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Original Article

Contribution of muscle activity at different gait phases for improving walking performance in chronic stroke patients with hemiparesis

Kazuki Fujita, PhD^{1)*}, Hideaki Hori, PhD¹⁾, Yasutaka Kobayashi, PhD²⁾

¹⁾ Department of Rehabilitation Physical Therapy, Faculty of Health Science, Fukui Health Science University: 55-13-1 Egami, Fukui-city, Fukui 910-3190, Japan

²⁾ Department of Rehabilitation Medicine, Fukui General Hospital, Japan

Abstract. [Purpose] The aim of this study was to clarify the optimal timing for increasing muscle activity in the paralyzed lower limb of stroke survivors by evaluating the relationship between gait muscle activity patterns and gait parameters. [Participants and Methods] Electromyography of the tibialis anterior, soleus, rectus femoris, and biceps femoris on the paralyzed side and spatiotemporal gait parameters were evaluated in 40 chronic post-stroke patients as they walked at a comfortable speed. The normalized average amplitude and asymmetry indexes of each gait phase were calculated. The correlations between gait velocity or asymmetry indexes and the activity amplitudes of various muscles during each gait phase were analyzed. Multiple regression analysis was performed with gait velocity or asymmetry indexes as the response variable and the muscle activity amplitudes in the various gait phases as explanatory variables. [Results] The major determinants of gait velocity were the tibialis anterior activity (β =-0.35) and biceps femoris activity (β =0.45) during the swing phase. In addition, the biceps femoris activity during the swing phase was the major determinant of the asymmetry index for the swing phase duration (β =-0.41). [Conclusion] For patients with hemiparesis, increasing the biceps femoris activity during the swing phase is considered optimal, which may lead to improvement in walking performance.

Key words: Stroke, Gait, Electromyography

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INTRODUCTION

Common features of walking after stroke include decreased gait velocity and an asymmetrical gait pattern^{1, 2)}. Gait velocity is an indicator of the patient's functional walking ability at home and in the community³⁾, and it is often used when defining the minimum clinically important difference that represents functional improvement^{4, 5)}. Thus, physical therapy for hemiparetic stroke patients usually includes an aim to increase walking speed. Although various factors influence the gait velocity of stroke patients, many studies have reported a relationship with the muscle strength of the paralyzed lower limb. For example, studies of isometric muscle force on the paralyzed side found significant moderate and strong correlations between gait velocity and the plantar flexion and dorsiflexion torques of the ankle, as well as the flexion and the extension torques of the hip and knee^{6, 7)}. Similarly, studies of isokinetic muscle force on the paralyzed side found strong correlations for gait velocity with the flexion and extension torques of the knee and the flexion torque of the hip^{8,9)}. In addition, the muscle strength of paralyzed lower limb and the spatiotemporal symmetry of gait have been shown to be significantly correlated^{6, 10}). Thus, the muscular strength of the paralyzed lower limb has a considerable influence on walking ability. Increasing the muscle activity of the paralyzed lower limb during gait is therefore important.

However, the temporal order of muscle activity during walking can often be disrupted following cerebral stroke¹¹). For

*Corresponding author. Kazuki Fujita (E-mail: k.fujita-fchs@kvj.biglobe.ne.jp)

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Table 1. Characteristics of the participants (N=40)

Age (yrs)	58.4 ± 10.4
Gender (female/male)	10/30
Type of stroke (CI/ICH/SAH)	13/25/2
Months since onset	55.1 ± 47.6
Paretic side (left/right)	16/24
Fugl-Meyer assessment the LE	20 ± 5
Modified Ashworth scale	2 (1-3)
Assistive device (none/T-handled cane/AFO)	8/27/30

CI: cerebral infarction; ICH: intracerebral hemorrhage; SAH: subarachnoid hemorrhage; LE: lower extremity; AFO: ankle foot orthosis.

example, in case of hemiparetic patients, premature plantarflexor muscle activity on the paretic side occurs to supplement lower extremity extension force¹²). Over time, stroke patients develop various compensatory strategies to try to achieve an efficient method of walking¹³). It is possible that the gait muscle activation patterns of neurologically healthy individuals may not result in optimal performance in individuals with chronic stroke.

The aim of this study was to clarify the optimal timing for increasing muscle activity in the paralyzed lower limb of stroke survivors by evaluating the relationship between gait muscle activity patterns and gait parameters.

PARTICIPANTS AND METHODS

The participants were 40 stroke survivors with hemiparesis. Their mean (\pm standard deviation) age was 58.4 \pm 10.4 years, and the mean period since the onset of stroke was 55.1 \pm 47.6 months (Table 1). The inclusion criteria were as follows: a unilateral lesion in the cerebrum; at least 6 months since stroke onset; ability to walk at the monitoring level (either unassisted or with a T-shaped cane, but without leg braces); and a walking speed of 0.1 to 1 m/s without leg braces. The exclusion criteria were as follows: passive ankle dorsiflexion range of motion \leq 0; higher brain dysfunction that presented problems for intervention and/or evaluation; and cardiovascular disease that restricted exercise.

All evaluations were performed at Fukui General Hospital, Japan. The study was approved by the hospital's Ethical Review Committee (approval no.: Nittazuka Ethics 29-105), and all participants provided written informed consent.

Electromyography and spatiotemporal gait parameters were evaluated with the participant walking at a comfortable walking speed and mode along a straight walkway 16 m long with an extra 3 m space at each end. A video camera (HD Pro WebCamera; Logicool, Inc., Tokyo, Japan) with a sampling frequency of 30 Hz was set up at 5 m lateral to the midpoint of the walkway, and a 1 m line was drawn at the midpoint of the walkway. The time taken to walk 10 m was measured using a stopwatch. During the evaluation, the participant was allowed to use a walking cane but not a leg brace. Shoes were worn while walking.

A TeleMyo DTS electromyography system (Noraxon Inc., Scottsdale, AZ, USA) was used for the electromyographic recording. The sampling frequency was 1,500 Hz, and a bandpass filter of 10–500 Hz was applied. Electromyography was recorded in four muscles in which previous studies^{6–10)} have reported correlation between muscle strength and gait performance in stroke survivors: the tibialis anterior, soleus, rectus femoris, and biceps femoris on the paralyzed side of the body. Muscle action potential was induced using bipolar leads. Skin impedance was reduced to no more than 10 k Ω using alcohol-soaked cotton swabs and an abrasive (Skin Pure; Nihon Kohden Co., Ltd., Tokyo, Japan), and Ag–AgCl electrodes (EM-272; Noraxon Inc.) were placed 2 cm apart at positions recommended by the Surface Electromyography for the Non-Invasive Assessment of Muscle project¹⁴). Four foot switches were placed on the sole of each foot. All the devices were synchronized using synchronization and optical signals.

The electromyographic waveforms were analyzed using an MR3 processing system (Noraxon Inc.). Full-wave rectification was performed for all the raw waveforms. The analysis interval was set as three continuous gait cycles at around halfway along the walkway. The durations of the three gait cycles were normalized so that each gait cycle was considered to be 100%. The arithmetic mean amplitude of muscle activity for the three gait cycles was calculated, followed by normalization using the average amplitude of the entire gait cycle.

The gait phases (loading response, single support, pre-swing, and swing) were distinguished on the basis of the foot switch data, and the levels of muscle activity were calculated from the mean amplitudes of the respective phases, following the method of Turns et al¹⁵⁾. The temporal gait parameters were walking speed (calculated from the walking time) and cadence and swing phase duration, which were calculated from the foot switch data. The asymmetry index for the swing phase duration (AI_{SPD}) was calculated from the paralyzed (P) and non-paralyzed (NP) side swing phase durations, SPD_P and SPD_{NP}, respectively, as $AI_{SPD}=SPD_P/(SPD_P + SPD_{NP})^{16}$. Spatial gait parameters were measured using still images extracted from the video data for the moment when the participant passed through the halfway point of the walkway¹⁷, using ImageJ image processing software (National Institutes of Health, MD, USA). Step length was measured as the linear distance between the point of heel contact

Table 2.	Gait parameters	(N=40)
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Gait velocity (m/s)	0.52 ± 0.22
Cadence (steps/min)	80.8 ± 19.5
Stride length (cm)	75.7 ± 21.3
Step duration, Ps (s)	0.60 ± 0.15
Step duration, NPs (s)	0.39 ± 0.08
AI for Sw duration	0.60 ± 0.05
Stride length (cm)	75.7 ± 21.3
Step length, Ps (cm)	40.4 ± 10.0
Step length, NPs (cm)	35.6 ± 13.7
AI for step length	0.54 ± 0.09

Table 3. Electromyogram results (N=40)

	LR	SS	PSw	Sw
TA	1.01 ± 0.32	0.67 ± 0.22	1.15 ± 0.31	1.16 ± 0.34
Sol	1.49 ± 0.34	1.35 ± 0.28	0.67 ± 0.21	0.49 ± 0.22
RF	1.41 ± 0.36	0.87 ± 0.27	0.93 ± 0.35	0.79 ± 0.27
BF	1.64 ± 0.35	1.23 ± 0.31	0.47 ± 0.30	0.66 ± 0.29

Values are normalized mean amplitudes during each gait phase (mean \pm standard deviation).

TA: tibialis anterior; Sol: soleus; RF: rectus femoris; BF: biceps femoris; LR: loading response phase; SS: single support phase; PSw: pre-swing phase; Sw: swing phase.

Values are presented as the mean ± standard deviation. Ps: paretic side; NPs: nonparetic side; Sw: swing; AI: asymmetry index.

Table 4.	Correlations (Pe	earson's r) between	gait velocity	or the asymmetry	indices and
	the mean muscle	e activity amplitude	es of the gait	phases	

Muscles	Gait phase	Gait velocity (r)	AI, Sw duration (r)	AI, step length (r)
TA	LR	0.28	-0.18	-0.13
	SS	-0.05	-0.01	0.17
	PSw	0.23	-0.16	-0.03
	Sw	-0.44^{**}	0.33*	0.04
Sol	LR	0.24	-0.13	0.06
	SS	-0.17	0.16	-0.17
	PSw	-0.15	-0.05	-0.13
	Sw	-0.01	0.05	0.25
RF	LR	0.20	-0.05	-0.15
	SS	-0.06	-0.17	-0.04
	PSw	0.01	-0.08	0.03
	Sw	-0.22	0.34*	0.19
BF	LR	-0.20	0.24	0.04
	SS	-0.33^{*}	0.32*	0.10
	PSw	0.11	-0.23	-0.04
	Sw	0.47**	-0.39*	-0.11

*p<0.05, **p<0.01.

TA: tibialis anterior; Sol: soleus; RF: rectus femoris; BF: biceps femoris; LR: loading response phase; SS: single support phase; PSw: pre-swing phase; Sw: swing phase; AI: asymmetry index.

of one foot and the successive point of heel contact of the opposite foot. The asymmetry index for the step length, AI_{SL} , was calculated from the paralyzed and non-paralyzed side step lengths (SL_P and SL_{NP} , respectively) as $AI_{SL}=SL_P/(SL_P+SL_{NP})^{18}$. Mean values for three walking trials were calculated for all the electromyographic data and gait parameters. Thus, for the

electromyographic data and temporal parameters, the means calculated were for three gait cycles (three gait cycles × three trials), whereas for spatial parameters, the means calculated were for three gait cycles (one gait cycle × three trials).

The relationships between gait velocity or the asymmetry indices and the mean amplitudes of the respective gait phases were evaluated by Pearson correlation analysis. Multiple regression analysis was performed with the gait velocity or asymmetry indices as the response variable and the amplitudes of each of the four muscles as explanatory variables. This analysis was performed separately for each gait phase. The software used for these analyses was SPSS version 25 (IBM Corp., Armonk, NY, USA), with a significance level of 5%.

RESULTS

The gait parameters are presented in Table 2, and the electromyogram data are shown in Table 3. There were significant correlations between gait velocity and the mean activity amplitudes of the tibialis anterior (r=-0.44) and biceps femoris (r=0.47) during the swing phase and the biceps femoris (r=-0.33) during the single support phase (Table 4). There were also significant correlations between the AI_{SPD} and the mean amplitudes for the tibialis anterior (r=0.33), rectus femoris (r=-0.34), and biceps femoris (r=-0.39) during the swing phase and the biceps femoris (r=-0.32) during the single support phase (Table 4).

Table 5. Multiple regression analysis for gait velocity and asymmetry indices (N=40)

Response variable	Explanatory variables	β	\mathbb{R}^2	F
Gait velocity	TA-Sw	-0.35	0.38	4.80^{*}
	BF-Sw	0.45	0.38	10.14**
AI for Sw duration	BF-Sw	-0.41	0.31	7.81**

*p<0.05, **p<0.01.

TA: tibialis anterior; BF: biceps femoris; Sw: swing; AI: asymmetry index.

4). However, no significant correlations with the asymmetry index for the step length were observed.

The multiple regression analysis revealed the major determinants of gait velocity to be the muscle activity of the tibialis anterior (β =-0.35, p=0.035) and biceps femoris (β =0.45, p=0.003) activity during the swing phase. In addition, the muscle activity of the biceps femoris during the swing phase was the major determinant of the AI_{SPD} (β =-0.41, p=0.008). No factors that significantly affected the asymmetry index for the step length were found (Table 5).

DISCUSSION

In this study, muscle activity patterns on the paralyzed side were evaluated from the electromyogram data by normalizing the average amplitude of each gait phase according to the average amplitude of the entire gait cycle, thereby showing the timing of increases in muscle activity as the relative magnitude of muscle activity of each gait phase. The results showed that the magnitude of activity of the biceps femoris during the swing phase affected the gait velocity. In individuals with hemiparesis, a common abnormality is a stiff knee gait, in which the knee flexion angle during swing phase decreases because of hyperactivity of the rectus femoris¹⁹. Conversely, in these individuals, the magnitude of biceps femoris activity during gait is related to the increase in the flexion angle of the knee²⁰. In addition, the gait velocity of stroke survivors has been shown to be related to the magnitude of the angle of knee flexion and its torque during gait^{21, 22}. It is possible that the biceps femoris activity increase the knee flexion angle in compensation for the decrease during the swing phase and that this affected the relationship with gait velocity.

During normal walking, the peak activity of the tibialis anterior occurs during the loading response phase²³, but for the participants of this study, the tibialis anterior activity was highest in the swing phase and was negatively correlated with gait velocity. This tendency suggested that people with a fast walking speed have high activity of the biceps femoris but low activity of the tibialis anterior during the swing phase, whereas those with a slow walking speed have low activity of the biceps femoris but high activity of the tibialis anterior during the swing phase. Those with a slow walking speed may have increased activity of the tibialis anterior to compensate for the decreases in foot clearance resulting from decreased knee flexion angle because of lower activity of the biceps femoris. In addition, it has been shown in individuals with hemiparesis that co-activation of the flexor and extensor muscles of the ankle joint²⁵. Thus, hyperactivity of the tibialis anterior increased co-activation with the soleus muscle and may have contributed to a decrease in gait velocity. Although the strength of ankle dorsiflexion due to the resting position of hemiparetic individuals is positively correlated with gait velocity⁶, high activity of the tibialis anterior during the swing better.

The analysis of the relationship between the swing phase duration asymmetry index and gait muscle activity in this study showed that the swing duration was symmetrical when there was high activity of the biceps femoris muscle during the swing phase. The mean swing duration in this study was clearly prolonged on the paralyzed side (0.60 s compared with 0.39 s on the non-paralyzed side). The activity of the biceps femoris during the paralyzed side swing phase is unlikely to affect the non-paralyzed side swing duration, so if an increase in biceps femoris activity on the paralyzed side results in a reduction in the prolonged swing phase duration, this would result in a more symmetrical gait. This may be explained by increases in knee flexion angle and foot clearance because of the increased biceps femoris activity, as described above.

Although the muscular strength of the paralyzed lower limb was found to be correlated with gait speed or asymmetry in many previous studies^{6–10}, in this study, gait muscle activity was associated only with the biceps femoris and tibialis anterior. Buurke et al.²⁶ tracked changes in functional evaluations and gait muscle activity patterns after the onset of stroke and reported that the gait velocity showed a statistically significant improvement but that there were no significant changes in the muscle activity patterns. Furthermore, stronger correlations have been reported between gait velocity and the muscular strength of paralyzed lower limb^{6–9} or the peak joint torque during walking²² than the correlation with muscle activity patterns in the present study. Conversely, Clark et al.²⁷ reported that the number of modules showing coordination between lower limb muscles decreased in the paralyzed side of post-stroke participants during walking and that the number of modules was correlated with gait velocity and step length asymmetry. This suggested that the activity pattern of each muscle has little influence on gait ability, whereas the influence of the muscular strength and cooperativeness between the lower limb muscles is much greater.

In this study, relative muscle activity during the loading response, single support, pre-swing, and swing phases was

calculated as muscle activity patterns, and the relationship between these and the gait velocity and asymmetry indices were investigated. The activity of the biceps femoris during the swing phase showed the strongest relationship with the gait velocity and the asymmetry index for swing duration. This activity may be compensatory, increasing the reduced knee flexion angle. For patients with hemiparesis, it is considered optimal that the biceps femoris activity increases during swing phase, which may lead to improvement in walking performance.

Conflict of interest

None.

REFERENCES

- Titianova EB, Tarkka IM: Asymmetry in walking performance and postural sway in patients with chronic unilateral cerebral infarction. J Rehabil Res Dev, 1995, 32: 236–244. [Medline]
- Goldie PA, Matyas TA, Evans OM: Gait after stroke: initial deficit and changes in temporal patterns for each gait phase. Arch Phys Med Rehabil, 2001, 82: 1057–1065. [Medline] [CrossRef]
- 3) Perry J, Garrett M, Gronley JK, et al.: Classification of walking handicap in the stroke population. Stroke, 1995, 26: 982–989. [Medline] [CrossRef]
- 4) Tilson JK, Sullivan KJ, Cen SY, et al. Locomotor Experience Applied Post Stroke (LEAPS) Investigative Team: Meaningful gait speed improvement during the first 60 days poststroke: minimal clinically important difference. Phys Ther, 2010, 90: 196–208. [Medline] [CrossRef]
- Perera S, Mody SH, Woodman RC, et al.: Meaningful change and responsiveness in common physical performance measures in older adults. J Am Geriatr Soc, 2006, 54: 743–749. [Medline] [CrossRef]
- 6) Lin PY, Yang YR, Cheng SJ, et al.: The relation between ankle impairments and gait velocity and symmetry in people with stroke. Arch Phys Med Rehabil, 2006, 87: 562–568. [Medline] [CrossRef]
- 7) Bohannon RW: Muscle strength and muscle training after stroke. J Rehabil Med, 2007, 39: 14-20. [Medline] [CrossRef]
- 8) Nadeau S, Arsenault AB, Gravel D, et al.: Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. Am J Phys Med Rehabil, 1999, 78: 123–130. [Medline] [CrossRef]
- 9) Flansbjer UB, Downham D, Lexell J: Knee muscle strength, gait performance, and perceived participation after stroke. Arch Phys Med Rehabil, 2006, 87: 974–980. [Medline] [CrossRef]
- 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: 1185–1193. [Medline] [CrossRef]
- Den Otter AR, Geurts AC, Mulder T, et al.: Abnormalities in the temporal patterning of lower extremity muscle activity in hemiparetic gait. Gait Posture, 2007, 25: 342–352. [Medline] [CrossRef]
- Fujita K, Miaki H, Fujimoto A, et al.: Factors affecting premature plantarflexor muscle activity during hemiparetic gait. J Electromyogr Kinesiol, 2018, 39: 99–103. [Medline] [CrossRef]
- Kim CM, Eng JJ: Magnitude and pattern of 3D kinematic and kinetic gait profiles in persons with stroke: relationship to walking speed. Gait Posture, 2004, 20: 140–146. [Medline] [CrossRef]
- 14) The SENIAM project: SEMG sensors. http://www.seniam.org/lowerleg_location.htm (Accessed Dec. 1, 2014)
- 15) Turns LJ, Neptune RR, Kautz SA: Relationships between muscle activity and anteroposterior ground reaction forces in hemiparetic walking. Arch Phys Med Rehabil, 2007, 88: 1127–1135. [Medline] [CrossRef]
- 16) Lauziere S, Betschart M, Aissaoui R, et al.: Understanding spatial and temporal gait asymmetries in individuals post stroke. Int J Phys Med Rehabil, 2014, 2: 201.
- 17) Wall JC, Devlin J, Khirchof R, et al.: Measurement of step widths and step lengths: a comparison of measurements made directly from a grid with those made from a video recording. J Orthop Sports Phys Ther, 2000, 30: 410–417. [Medline] [CrossRef]
- Allen JL, Kautz SA, Neptune RR: Step length asymmetry is representative of compensatory mechanisms used in post-stroke hemiparetic walking. Gait Posture, 2011, 33: 538–543. [Medline] [CrossRef]
- Stoquart GG, Detrembleur C, Palumbo S, et al.: Effect of botulinum toxin injection in the rectus femoris on stiff-knee gait in people with stroke: a prospective observational study. Arch Phys Med Rehabil, 2008, 89: 56–61. [Medline] [CrossRef]
- 20) Wang W, Li K, Yue S, et al.: Associations between lower-limb muscle activation and knee flexion in post-stroke individuals: a study on the stance-to-swing phases of gait. PLoS One, 2017, 12: e0183865. [Medline] [CrossRef]
- 21) 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: 872–885. [Medline] [CrossRef]
- 22) Kim CM, Eng JJ: The relationship of lower-extremity muscle torque to locomotor performance in people with stroke. Phys Ther, 2003, 83: 49–57. [Medline]
- 23) Schmitz A, Silder A, Heiderscheit B, et al.: Differences in lower-extremity muscular activation during walking between healthy older and young adults. J Electromyogr Kinesiol, 2009, 19: 1085–1091. [Medline] [CrossRef]
- 24) Aymard C, Giboin LS, Lackmy-Vallée A, et al.: Spinal plasticity in stroke patients after botulinum neurotoxin A injection in ankle plantar flexors. Physiol Rep, 2013, 1: e00173. [Medline] [CrossRef]
- 25) Chow JW, Yablon SA, Stokic DS: Coactivation of ankle muscles during stance phase of gait in patients with lower limb hypertonia after acquired brain injury. Clin Neurophysiol, 2012, 123: 1599–1605. [Medline] [CrossRef]
- 26) Buurke JH, Nene AV, Kwakkel G, et al.: Recovery of gait after stroke: what changes? Neurorehabil Neural Repair, 2008, 22: 676–683. [Medline] [CrossRef]
- 27) Clark DJ, Ting LH, Zajac FE, et al.: Merging of healthy motor modules predicts reduced locomotor performance and muscle coordination complexity poststroke. J Neurophysiol, 2010, 103: 844–857. [Medline] [CrossRef]