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Original Article

Effects of increasing non-paretic step length on paretic leg movement during hemiparetic gait: a pilot study

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Abstract. [Purpose] Gait training that increases non-paretic step length in stroke patients increases the propulsive force of the paretic leg. However, it limits knee flexion during the swing phase of gait, and this may cause gait disturbances such as worsening of gait pattern and increased risk of falling. Therefore, this study aimed to investigate the effects of increasing non-paretic step length on the joint movement and muscle activity of a paretic lower limb during hemiparetic gait. [Participants and Methods] A total of 15 hemiparetic patients with chronic stroke were enrolled in this study. Spatiotemporal parameters, along with kinematic and electromyography data of their paretic lower limbs, were measured during a 10-m distance overground walking. Two walking conditions were assessed: normal (comfortable gait) and non-paretic-long (gait with increased non-paretic step length) conditions. [Results] Under the non-paretic-long condition, the trailing limb angle was larger than under the normal condition. However, no significant difference was observed in the knee flexion angle during the swing phase. [Conclusion] Increasing non-paretic step length during gait is unlikely to limit knee flexion during the swing phase and can safely improve the propulsive force of a paretic leg.

Key words: Stroke, Gait, Electromyography

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INTRODUCTION

The walking ability of stroke patients is often reduced, and their activities of daily living are limited by reduced walking speed and distance. The propulsive force of the paretic lower limb strongly influences the walking speed and distance[1, 2)], and improves the propulsive force of the paretic lower limb is important to enhance the walking ability. Factors used to determine the propulsive force include ankle plantarflexion moment and trailing limb angle (TLA; the angle between the vertical and straight lines from the greater trochanter to the fifth metatarsal head)[3)] to improve propulsive force; thus, the hip extension angle should be increased at the terminal stance. Clark et al.[4)] reported that increasing the nonparetic (NP) step length during walking increased the propulsive force of the paretic lower limb by lengthening the horizontal distance between the backward-positioned paretic foot and the pelvis and concluded that gait training by increasing the NP step length is useful. However, changes in paretic leg movements during the swing phase due to an increased NP step length have not been investigated.

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In the swing phase, foot clearance is maintained by knee flexion. The limitation of knee flexion should be avoided because it increases the risk of falling and energy costs[5]. Lewek et al.[6] observed overactivity of the knee extensor muscles in stroke patients by rapidly extending the paretic hip using the Biodex System 3, suggesting that rapid hip extension during gait may cause overactivity of the knee extensor muscles and limit knee flexion during the swing phase. Further, they considered that stroke patients may limit the hip extension angle to minimize overactivity of the knee extensors during walking[7]. Therefore, gait training that increases NP step length increases the propulsive force of the paretic lower limb; however, it limits knee flexion during the swing phase, which may cause new gait disturbances, such as increasing the risk of falling and compromising gait stability, thereby greatly impairing the daily living activities[8]. Clarifying the relationship between the change in NP step length and the kinematics of the paretic swing phase is expected to aid in the planning of rehabilitation programs that improve propulsive force while taking gait disorders into account. Therefore, this study aimed to investigate the effects of increasing the NP step length on the joint movement and muscle activity of the paretic lower limb in a hemiparetic gait. The hypothesis was that an increased NP step length would force paretic hip extension, induce quadriceps overactivity, and limit knee flexion during the swing phase.

PARTICIPANTS AND METHODS

Of the 41 hemiparetic patients with chronic stroke who used our short-term intensive rehabilitation program during October 2019–August 2020, 15 patients who met the following inclusion criteria were included (61.7 ± 9.4 years; 67.5 ± 14.8 kg; 166.3 ± 10.7 cm). The inclusion criteria were (1) hemiplegia due to stroke, (2) at least 6 months post-stroke, (3) sufficient passive knee joint range of motion (0–100°), and (4) the ability to walk unaided or with the use of a T-cane at a monitored level or higher. Exclusion criteria were (1) history of lower limb joint surgery, (2) respiratory or cardiovascular symptoms while walking, and (3) dementia with a Mini-Mental State Examination score of ≤23. This study was approved by the Ethical Review Committee of Nittazuka Medical Welfare Center (approval no. Nittazuka Ethics 2019-42), and all participants provided written informed consent.

Physical function assessment was evaluated using Fugl–Meyer assessment of lower extremity (FMA); Motricity Index (MI)[9]; modified Ashworth Scale (MAS) for hip flexors, knee extensors, and ankle plantar flexors; and range of motion for hip extension and ankle dorsiflexion. Gait assessment was based on spatiotemporal parameters, with the kinematic and Electromyography (EMG) data of the paretic lower limb, which was measured during a 10-m-distance overground walking. The evaluation was performed by walking alone, and the use of a T-cane or a short leg brace (Orthop AFO) was allowed only a risk of falling exists. According to previous studies[4], the following two walking conditions were used: normal (comfortable gait) and NP-long (gait with increased NP step length). In the NP-long condition, participants were explained that “Please increase your nonparetic step length as long as you did not lose balance while walking”. The order of walking conditions was normal, NP-long, and a 10-min walking practice period was provided before performing the NP-long. Participants were allowed to walk at their own speed during NP-long because the defined walking speed might result in an unnatural gait.

An Electromyography system TelemyoDTS (Noraxon Inc., Scottsdale, AZ, USA) and a 3D motion analysis system Myomotion (Noraxon Inc.) were used for measurements. Electromyography sampling frequency was 1,500 Hz, and the band-pass filter was 10–500 Hz. In this study, the rectus femoris, vastus lateralis, biceps femoris, tibialis anterior, medial head of gastrocnemius, and soleus on the paretic side were targeted, referring to the Surface ElectroMyoGraphy for the Non-invasive Assessment of Muscles[10]. After the skin impedance was lowered to <10 kΩ by skin processing, silver chloride surface electrodes (Noraxon Inc.) were applied with a 2-cm distance between electrodes. Myomotion is a measuring instrument that calculates angles and angular velocities from motion sensors with built-in combinations of gyro-, acceleration, and magnetic sensors. Myomotion sampling frequency was 100 Hz. Motion sensors were attached to the sacrum, bilateral thighs, shanks, and foot. Zero calibration to determine joint angle information was performed in standing position. Moreover, reflective markers were placed on the paretic greater trochanter and the fifth metatarsal head. A video camera (sampling frequency 30 Hz) was located 5 m from the midpoint of the walking path as a peripheral device. All devices were synchronized using synchronization and optical signals. All participants had three sessions of normal, followed by three sessions of NP-long.

MR3 (Noraxon Inc.) was used for analysis. Data used in the analysis were for three gait cycles at the halfway point of a 10-m walk. At first, the raw EMG waveform was full-wave rectified. Next, the initial contact and toe-off of both lower limbs were determined based on the acceleration information from MR3 and EMG of one gait cycle was considered to be 100%, and three gait cycles were additionally averaged. The gait phase was divided into the loading response, single support, pre-swing, and swing phase. The second half of the single support phase was defined as the latter 50% of the single support phase[11], and the early swing phase was defined as the period from the toe-off to the maximum knee flexion[12]. EMG data analysis was normalized by calculating the mean amplitude of each gait phase and dividing by the mean amplitude of the entire gait cycle[11]. Kinematic data were extracted for the peak hip extension angle and peak extension angular velocity during the second half of the single-support phase; the peak hip and knee flexion angular velocity and peak ankle plantar flexion angular velocity during the pre-swing; the knee flexion angle and knee flexion angular velocity at the toe-off; and the peak knee flexion angle during the swing phase.

Gait speed was calculated using a stopwatch, and gait cycle rate and cadence were calculated using MR3. The step length and stride were calculated from a 1-m calibration scale set at the center of the gait path using ImageJ image processing

software (National Institutes of Health, Bethesda, MD, USA). In addition, the TLA at toe-off was calculated[13]. Kinematic and EMG data were averaged over nine gait cycles, while step length, stride, and TLA were averaged over the three gait cycles. The statistical software Bell Curve for Excel was used to compare the normal and NP-long gait parameters using the Wilcoxon signed-rank test. The significance level was $p<0.05$. The effect size (ES) of each dataset was calculated, and r=0.1 was defined as a small ES, 0.3 as a medium ES, and 0.5 as a large ES[14].

RESULTS

All the participants were males. FMA average was 22.3 ± 3.7 points, indicating moderate motor paralysis. All participants had spasticity (MAS ≥1) in the hip flexors, knee extensors, and ankle plantar flexors. Of the 15 participants, 8 used a T-cane and 4 used an ankle -foot orthosis (Table 1). In the spatiotemporal parameters, NP-long showed a significant increase in the gait speed ($p<0.05$, ES=0.81), stride ($p<0.05$, ES=0.79), and nonparetic step length ($p<0.05$, ES=0.88) compared with normal (Table 2). In the kinematic data, NP-long showed a significant increase in the peak hip extension angle ($p<0.05$, ES=0.88) and peak extension angular velocity ($p<0.05$, ES=0.76) in the second half of the single-support phase compared with normal. No significant differences were found in the peak knee flexion angle during the swing phase. Moreover, a significant increase in TLA ($p<0.05$, ES=0.85) was observed (Table 3). In the EMG data, NP-long showed a significant increase in the rectus femoris ($p<0.05$, ES=0.57) and soleus ($p<0.05$, ES=0.63) activities during the pre-swing phase compared with normal (Table 4).

Table 1. Participant characteristics

Age (years)		61.7 ± 9.4
Gender (female/male)		0/15
Months of onset		73.9 ± 57.4
Type of stroke (CI/ICH)		5/10
Paretic side (right/left)		11/4
Fugl-Meyer assessment LE		22.3 ± 3.7
Motricity Index	Hip	74 (0)
	Knee	74 (26)
	Ankle	56 (25)
MAS	Hip flexors	1 (0.25)
	Knee extensors	1.5 (1)
	Ankle plantar flexors	1.5 (0.5)
ROM	Hip extension (degree)	10.9 ± 5.4
	Ankle dorsiflexion (degree)	5.5 ± 4.9
Assistive device (none/T-cane/AFO)		6/8/4

Mean ± SD; median (quartile deviation).
CI: cerebral infarction; ICH: intracerebral hemorrhag; LE: lower extremity; MAS: modified Ashworth Scale; AFO: ankle-foot orthosis.

Table 2. Spatiotemporal parameters

		Normal	NP-long		ES
LR	%	13.2 ± 4.2	11.0 ± 3.8	*	0.56
SS	%	31.2 ± 6.0	33.1 ± 5.7	*	0.56
PSw	%	15.0 ± 5.5	12.6 ± 6.1	**	0.87
Sw	%	40.6 ± 4.2	43.2 ± 3.9	**	0.84
Cadence	steps/min	87.6 ± 17.8	85.3 ± 18.1		0.43
Gait cycle time	s	1.44 ± 0.37	1.42 ± 0.52		0.22
Gait speed	m/s	0.61 ± 0.35	0.73 ± 0.42	**	0.81
Stride	cm	93.0 ± 26.1	109.4 ± 29.6	**	0.79
Step length paretic	cm	46.7 ± 13.3	48.1 ± 16.0		0.29
nonparetic	cm	46.3 ± 14.0	61.3 ± 16.6	**	0.88

Mean ± SD; *p<0.05; **p<0.01; ES: effect size; Normal: comfortable gait; NP-long: gait with increased non-paretic step length; LR: loading response phase; SS: single support phase; PSw: pre-swing phase; Sw: Swing phase.

DISCUSSION

In this study, we compared and investigated the effects of the increased NP step length on paretic leg movements in hemiparetic patients with chronic stroke, using spatiotemporal parameters and kinematic and EMG data. The results showed that the peak hip extension angle and peak hip extension angular velocity during the second half of the single support phase and TLA were increased, and the rectus femoris activity increased during the pre-swing phase; however, the peak knee flexion angle during the swing phase was unchanged. Contrary to our hypothesis, increasing the NP step length did not limit knee flexion during the swing phase and increased TLA.

During the second half of the single-support phase, the peak hip extension angle and the peak extension angular velocity increased, as did the TLA at toe-off. Similar to a previous study[4], this result may be due to increased horizontal distance between the pelvis and paretic foot by increasing the NP step length. Because participants had spasticity in hip flexors and knee

Table 3. Kinematic data

				Normal	NP-long		ES
Late SS	Hip	Peak extension angle	°	7.5 ± 5.2	10.2 ± 5.3	**	0.88
		Peak extension anguler velocity	deg/s	49.8 ± 27.1	62.7 ± 36.3	**	0.76
PSw	Hip	Peak flexion angular velocity	deg/s	101.6 ± 42.7	85.6 ± 38.6	*	0.54
	Knee	Peak flexion angular velocity	deg/s	215.5 ± 82.6	196.1 ± 89.9		0.37
	Ankle	Peak plantar flexion angular velocity	deg/s	145.0 ± 103.3	181.3 ± 115.0	**	0.78
Toe-off	Knee	Flexion angle	°	26.9 ± 11.0	24.0 ± 9.4		0.35
		Flexion angular velocity	deg/s	134.8 ± 112.2	153.8 ± 100.5		0.34
Sw	Knee	Peak flexion angle	°	35.3 ± 16.8	36.9 ± 16.0		0.29
	Trailing Limb Angle (TLA)		°	13.7 ± 6.7	18.7 ± 8.7	**	0.85

Mean ± SD; *p<0.05; **p<0.01; ES: effect size; Normal: comfortable gait; NP-long: gait with increased non-paretic step length; Late SS: second half of single support phase; PSw: pre-swing phase; Sw: swing phase.

Table 4. EMG data (%)

		Normal	NP-long		ES
RF	Late SS	107.1 ± 38.1	113.4 ± 37.0		0.16
	PSw	81.6 ± 32.4	99.1 ± 45.6	*	0.57
	Esw	80.1 ± 44.0	76.9 ± 41.8		0.10
VL	Late SS	114.3 ± 58.3	125.2 ± 38.7		0.31
	PSw	64.1 ± 39.1	70.2 ± 48.7		0.22
	Esw	29.7 ± 17.3	29.4 ± 17.1		0.19
BF	Late SS	125.9 ± 36.4	122.3 ± 38.2		0.09
	PSw	63.0 ± 28.4	70.5 ± 37.6		0.29
	Esw	36.3 ± 19.1	36.4 ± 20.6		0.03
TA	Late SS	90.4 ± 39.8	93.3 ± 32.1		0.29
	PSw	105.0 ± 31.2	110.7 ± 41.8		0.28
	Esw	120.2 ± 48.7	103.9 ± 32.9		0.29
MG	Late SS	154.5 ± 32.4	148.8 ± 34.9		0.31
	PSw	90.2 ± 36.9	113.2 ± 45.2		0.34
	Esw	55.8 ± 27.4	60.1 ± 31.1		0.16
Sol	Late SS	171.6 ± 50.3	159.6 ± 45.6		0.31
	PSw	81.5 ± 33.4	99.2 ± 42.9	*	0.63
	Esw	59.0 ± 33.5	63.6 ± 36.4		0.06

Mean ± SD; *p<0.05; EMG: electromyography; ES: effect size; Normal: comfortable gait; NP-long: gait with increased non-paretic step length: RF: rectus femoris; VL: vastus lateralis; BF: biceps femoris; TA: tibialis anterior; MG: medial head of gastrocnemius; Sol: soleus; Late SS: second half of single support phase; PSw: pre-swing phase; Esw: early swing phase.

extensors, an increase in hip extension angular velocity was presumed to increase the rectus femoris activity, but no changes in rectus femoris activity were observed during the second half of the single-support phase. Lewek et al.[6] reported heteronymous reflexes from hip flexors to knee extensors produced by passive paretic hip extension (extension angular velocity 60°/s, 90°/s, and 120°/s) in stroke patients; however, the peak hip extension angular velocity in the second half of the single-support phase was normal, 49.8 ± 27.1°/s and NP-long, 62.7 ± 36.3°/s, which were lower than the hip extension angular velocity set by Lewek et al. This suggests that NP-long increased the peak hip extension angular velocity, although the original gait speed was slower and below the angular velocity at which the stretch reflex was elicited, no changes in rectus femoris activity were observed. In the pre-swing phase, the rectus femoris activity increased, and the hip flexion angular velocity decreased. The rectus femoris in the pre-swing phase assists in the advancement of the lower limb through its hip flexion action[15]; however, the rectus femoris in the hip extension range is reported to have a hip extension action[16]. In the NP-long, the rectus femoris is overactivated to advance the lower limb from hip extension and the hip flexion angular velocity may be decreased by the force applied in the hip extension direction. The soleus activity and ankle plantarflexion angular velocity increased during the pre-swing phase. Smooth weight transfer from the posterior to the anterior leg is necessary during the pre-swing phase. NP-long required more weight transfer to the anteriorly positioned nonparetic lower extremity than the normal, which may have resulted in increased soleus activity and ankle plantarflexion angular velocity. No changes were observed in the knee flexion angular velocity at toe-off and the peak knee flexion angle during the swing phase. Previous studies have reported not only rectus femoris overactivity but also insufficient ankle push off[17] as a factor limiting the knee flexion.

In the present study, increased soleus activity and ankle plantar flexion angular velocity were observed during the pre-swing phase, in addition to increased rectus femoris activity. In normal gait, the shank tilts forward and the knee flexes due to plantar flexor muscle activity during the pre-swing phase, suggesting that increased rectus femoris activity during the pre-swing phase was a factor that decreased the knee flexion angle during the swing phase, whereas increased soleus activity during the same period contributed to increased knee flexion angle, which may explain why the knee flexion angle did not change during the swing phase.

In this study showed that increasing the NP step length during gait increased the hip extension angle and TLA, rectus femoris, and soleus activity during the pre-swing phase but did not limit the peak knee flexion angle during the swing phase in hemiparetic patients with chronic stroke. Therefore, increasing the NP step length during gait was suggested to unlikely limit the knee flexion during the swing phase and can safely improve the propulsive force of the paretic leg. A limitation of this study is that we did not measure healthy participants; thus, we cannot say that our results are characteristics of hemiparetic patients with chronic stroke. As for the gait speed, gait pattern and use of ankle -foot orthoses may influence the results, but this study did not determine the effects of these factors. Further studies are needed to increase the number of healthy participants and stroke patients.

Conflict of interest

None.

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