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Capacity to Increase Walking Speed is Limited by impaired hip and ankle power generation in lower functioning persons post-stroke

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Abstract

While limited walking speed characterizes gait in the majority of persons post-stroke, the potential to increase walking speed can also be markedly impaired and has not been thoroughly investigated. We hypothesized that failure to effectively recruit both hip flexor and 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 mechanics of twelve persons with post-stroke hemiparesis 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/sec (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 data collected at matched 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 control subjects, the higher functioning hemiparetic subjects increased paretic limb hip flexion power and ankle plantarflexion power to increase walking speed. The lower functioning subjects demonstrated impaired ankle power generation combined with excessive power generation at the paretic hip during pre-swing at their self selected speed. This lower functioning group 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.

Keywords

Stroke; gait; kinematics; power; biomechanics

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, balance and impaired sensation [1,2,3,4,⁵,6,7,8,9].

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. The ability to increase walking speed reflects the capacity to modulate gait performance. This may reveal other significant impairments of locomotor function than 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, joint moments and powers, and muscle coordination [10,11,12,13,14]. The combination of increased ankle power generation and increased hip power generation has been proposed as a major mechanism to increase walking speed [10,12,13,14]. Requiao et al. analyzed the muscle utilization ratio concluding that an increased contribution of both plantarflexors and hip flexors is associated with increased walking speed in control subjects [15]. Nadeau [8] used the muscle utilization ratio with hemiparetic subjects and determined that, in the presence of plantarflexor weakness, additional recruitment of the hip flexor muscles was required to achieve faster walking speeds. Milot et al [16] reported a shift towards similar muscular utilization levels of plantarflexors and hip flexors at higher speeds. However, these studies focus on hemiparetic persons capable of walking at relatively normal walking speeds. Little is known about the mechanisms used to increase walking speed in hemiparetic persons with lower locomotor function.

The purpose of this study was to compare the biomechanical 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 (SS) and fast conditions. We evaluated the changes in joint kinematics and joint powers that occurred 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 in contrast to higher functioning hemiparetic subjects (HFH), lower functioning hemiparetic subjects (LFH) fail to effectively recruit both the hip flexors and plantarflexor muscles of the paretic side. This failure not only limits the potential to increase walking speed but introduces the need for compensations on the non-paretic side.

Subjects and Methods

All procedures were approved by the Stanford University panels on human subjects in research.

The study sample included twelve persons with post-stroke hemiparesis who were capable of walking at least 10 m without assistance of either an ankle foot orthosis or walking aid. Subject characteristics and demographics are presented in Table 1. Control data were collected in a group of 10 non-disabled subjects (6 males – 4 females) with average age of 43 years (SD: 11.6) and no major orthopedic or neurologic pathology affecting their gait performance.

Instrumented gait analysis was performed using a 7 camera digital motion capture system (Qualysis, Inc., Goteborg, Sweden, 240 Hz) with 3 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 customary footwear. For each condition, a minimum of three valid trials were collected for the paretic and non-paretic limbs.

Control subjects were studied at their self-selected walking speed after which they were 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:

- 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 in non-disabled controls and were able to increase walking speed to the range of self-selected walking speed in non-disabled controls. Average walking speed in 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.
- Lower functioning hemiparetic (LFH) subjects (n=6) walked at a self-selected normalized walking speed less than half of the normalized self-selected walking speed in non-disabled controls and were unable to reach the range of self-selected walking speed in non-disabled 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.

Data Analysis

To facilitate statistical comparisons between different sized subjects, spatiotemporal parameters were normalized 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 0.01m); in the control population the average leg length was 0.91 m (SEM 0.01m). 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 weight.

We determined the maximal and minimal values of pelvic rotation, hip flexion -extension, knee flexion-extension and ankle plantar- and dorsiflexion at initial contact (IC) and toe off (TO) and during loading reponse (LR), single stance (SST), preswing (PS) and swing (S).

These data are illustrated for the hemiparetic subjects at both self-selected and fast speeds in conjunction with the speed-matched data obtained from non-disabled controls (Figures 1A–1B).

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).

Figure 2A–2B presents 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 non-disabled control subjects.

Statistical Analysis

Changes due to increased walking speed in non-disabled control subjects and persons with post-stroke hemiparesis were compared using a Wilcoxon Signed-rank statistic for paired data sets. To test differences between hemiparetic subjects and speed-matched controls, a Kruskal-Wallis test was used. For the HFH-group, data at self-selected and fast walking speeds were compared to non-disabled control subjects at 66% of self-selected and self-selected walking speed, respectively. For the LFH-group, data from the self-selected and fast walking conditions were compared to control data 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 $p < 0.01$ (**).

Results

Age and mean time since stroke were similar between the hemiparetic subject groups ($p > .05$). LFH-subjects revealed significantly lower scores on the lower extremity portion of the Fugl-Meyer Motor Assessment ($p < 0.05$; Table 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 correspond to 25%, 45% and 66% and 100% of SS walking speed of the control subjects (Table 2). 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 increase cadence, decrease stride duration and increase 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 swing length contribute to increased normalized stride length (Table 2A–2B).

A similar strategy to increase walking speed was observed in both groups of hemiparetic subjects as compared to control subjects. However, both hemiparetic subject groups fail to significantly shorten the duration of the paretic limb single limb stance phase. In addition, increased paretic limb swing length failed to reach statistical significance in either group of hemiparetic subjects (Table 2A–2B).

Kinematics

With increased walking speed, control subjects demonstrate increased ROM in the sagittal plane:

At the hip, flexion increases at IC, during LR, SST, at TO and during swing, whereas extension increases during SST and, in the fast condition only, during PS. At the knee, peak knee flexion increases during swing. Knee flexion during LR and SST increases only in the fast walking condition. Ankle plantarflexion decreases during LR. At the pelvis, internal pelvic rotation increases at IC and LR while external pelvic rotation increases during PS. The stance phase increase in internal pelvic rotation reached statistical significance only during the slow walking condition (Figure 1).

With increased walking speed, *HFH-subjects* produce 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 is however less than in controls. At the knee, *HFH-subjects* increase peak flexion during swing but fail to increase knee flexion during LR and SST. At the ankle, no changes in kinematics were found. None of these changes in pelvic kinematics observed were revealed in control subjects.

With increased walking speed, *LFH-subjects* fail to increase hip flexion at IC, during LR and swing. The increased hip extension during SST observed in control subjects is preserved. At the knee, none of the changes observed in the control subjects is confirmed. In contrast, *LFH-subjects* demonstrate 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* demonstrate increased external rotation of the paretic hemipelvis during LR and SST at fast walking speeds.

Joint Powers

With increased walking speed, *control subjects* produce 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 is increased during LR (H1) and persists into SST at the fastest speed (Table 3- Figure 2).

HFH subjects show a similar strategy to increase walking speed as observed in speed matched controls. Plantarflexor power generation is increased during preswing (A2) the paretic limb. For the non-paretic limb, a similar trend is found. Furthermore, hip flexor power generation during preswing and swing (H3) increases for the paretic and non-paretic limb. However, *HFH-subjects* fail to sufficiently increase hip power generation (H1) during LR for either the paretic or non-paretic limbs.

LFH subjects fail to adopt a similar strategy to increase walking speed as speed matched controls. They fail to either 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) is increased in only the non-paretic limb. Furthermore, *LFH-subjects* fail to sufficiently increase hip power generation (H1) during LR in either the paretic or non-paretic limbs (Table 3- Figure 2).

With increased walking speed, *control subjects* increase hip flexor power absorption during SST and PS (H2). In the slowest walking speed, knee power absorption during PS (K3) and at TO is also increased. Plantarflexor power absorption during SST (A1) increases for both speeds (Table 3- Figure 2).

Neither hemiparetic subject group increased power absorption (H2) during PS. In contrast to the non-disabled control subjects, *LFH-subjects* do not demonstrate increased knee extensor power absorption (K3) during PS or at TO. In contrast, *HFH-subjects* do demonstrate increased knee extensor power absorption (K3) during PS. Neither group of hemiparetic subjects demonstrates increased plantarflexor power absorption in the paretic limb during SST (A1) (Table 3).

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 plantarflexion power and

hip flexor power to increase walking speed, whereas lower functioning hemiparetic subjects failed to demonstrate this mechanism and produced more limited ability to modulate walking speed. Differentiation between hemiparetic subject groups and comparison to reference normal data advance understanding of gait dysfunction post-stroke and elucidate 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 which can identify targets for rehabilitation.

Gait speed is often used as a means to characterize hemiparetic severity [9,19]. Our characterization of subjects as lower and higher functioning involved both functional (self-selected and fast walking speed) and clinical (lower extremity Fugl-Meyer Motor scores) criteria and revealed distinctly different groups at all levels: functional, clinical and biomechanical. Inclusion of lower functioning hemiparetic subjects in the present study extends understanding of hemiparetic gait dysfunction to more severely affected individuals. We report 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 clearly identifies and differentiates biomechanical mechanisms underlying gait dysfunction in these two distinct groups of hemiparetic subjects.

Gait Speed Modulation

The potential for hemiparetic persons to modulate gait speed has not been explored in great detail. Indeed, reports available in the literature are limited to analysis of changes in the spatio-temporal parameters [17]. Comparison of gait speed modulation in two functionally distinct groups of hemiparetic subjects, and further, comparison to control subjects over a comparable range of walking speeds thus affords a significant opportunity to determine the extent to which increased walking speed relies 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 both failure of the paretic limb to sufficiently decrease the duration of single stance and inability to increase swing length. These limitations were revealed in both the higher and lower functioning hemiparetic subject groups but indicate that the relative timing of stance and swing remain unchanged in hemiparetic gait.

Relative to control subjects, HFH-subjects are hindered in increasing stride length as a result of limitations in both hemipelvis rotational ROM and hip extension during preswing. Moreover, HFH-subjects are unable to engage additional knee flexion to increase shock absorption during LR and SST. In the LFH-group gait speed modulation is further compromised by increased retraction of the paretic hemipelvis during the first half of stance. Additional limitations to forward progression include: decreased ankle dorsiflexion ROM during stance and deficient hip and knee flexion ROM during swing which impairs limb clearance. Inability to increase hip flexion during swing and at initial contact, coupled with increased knee flexion at initial contact drastically limits changes in swing length in these LFH-subjects.

Joint Power: Primary Impairment or Functional Compensation?

To increase walking speed, HFH-subjects increase 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 observed difference in biomechanical strategy corresponds with previous findings [8,16] indicating that hemiparetic subjects preferably engage hip flexor power generation to compensate for plantarflexor muscle weakness. In contrast, LFH-subjects fail 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).

That HFH-subjects are able to generate increased ankle plantarflexor power when adjusting between self-selected and fast walking is an important observation indicating that maximal power generating capacity is not fully engaged during self-selected walking in HFH-subjects. In conjunction with preferential recruitment of excessive hip flexor power, submaximal plantarflexor power suggests that gait speed modulation in HFH-subjects results 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) does not increase when the hemiparetic subjects walk at higher speeds. Similarly, no additional power absorption is 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 the knee extensors.

Increased hip flexor power generation is similar to non-disabled controls and therefore appears to be preserved in both hemiparetic groups during swing. Lower functioning hemiparetic subjects engage 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 is preserved.

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

Role of Biomechanical Gait Mechanisms

Recent findings describe increased treadmill walking speed over the course of acute post-stroke recovery [19]. However, the biomechanical factors underlying increased walking speed were not probed systematically. Findings of the present study affirm that further research on the capacity of hemiparetic persons to modulate walking speed is relevant. Analysis of the biomechanical parameters, kinematics and kinetics, characterizing the nature of the underlying control strategies, should be further explored for their role in the functional classification of hemiparetic gait as well for their role as indicators of sensorimotor recovery.

Conclusion

Data from the present study support our hypothesis that both saturation of ankle plantarflexor power and inability to recruit additional hip flexor power limit increased walking speed in lower functioning hemiparetic subjects. Rehabilitation interventions to improve gait function should therefore target strategies to enhance power generation of both ankle plantarflexors and hip flexors and promote effective recruitment of both hip flexors and ankle plantarflexors during key phases of gait.

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References

1. Bohannon RW. Gait performance of hemiparetic stroke patients: selected variables. *Arch Phys Med Rehabil* 1987;68(11):777–781. [PubMed: 3675175]
2. Brandstatter ME, de Bruin H, Gowland C, Clark BM. Hemiplegic gait. Analysis of temporal variables. *Arch Phys Med Rehabil* 1983;64(12):583–587. [PubMed: 6661021]
3. Hsu AL, Tang PF, Jan MH. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. *Arch Phys Med Rehabil* 2003;84(8):1185–1193. [PubMed: 12917858]
4. Nadeau S, Arsenault AB, Gravel D, Bourbonnais D. Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. *Am J Phys Med Rehabil* 1999;78(2):123–130. [PubMed: 10088586]
5. Bohannon RW. Strength of lower limb related to gait velocity and cadence in stroke subjects. *Physiother Canada* 1986;38(4):204–206.
6. Bohannon RW, Andrews AW. Correlation of knee extensor muscle torque and spasticity with gait speed in patients with stroke. *Arch Phys Med Rehabil* 1990;71(5):330–333. [PubMed: 2327887]
7. Ada L, Dorsch S, Canning C. Strengthening interventions increase strength and improve activity after stroke: a systematic review. *Aust J Physiother* 2006;52(4):241–248. [PubMed: 17132118]
8. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. *Clin Biomech (Bristol, Avon)* 1999;14(2):125–35.
9. Olney SJ, Griffin MP, McBride ID. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. *Phys Ther* 1994;74(9):872–885. [PubMed: 8066114]
10. Murray MP, Mollinger LA, Gardner GM, Sepic SB. Kinematic and EMG patterns during slow, free, and fast walking. *J Orthop Res* 1984;2(3):272–280. [PubMed: 6491818]
11. den Otter AR, Geurts AC, Mulder T, Duysens J. Speed-related changes in muscle activity from normal to very slow walking speeds. *Gait Posture* 2004;19(3):270–278. [PubMed: 15125916]
12. Winter, DA. The biomechanics and motor control of human gait. University of Waterloo Press; Waterloo: p. 1-72.
13. Stansfield BW, Hillman SJ, Hazelwood ME. Sagittal joint kinematics, moments and powers are predominantly characterized by speed of progression, not age, in normal children. *Journal of Pediatric Orthopaedics* 2001;21:403–411. [PubMed: 11371829]
14. Van der Linden, Kerr AM, Hazlewood ME, Hillman SJ, Robb JE. Kinematic and Kinetic Gait Characteristics of normal children walking at a range of clinically relevant speeds. *Journal of Pediatric Orthopaedics* 2002;22:800–806. [PubMed: 12409911]
15. Requia LF, Nadeau S, Milot MH, Gravel D, Bourbonnais D, Gagnon D. Quantification of level of effort at the plantarflexors and hip extensors and flexor muscles in healthy subjects walking at different cadences. *Journal of Electromyography and Kinesiology* 2005;15:393–405. [PubMed: 15811610]
16. Milot MH, Nadeau S, Gravel D. Muscular utilization of the plantarflexors, hip flexors and extensors in persons with hemiparesis walking at self-selected and maximal speeds. *Journal of Electromyography and Kinesiology*. 2007 in print.
17. Bayat R, Barbeau H, Lamontagne A. Speed and temporal-distance adaptations during treadmill and overground walking following stroke. *Neurorehabil Neural Repair* 2005;19(2):115–24. [PubMed: 15883355]

18. Graf A, Judge JO, Ounpuu S, Thelen DG. The effect of walking speed on lower-extremity joint powers among elderly adults who exhibit low physical performance. *Arch Phys Med Rehabil* 2005;86:2177–2183. [PubMed: 16271567]
19. den Otter AR, Geurts ACH, Mulder T, Duysens J. Gait recovery is not associated with changes in the temporal patterning of muscle activity during treadmill walking in patients with post-stroke hemiparesis. *Clinical Neurophysiology* 2006;117:4–15. [PubMed: 16337186]

Figure 1.

A: Sagittal plane joint kinematics of hip, knee and ankle for the lower functioning stroke subjects walking at self-selected (solid) and fast speed (dashed) with superposition of the relevant speed related controls (Average $\pm$ Stdev, slow speed is indicated in light grey – fast speed in dark grey); Pelvis rotation (paretic side in dark – non-paretic side in grey) in lower functioning hemiparetic subjects walking at self-selected (solid) and fast (dashed) walking speed as compared to speed matched controls (Average $\pm$ Stdev, slow speed is indicated in light grey – fast speed in dark grey)

Figure 1B: Sagittal plane joint kinematics of hip, knee and ankle for the higher functioning stroke subjects walking at self-selected (solid) and fast speed (dashed) with superposition of

the relevant speed related controls (Average $\pm$ Stdev, slow speed is indicated in light grey – fast speed in dark gray); Pelvis rotation (paretic side in dark – non-paretic side in grey) in higher functioning hemiparetic subjects walking at self-selected (solid) and fast (dashed) walking speed as compared to speed matched controls (Average $\pm$ Stdev, slow speed is indicated in light grey – fast speed in dark gray)

Figure 2.

A: Joint power at hip and ankle for the lower functioning hemiparetic subjects walking at self-selected (solid) and fast speed (dashed) compared controls walking at relevant speeds (Average $\pm$ Stdev) with superposition of the relevant speed related controls (Average $\pm$ Stdev, slow speed is indicated in light grey – fast speed in dark gray);

Figure 2B: Joint power at hip and ankle for the higher functioning hemiparetic subjects walking at self-selected (solid) and fast speed (dashed) compared controls walking at relevant speeds (Average $\pm$ Stdev) with superposition of the relevant speed related controls (Average $\pm$ Stdev, slow speed is indicated in light grey – fast speed in dark gray);

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

	Gender	Side Stroke	Age (y)	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 (Sdev)			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 (Sdev)			57.8 (8.6)	9.6 (4.3)	82.5 (3.2)

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	Sig.
WALKING VELOCITY (m/s)			
Higher functioning hemiparetic subjects	0.88 ($\pm$ 0.04)	1.4 ($\pm$ 0.03)	**
Speed Matched Control subjects	0.92 ($\pm$ 0.04)	1.4 ($\pm$ 0.04)	*
Lower functioning hemiparetic subjects	0.52 ($\pm$ 0.04)	0.72 ($\pm$ 0.03)	**
Speed Matched Control subjects	0.35 ($\pm$ 0.03)	0.63 ($\pm$ 0.02)	*
STRIDE LENGTH (m)			
Higher functioning hemiparetic subjects	1.17 ($\pm$ 0.03)	1.42 ($\pm$ 0.04)	**
Speed Matched Control subjects	1.37 ($\pm$ 0.04)	1.59 ($\pm$ 0.04)	*
Lower functioning hemiparetic subjects	0.81 ($\pm$ 0.05)	0.93 ($\pm$ 0.06)	**
Speed Matched Control subjects	1.0 ($\pm$ 0.04)	1.25 ($\pm$ 0.03)	*
STRIDE DURATION (s)			
Higher functioning hemiparetic subjects	1.34 ($\pm$ 0.04)	1.04 ($\pm$ 0.07)	**
Speed Matched Control subjects	1.53 ($\pm$ 0.04)	1.17 ($\pm$ 0.02)	*
Lower functioning hemiparetic subjects	1.56 ($\pm$ 0.07)	1.28 ($\pm$ 0.05)	**
Speed Matched Control subjects	2.91 ($\pm$ 0.15)	1.97 ($\pm$ 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,

**
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	Sig.
STANCE DURATION (s)			
Higher functioning hemiparetic subjects – paretic limb	0.83 ($\pm$ 0.03)	0.61 ($\pm$ 0.04)	**
Higher functioning hemiparetic subjects - non-paretic limb	0.92 ($\pm$ 0.03)	0.69 ($\pm$ 0.05)	*
Speed Matched Control subjects	1.0 ($\pm$ 0.03)	0.72 ($\pm$ 0.01)	*
Lower functioning hemiparetic subjects - paretic limb	1.02 ($\pm$ 0.07)	0.82 ($\pm$ 0.05)	**
Lower functioning hemiparetic subjects - non-paretic limb	1.22 ($\pm$ 0.07)	0.94 ($\pm$ 0.04)	*
Speed Matched Control subjects	2.15 ($\pm$ 0.14)	1.3 ($\pm$ 0.04)	*
SINGLE STANCE DURATION (s)			
Higher functioning hemiparetic subjects – paretic limb	0.41 ($\pm$ 0.02)	0.36 ($\pm$ 0.02)	ns
Higher functioning hemiparetic subjects - non-paretic limb	0.51 ($\pm$ 0.02)	0.44 ($\pm$ 0.01)	*
Speed Matched Control subjects	0.53 ($\pm$ 0.01)	0.43 ($\pm$ 0.03)	**
Lower functioning hemiparetic subjects - paretic limb	0.37 ($\pm$ 0.03)	0.34 ($\pm$ 0.02)	ns
Lower functioning hemiparetic subjects - non-paretic limb	0.55 ($\pm$ 0.03)	0.48 ($\pm$ 0.03)	*
Speed Matched Control subjects	0.76 ($\pm$ 0.02)	0.67 ($\pm$ 0.03)	**
CADENCE (steps/min)			
Higher functioning hemiparetic subjects – paretic limb	203 ($\pm$ 52)	338 ($\pm$ 112)	*
Higher functioning hemiparetic subjects - non-paretic limb	171 ($\pm$ 38)	248 ($\pm$ 60)	*
Speed Matched Control subjects	169 ($\pm$ 28)	266 ($\pm$ 50)	**
Lower functioning hemiparetic subjects - paretic limb	139 ($\pm$ 32)	187 ($\pm$ 40)	*
Lower functioning hemiparetic subjects - non-paretic limb	129 ($\pm$ 30)	165 ($\pm$ 38)	*
Speed Matched Control subjects	71.31 ($\pm$ 13)	129 ($\pm$ 21)	**
STEP LENGTH (II)			
Higher functioning hemiparetic subjects – paretic limb	0.59 ($\pm$ 0.02)	0.71 ($\pm$ 0.03)	*
Higher functioning hemiparetic subjects - non-paretic limb	0.62 ($\pm$ 0.01)	0.74 ($\pm$ 0.02)	*
Speed Matched Control subjects	0.71 ($\pm$ 0.02)	0.82 ($\pm$ 0.02)	**
Lower functioning hemiparetic subjects - paretic limb	0.45 ($\pm$ 0.02)	0.51 ($\pm$ 0.02)	*
Lower functioning hemiparetic subjects - non-paretic limb	0.5 ($\pm$ 0.04)	0.54 ($\pm$ 0.04)	ns
Speed Matched Control subjects	0.52 ($\pm$ 0.01)	0.63 ($\pm$ 0.01)	**
SWING LENGTH (II)			
Higher functioning hemiparetic subjects – paretic limb	1.11 ($\pm$ 0.1)	1.35 ($\pm$ 0.06)	ns
Higher functioning hemiparetic subjects - non-paretic limb	1.01 ($\pm$ 0.03)	1.21 ($\pm$ 0.04)	*
Speed Matched Control subjects	0.9 ($\pm$ 0.09)	1.17 ($\pm$ 0.03)	**
Lower functioning hemiparetic subjects - paretic limb	0.82 ($\pm$ 0.19)	1.16 ($\pm$ 0.15)	ns
Lower functioning hemiparetic subjects - non-paretic limb	0.67 ($\pm$ 0.04)	0.77 ($\pm$ 0.04)	*
Speed Matched Control subjects	0.78 ($\pm$ 0.03)	0.99 ($\pm$ 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,
**
p<0.01

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	Sig.
H1 (Watts/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 (Watts/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 Matched 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 (Watts/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 (Watts/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 (Watts/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 (Watts/Kg)			

	Self-Selected Condition	Fast Condition	Sig.
Higher functioning hemiparetic subjects – paretic limb	1.22 ($\pm$ 0.3)	1.78 ($\pm$ 0.51)	[^]
Higher functioning hemiparetic subjects - non-paretic limb	2.22 ($\pm$ 0.36)	2.57 ($\pm$ 0.55)	ns
Speed Matched Control subjects	1.46 ($\pm$ 0.11)	2.41 ($\pm$ 0.13)	**
Lower functioning hemiparetic subjects - paretic limb	0.29 ($\pm$ 0.07)	0.44 ($\pm$ 0.08)	ns
Lower functioning hemiparetic subjects - non-paretic limb	1.42 ($\pm$ 0.24)	0.86 ($\pm$ 0.29)	ns
Speed Matched Control subjects	0.59 ($\pm$ 0.07)	0.89 ($\pm$ 0.07)	**
K3 (Watts/Kg)			
Higher functioning hemiparetic subjects – paretic limb	-0.29 ($\pm$ 0.09)	-0.73 ($\pm$ 0.22)	*
Higher functioning hemiparetic subjects - non-paretic limb	-0.36 ($\pm$ 0.06)	-0.9 ($\pm$ 0.24)	[^]
Speed MatchedControl subjects	-0.35 ($\pm$ 0.04)	-0.34 ($\pm$ 0.09)	ns
Lower functioning hemiparetic subjects - paretic limb	-0.19 ($\pm$ 0.03)	-0.3 ($\pm$ 0.06)	ns
Lower functioning hemiparetic subjects - non-paretic limb	-0.31 ($\pm$ 0.05)	-0.3 ($\pm$ 0.05)	ns
Speed Matched Control subjects	-0.17 ($\pm$ 0.02)	-0.3 ($\pm$ 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

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p<0.1,

*
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