

Case Report

Immediate effect of Walkbot robotic gait training on neuromechanical knee stiffness in spastic hemiplegia: A case report

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Abstract.

PURPOSE: The purpose of this study was to investigate the immediate effect of Walkbot gait training on knee joint stiffness in an individual with spastic hemiplegia.

METHOD: A woman with hemiparetic stroke underwent a 30-minute Walkbot robotic-assisted gait training session. Knee flexion stiffness associated with hamstring spasticity and knee extension torques during the terminal swing phase was determined before and after the intervention using the Walkbot-STIFF measurement system.

DESIGN: Descriptive case analysis.

RESULTS: Knee joint extension kinematic at the terminal swing phase increased from 2.44° to −0.28°. Knee joint torque increased from 0.26 Nm to 0.32 Nm. The knee flexion stiffness decreased from 0.0083 Nm/degree to 0.0022 Nm/degree following the training.

CONCLUSIONS: The Walkbot robotic-assisted locomotor training was effective for reducing knee joint stiffness and improving extensor torque during functional gait. Moreover, the Walkbot-STIFF system was useful for assessing and monitoring spasticity during locomotor training.

Keywords: Assistive device, cortical disinhibition, muscle stiffness, robotic-assisted gait training, stroke

1. Introduction

Abnormal knee stiffness associated with spasticity or hypertonicity is an important biomarker in spastic hemiplegia, which is often associated with functional knee joint terminal extension during locomotion. Spasticity is a common neurological impairment in stroke, and is characterized as a velocity-dependent stretch response

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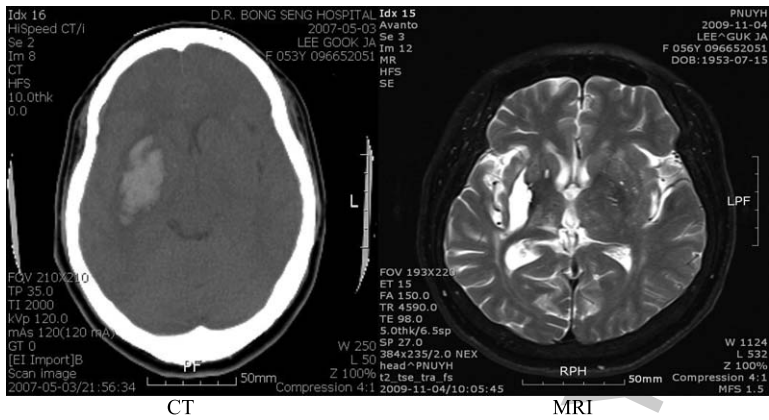


Fig. 1. Neurodiagnostic imaging.

to passive elongation of the involved muscle [10, 13, 24]. In particular, hamstring (antagonist) muscle spasticity co-activates the quadriceps muscle (agonist) from the midswing to the terminal swing phases of the gait cycle, thus constraining knee terminal extension. This limited kinematic knee extension results in decreased step length, stride length and walking velocity [4], and knee extension torque and moment. Moreover, a lack of mobilization of the knee joint muscle or prolonged immobilization of the hamstring muscle at a shortened length will create muscle stiffness and neuroreflexive sensitivity or threshold, which is associated with disused muscle atrophy, or decreased sarcomere or viscoelastic properties. This will eventually lead to joint contractures and limited functional range of motion (ROM) [20, 27] during the terminal swing phase of the gait cycle. Hence, it is important to mobilize the affected or hemiparetic limb as early as possible in stroke rehabilitation to prevent muscle stiffness and neuroreflexive sensitivity or threshold associated with disused muscle atrophy. Early mobilization through locomotor training has been shown to reduce spasticity and improve lower limb ROM or gait speed in hemiparetic stroke [6, 19].

Despite the important contribution of robotic-assisted training in lower-limb mobilization, there is a dearth of evidence on the effect of robotic-assisted gait training on stiffness control in hemiplegic stroke. We recently developed the Walkbot system, which is designed to provide active-assistive gait training with selective ankle joint actuation. Robotic-assisted gait training has gradually gained widespread acceptance among stroke rehabilitation clinicians because it offers the advantage of an intensive, repetitive, task-specific gait training paradigm [28] with comparable results to conventional stroke rehabilitation. Therefore, the

Table 1
General characteristics of the patient

Sex	Age	Height	Weight	Month after stroke	Paretic side	Stroke type
F	61	155	78	60	left	Hemorrhage

specific aim was to investigate the immediate effect of Walkbot gait training on knee joint stiffness in an individual with spastic hemiplegia during locomotion.

2. Methods

2.1. Case description

The subject (KJ) was a 61-year-old woman with left hemiparesis following a right intracerebral (basal ganglia) hemorrhage. She underwent a total hip replacement (arthroplasty) and craniotomy. The patient was initially admitted to the university medical center due to the sudden onset of muscle weakness and sensation in the left side, accompanied by loss of balance during gait. Neurodiagnostic imaging with MRI and CT confirmed intracerebral hemorrhage (Fig. 1). Once her hemorrhage was stabilized, she received physical therapy five times a week and had no prior experience of robotic-assisted gait training. At the time of the present study, she was able to walk about 30 feet independently, using a straight cane.

The patient provided written informed consent and the study was approved by the institutional review board (IRB) of Pusan National University Hospital. The patient's demographic and clinical characteristics are shown in Table 1.



Fig. 2. Robotic assisted gait training using the Walkbot system.

Table 2
Electronics and mechanical characteristics

Actuator	2 AC + 2DC servomotors
Ankle dorsiflexion control mode	Universal ^a EMG-triggered ^b FES modes
Foot switch	Pressure sensors (heel, metatarsal, toe)
Degrees of freedom	4
Automatic length adjustability	Thigh, Leg
Operating control for step motion	Remote JOG
Damper	Automatic On/Off
Operating system module	Unilateral, Bilateral, Single joint compartment

^aEMG: electromyography; ^bFES: functional electrical stimulation.

2.2. Apparatus

The Walkbot System (P&S Mechanics, Seoul, Korea) is a robotic-assisted locomotor training device, which is designed to facilitate earlier ambulation by providing adjusted body-weight-bearing control and real-time visual biofeedback for torque and stiffness, and kinematics for the hip, knee and ankle joints. The device consists of a suspension harness for body weight support (BWS), a motorized treadmill and an actuator-controlled exoskeleton (Fig. 2). The treadmill speed and torque (assistance and resistance) can be adjusted to accommodate the patient's level of locomotor performance as the training progresses. The characteristics of the Walkbot system are summarized in Table 2.

2.3. Experimental tasks and procedures

KJ's anthropometrical data, including her age, height, weight, foot length and width, femur and tibia length, were initially measured and used for assessment. KJ wore soft, light clothing to prevent interference

Table 3
Anthropometric and spatiotemporal data

Anthropometric data		Spatiotemporal data	
Thigh length	0.360 m	Step length	1.10 m
Leg length	0.360 m	Walking velocity	0.77 m/s
Ankle height	0.065 m		

with the stiffness measurements. She was secured with the Walkbot's harness and exoskeletal attachments for the thigh, shank and foot, which were adjusted to her anthropometric characteristics. Predetermined gait parameter data, including velocity (0.77 m/s), joint angle (the total knee joint excursion angle was set between 5° and 80°), percentage of body weight (BW) suspension, and walking time (30 minutes) were entered into the system (Table 3). KJ was then familiarized with the experimental procedure by practicing the robotic-assisted walking on the motorized treadmill.

2.4. Neuromechanical measurements

Neuromechanical measurements entailed knee joint kinematics (angular displacement), torque, and joint stiffness. The Walkbot system is equipped with Kinematic and Kinetic Computing software, which is capable of calculating the angular displacement, moment or torque of the hip, knee and ankle joints on a real-time base. The net momentum or torque was computed with the following equation:

$$\tau_{net} = \frac{dL}{dt} = \frac{d(I\omega)}{dt} = I \frac{d\omega}{dt} = I\alpha,$$

where τ is the total torque exerted on the body, L is the angular momentum vector, t is time, I is the mass moment of inertia of the body, ω is the angular velocity, and α is the angular acceleration of the body, measured in rad/S^2 .

The Walkbot system is modulated by five servomotors for the bilateral hip, knee and ankle joints, which enable the safe, coordinated control of locomotor kinematics and kinetics using encoders and a full dynamic model of the exoskeletal system. Each actuated servomotor has a built-in encoder to instantaneously detect the joint angle, angular velocity and acceleration, which are used for moment computation. Foot load sensors are used to monitor each foot's pressure distribution when the exoskeleton's feet are in contact with the treadmill surface, thus providing information on the center of the pressure trajectories during the stance phase. All of the foot sensors are hardwired to a network circuit of

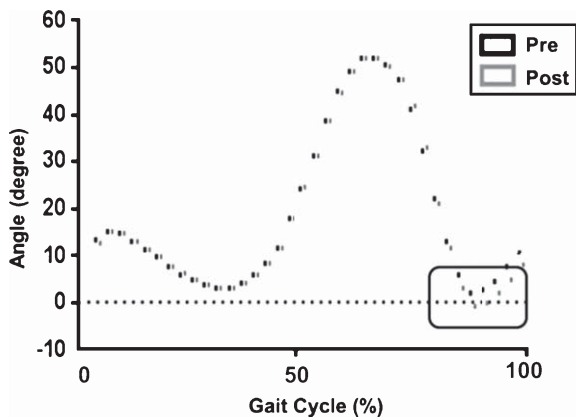


Fig. 3. Knee joint kinematics.

electronic input/output module (IOM) boards, which are then linked to a central command and operating system. The validity and feasibility of the intelligent Walkbot system have been well established [11].

Based on the kinematic and kinetic data, we determined the knee joint stiffness during the terminal swing phase of the gait cycle. This terminal swing phase event was chosen because hamstring (antagonist) muscle spasticity co-activates the quadriceps muscle (agonist) from the midswing to the terminal swing phases of the gait cycle, thereby constraining the terminal extension of the knee. This limited kinematic knee extension results in decreased step length and stride length, walking velocity and knee extension torque or moment [4].

The stiffness was expressed by estimating the slope of the linear regression in the torque-displacement curve [8, 21]. The knee joint angle, moment and torque data were collected before and after 30 minutes of Walkbot robotic-assisted gait training.

3. Results

3.1. Kinematic data

The knee joint kinematic at the terminal swing phase increased from 2.44° (pre-robotic walking) to -0.28° (post-robotic walking) (Fig. 3).

3.2. Torque data

Knee joint torque increased from 0.26 Nm (pre-robotic walking) to 0.32 Nm (post-robotic walking) at the terminal swing phase, yielding a 20% improvement in muscle strength (Fig. 4).

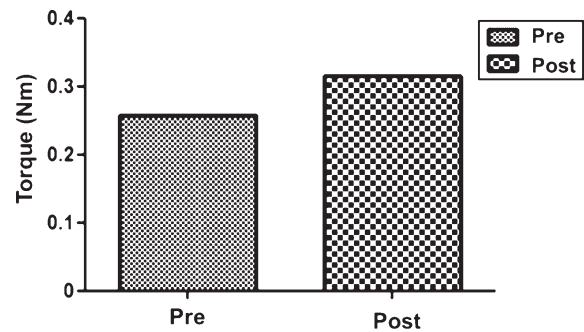


Fig. 4. Knee joint torque data.

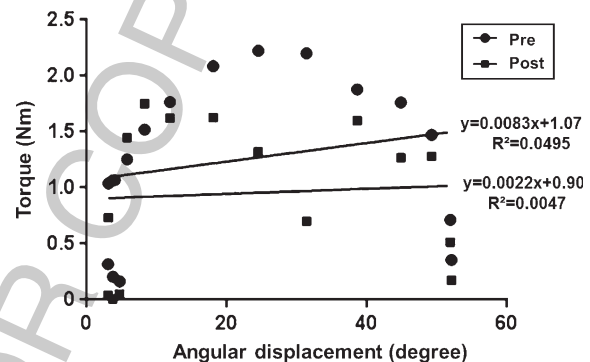


Fig. 5. Knee joint stiffness.

3.3. Stiffness (resistance) data

The total knee joint stiffness recorded for the entire gait cycle decreased from 0.0083 Nm/degree (pre-robotic walking) to 0.0022 Nm/degree (post-robotic walking), yielding a 70% reduction in muscle stiffness (Fig. 5).

4. Discussion

The present study highlighted the effect of robotic-assisted gait training on knee joint kinematics, torque and stiffness in a chronic hemiparetic stroke patient. As anticipated, we found that as little as 30-minutes of robotic-assisted gait training can produce a significant reduction in knee joint stiffness and increased joint kinematics during gait. This is the first clinical case trial to demonstrate the immediate effects of robotic-assisted gait training on knee joint stiffness, kinematics and torque in gait performance. Most importantly, our knee joint stiffness data showed that slope (angle-torque

relationship) decreased. Spasticity has been suggested to be positively related to muscle stiffness, which is a common neuromotor impairment following stroke [13, 14, 25]. Although the underlying neural mechanism remains unclear, motor cortex disinhibition is responsible for neuromotor impairment [7, 18, 22]. In fact, a recent transcranial magnetic stimulation study showed that intracortical inhibition was significantly decreased in the involved hemisphere [16]. Such cortical disinhibition may contribute inappropriate reciprocal inhibition to the antagonistic hamstring and gastrocnemius, which can lead to spasticity and associated plantar flexion and knee flexion stiffness during the terminal swing phase. Our knee joint stiffness data during the terminal phase showed a substantial reduction, from 0.0013 Nm/degree to 0.0004 Nm/degree. This improvement was also associated with improvements in the joint angle and torque. It has been theorized that cerebral insult can result in cortical disinhibition, leading to disruption of the normal reciprocal inhibition, which causes increased knee flexor (hamstring and gastrocnemius) spasticity.

Our findings are consistent with previous results [1, 6, 17, 19, 23]. Diserens et al. reported that 3-weeks of repetitive arm cycling decreased spasticity in the elbow joints of patients with chronic hemiparetic stroke [6]. Other studies have demonstrated that repetitive, active and assisted joint movement exercise was effective for reducing spasticity in patients with paraplegia [17] and post-stroke [1]. Similarly, Nuyens et al. [19] revealed a significant reduction in knee joint spasticity after passive isokinetic training in patients with hemiparetic stroke. Schmartz et al. [23] also demonstrated a positive effect on stiffness control associated with spasticity following robotic-assisted gait training in children with cerebral palsy. It is possible that this reduction in stiffness – associated with spasticity or hypertonicity following intensive, repetitive, and cyclic-passive or active-assisted movement training – may have resulted from reciprocal inhibition, whereby the inhibited agonistic quadriceps muscle was facilitated and the spastic antagonist hamstring muscle was inhibited [26]. In fact, as clearly demonstrated in our torque and stiffness data, knee extension torque increased while knee flexion stiffness decreased at the terminal swing. Muscle thixotropy, the neuromechanical property by which muscle becomes more pliable and flexible due to long-term molecular reorganization following repetitive concentric and eccentric movement, may be another possible mechanism [9, 12]. Nuyens et al. [19] stated that repeated movement may presynapti-

cally inhibit calcium releasing channels and decrease synaptic transmission, resulting in the ‘habituation’ or desensitization of excessive reflexive activity due to cortical disinhibition and a subsequent ephemeral reduction in spasticity [2, 3].

Our preliminary investigation provides the first stepping-stone towards larger-scale clinical trials to investigate the short- and long-term effects of Walkbot assisted gait training on spasticity and associated muscle stiffness. The Walkbot-STIFF measurement may be a reliable and valid instrument for quantifying changes in muscle stiffness associated with spasticity following robotic-assisted gait training. However, as in all spasticity tests, such measurements are often influenced by multiple factors including segment inertia, viscoelastic properties, joint integrity (i.e., contractures, ROM), antagonistic co-contraction, compliance, psychophysical state and voluntary muscle activity [5, 15]. This study invites further investigations of the confounding effects of the aforementioned factors – particularly the influence of reflex-mediated stiffness, active contraction or coactivation-mediated muscle stiffness using additional measures of surface electromyography –, which can be first established in a normal population before being tested in a neurological population.

5. Conclusion

In this study, we demonstrated a significant reduction in resistive knee flexion stiffness and a concurrent improvement in knee extensor torques during functional locomotion following Walkbot robotic-assisted gait training in a chronic hemiparetic stroke case. Consequently, our novel study provides a theoretical treatment rationale for Sherrington’s reciprocal inhibition and provides further evidence that repeated movements can produce antispastic effects in individuals with spastic hypertonia. Furthermore, our Walkbot-STIFF measurement is useful for the real-time assessment and monitoring of locomotor training effects using a visual (kinematics, torque, stiffness) and kinesthetic (torque) biofeedback system.

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Both Mr. Do Hyeon Kim and Dr. Yong-Il Shin have equally contributed to this paper.

References

- [1] Bressel, E., & McNair, P. J. (2002). The effect of prolonged static and cyclic stretching on ankle joint stiffness, torque relaxation, and gait in people with stroke. *Physical Therapy*, 82(9), 880-887.
- [2] Castellucci, V., Carew, T., & Kandel, E. (1978). Cellular analysis of long-term habituation of the gill-withdrawal reflex of *Aplysia californica*. *Science*, 202(4374), 1306-1308.
- [3] Castellucci, V. F., & Kandel, E. R. (1974). A quantal analysis of the synaptic depression underlying habituation of the gill-withdrawal reflex in *Aplysia*. *Proceedings of the National Academy of Sciences*, 71(12), 5004.
- [4] Dietz, V., & Berger, W. (1983). Normal and impaired regulation of muscle stiffness in gait: A new hypothesis about muscle hypertonia. *Exp Neurol*, 79(3), 680-687.
- [5] Dietz, V., Quintern, J., & Berger, W. (1981). Electrophysiological studies of gait in spasticity and rigidity. Evidence that altered mechanical properties of muscle contribute to hypertonia. *Brain: A Journal of Neurology*, 104(3), 431.
- [6] Diserens, K., Perret, N., Chatelain, S., Bashir, S., Ruegg, D., Vuadens, P., & Vingerhoets, F. (2007). The effect of repetitive arm cycling on post stroke spasticity and motor control: Repetitive arm cycling and spasticity. *J Neurol Sci*, 253(1-2), 18-24.
- [7] Domann, R., Hagemann, G., Kraemer, M., Freund, H. J., & Witte, O. (1993). Electrophysiological changes in the surrounding brain tissue of photochemically induced cortical infarcts in the rat. *Neuroscience letters*, 155(1), 69-72.
- [8] Farley, C. T., & Gonzalez, O. (1996). Leg stiffness and stride frequency in human running. *Journal of Biomechanics*, 29(2), 181-186.
- [9] Hagbarth, K., Hägglund, J., Nordin, M., & Wallin, E. (1985). Thixotropic behaviour of human finger flexor muscles with accompanying changes in spindle and reflex responses to stretch. *The Journal of Physiology*, 368(1), 323-342.
- [10] Jones, E., & Mulley, G. (1982). *The measurement of spasticity*. *Advances in Stroke Therapy*, (pp. 187-195), Raven Press, New York.
- [11] Jung, J. H., Lee, N. G., You, J. H., & Lee, D. C. (2009). Validity and feasibility of intelligent Walkbot system. *Electronics Letters*, 45(20), 1016-1017.
- [12] Lakie, M., & Robson, L. G. (1988). Thixotropic changes in human muscle stiffness and the effects of fatigue. *Experimental Physiology*, 73(4), 487-500.
- [13] Lance, J. (1980). Symposium synopsis. *Spasticity: Disordered motor control*, 485-494.
- [14] Lance, J. W. (1990). What is spasticity? *The Lancet*, 335(8689), 606.
- [15] Levine, M. G., & Kabat, H. (1954). SPASTICITY—Its Nature and Treatment. *California Medicine*, 80(4), 306.
- [16] Liepert, J., Storch, P., Fritsch, A., & Weiller, C. (2000). Motor cortex disinhibition in acute stroke. *Clinical Neurophysiology*, 111(4), 671-676.
- [17] Lunenburger, L., Colombo, G., Riener, R., & Dietz, V. (2005). Clinical assessments performed during robotic rehabilitation by the gait training robot Lokomat (pp. 345-348): IEEE.
- [18] Mittmann, T., Luhmann, H., Schmidt-Kastner, R., Eysel, U., Weigel, H., & Heinemann, U. (1994). Lesion-induced transient suppression of inhibitory function in rat neocortex *in vitro*. *Neuroscience*, 60(4), 891-906.
- [19] Nuyens, G. E., De Weerd, W. J., Spaepen, A. J., Jr., Kiekens, C., & Feys, H. M. (2002). Reduction of spastic hypertonia during repeated passive knee movements in stroke patients. *Arch Phys Med Rehabil*, 83(7), 930-935.
- [20] Okita, M., Yoshimura, T., Nakano, J., Motomura, M., & Eguchi, K. (2004). Effects of reduced joint mobility on sarcomere length, collagen fibril arrangement in the endomysium, and hyaluronan in rat soleus muscle. *J Muscle Res Cell Motil*, 25(2), 159-166.
- [21] Padua, D. A., Arnold, B. L., Perrin, D. H., Gansneder, B. M., Carcia, C. R., & Granata, K. P. (2006). Fatigue, vertical leg stiffness, and stiffness control strategies in males and females. *Journal of Athletic Training*, 41(3), 294.
- [22] Reinecke, S., Lutzenburg, M., Hagemann, G., Bruehl, C., Neumann-Haefelin, T., & Witte, O. (1999). Electrophysiological transcortical diaschisis after middle cerebral artery occlusion (MCAO) in rats. *Neuroscience Letters*, 261(1-2), 85-88.
- [23] Schmartz, A. C., Meyer-Heim, A. D., Muller, R., & Bolliger, M. (2011). Measurement of muscle stiffness using robotic assisted gait orthosis in children with cerebral palsy: A proof of concept. *Disabil Rehabil Assist Technol*, 6(1), 29-37.
- [24] Sheean, G. (2002). The pathophysiology of spasticity. *European Journal of Neurology*, 9, 3-9.
- [25] Shepherd, R. B. (2001). Exercise and training to optimize functional motor performance in stroke: Driving neural reorganization? *Neural Plasticity*, 8(1/2), 121-130.
- [26] Sherrington, C. (1907). On Reciprocal Innervation of Antagonistic Muscles. Tenth Note. *Proceedings of the Royal Society of London Series B Containing Papers of a Biological Character*, 79(532), 337-349.
- [27] Williams, P. E., & Goldspink, G. (1978). Changes in sarcomere length and physiological properties in immobilized muscle. *J Anat*, 127(Pt 3), 459-468.
- [28] Winchester, P., & Querry, R. (2006). Robotic orthoses for body weight-supported treadmill training. *Phys Med Rehabil Clin N Am*, 17(1), 159-172.