle dorsiflexion range of motion during stance and deficient hip and knee flexion range of motion during swing, which impaired limb clearance. Inability to increase hip flexion during swing and at initial contact, coupled with increased knee flexion at initial contact, limited swing length in these lower functioning hemiparetic subjects.

4.2. Joint power: primary impairment or functional compensation?

To increase walking speed, higher functioning hemiparetic subjects increased paretic limb plantarflexor power generation (A2, average increase of 55% compared to 95% in speed-matched controls) and hip flexor generation (H3, average increase of 41% compared to 31% in speed-matched controls). This difference is consistent with previous findings [8,16] indicating that hemiparetic subjects preferably engage hip flexor power generation to compensate for plantarflexor muscle weakness. In contrast, lower functioning hemiparetic subjects failed to increase power generation at the paretic ankle (A2, average increase of 14% compared to 30% in the speed-matched controls) coupled with only a minor increase of the already-excessive paretic limb hip flexor power generation (H3, average increase of 5% compared to 10% in the speed-matched controls).

Please cite this article in press as: Jonkers I, et al. Capacity to increase walking speed is limited by impaired hip and ankle power generation in lower functioning persons post-stroke. Gait Posture (2008), doi:10.1016/j.gaitpost.2008.07.010

Higher functioning hemiparetic subjects were able to generate increased ankle plantarflexor power when adjusting from self-selected to fast walking. This is an important observation indicating that maximal power generating capacity is not fully engaged during self-selected walking in higher functioning hemiparetic subjects. In conjunction with preferential recruitment of excessive hip flexor power, submaximal plantarflexor power suggests that gait speed modulation in HFH-subjects resulted from a compensatory propulsive strategy rather than limitations induced by impairments in the maximal muscle power generating capacity. Similar results have been observed in elders demonstrating low physical performance [18].

It is important to note that power absorption by the hip flexors (H2) and ankle plantarflexors (A1) did not increase when hemiparetic subjects walked at higher speeds. Similarly, no additional power absorption was observed at the knee (K3) in the LFH-subjects at higher speeds. This observation suggests that increased walking speed is not associated with excessive restraint of these muscle groups.

Lower functioning hemiparetic subjects engaged excessive plantarflexor power generation at self-selected walking speeds. No further increase was revealed during the fast condition, suggesting a saturation of this mechanism. In higher functioning subjects modulation of plantarflexor power was preserved.

It can be expected that the extent to which hemiparetic persons were able to modulate walking performance through physiologic control mechanisms related to the level of motor recovery. Our results demonstrated that changes in spatiotemporal, kinematic and kinetic data in the higher functioning hemiparetic subjects (i.e. subjects presenting higher self-selected walking speed), were more similar to changes observed in speed-matched control subjects. It appears, therefore, that these subjects relied to a large extent on normal control rather than compensatory mechanisms of the paretic and non-paretic limbs to increase walking speed.

4.3. Role of biomechanical gait mechanisms

It has been suggested that treadmill walking speed increased over the course of acute post-stroke recovery [19]. The present study demonstrated the biomechanical mechanisms used by hemiparetic subjects to increase walking speed. Analysis of the biomechanical parameters, kinematics and kinetics, characterizing the nature of the underlying control strategies should be further explored to identify their role in the functional classification of hemiparetic gait as well for their role as indicators of sensorimotor recovery.

The findings of the present study support our hypothesis that saturation of ankle plantarflexor power and inability to recruit additional hip flexor power limit walking speed in lower functioning hemiparetic subjects.

Conflict of interest

I, Ilse Jonkers, hereby certify that there are no conflicts of interest.

Acknowledgements

Ilse Jonkers is a Postdoctoral Fellow of the Research Foundation, Flanders and receives additional funding from the Belgian Educational Foundation and the Koning Boudewijn Fonds. This work was supported by VA RR&D Merit Review Grant no. B2792R to CP. We thank Abigail Andrade, C. Maria Kim, M.Sc., PT, Kirsten Unfried, M.S., and Lise C. Worthen, M.S. for their assistance in collecting and reducing the kinematic data and Marilynn Wyatt, PT, MA for suggestions to a previous version of this manuscript. The scientific responsibility remains with its authors.

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