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Original Article

Effect of paralyzed side soleus muscle pressure on the gait of stroke patients as measured by a three-dimensional motion analysis system

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Abstract. [Purpose] The purpose of this study was to examine the effects of muscle belly compression by a supporter on the paralyzed side soleus muscle of patients with cerebrovascular disability, and to determine the intensity of compression that is effective for improving gait. [Subjects] Eleven patients with chronic cerebral vascular disorder. [Methods] Before setting the supporter, standing posture and 6 m free walking were measured 3 times with the three-dimensional motion analysis system, VICON. Then, supporters were placed on the center of the lower leg of the hemiplegic side of the subjects and inflated to 30 or 50 mmHg. Three minutes after wearing the supporters, the subjects walked again for 3 times. The data measured with VICON were processed using Visial3D.V4, and the angles of the ankle, steps of the hemiplegic and non-hemiplegic sides, walking speed, walk rate and cadence were calculated. [Results] Compared to without a supporter, a supporter with 30 mmHg pressure showed a significant reduction in the angle of the knee at Initial Contact (IC), and a significant increase in the power of the knee extension at Loading Response (LR). [Conclusion] The results reveal a supporter with that of the subjects during pressure over 30 mmHg applied for 3 minutes improved the knee angle power and hemiplegia walking.
Key words: Cerebrovascular disabilities, Pressure, Walking

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INTRODUCTION

Gait of hemiplegic patients with cerebral vascular disorders present several characteristics. Since eccentric contraction of the muscles of ankle dorsiflexion does not occur at the time of initial contact, hemiplegic patients often display equinovarus foot due to spasticity, the entire surface of the foot contacts the ground. Leg anteversion in mid stance does not occur with heel grounding; therefore, the anterior and upward movement of the center of gravity stops. The step length becomes short narrow due to inadequate locker function, because the potential energy using the gravity from up and the driving force to the front are decreased, and a posterior type and a matching-shaped gait frequently occur. In addition, since eccentric contraction in mid stance is insufficient without the action of the plantar flexors, the step length becomes short narrow, and it becomes difficult to perform kick in the terminal stance.

In contrast, repetition of brake action and accelerator action by eccentric contraction and concentric contraction of the lower limbs are clearly observed in the gait of a physically unimpaired subject, and three locker functions including potential energy are act. Focusing on the ankle joint in stance phase, the eccentric contraction of the ankle dorsiflexion at the time of the initial contact cushions the impact of foot grounding and the lower leg is inclined forward at the time from a loading response to the first half of the mid-stance phase. In the latter half of the mid stance phase, flexion of the ankle joint is braked by the plantar flexors, and center of gravity moves to the front of the foot. Concentric contraction of ankle flexor muscle occurs in the terminal stance phase, enabling kicking motion.

A contracture that causes talipes equinus of the foot part is a serious problem in physically healthy subjects and subjects with hemiparesis. The definition of contracture is a state in which muscle tone increases in an speed-dependent Malener due to cut-off through descending control from the brain[1)]. Increasing spinal cord motor nerve excitation causes a sthenia of the Achilles tendon reflex and hyperactivity of the triceps surae muscle affects the stability of contracture and motion. It was previously demonstrated that it is possible to suppress the excitability of spinal cord motor nerves by applying pressure to the paralyzed side soleus muscle belly of stroke patients[2)]. This study was conducted to show that contracture during motion can be suppressed by the application of moderate pressure to the soleus muscle, improving dynamic balance, for the development of physiotherapy treatments based on emperical evidence.

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Table 1. Subjects' characteristics

Subjects	Gender	Age	diagnosis	Paralysis side	L/E Br. S	Ankle joint passive ROM	Plantar muscle MAS	The years from the onset	Level of walking		Assistive devices	
									inside	outside		
A	Male	63	Cerebral infarction	L	III	0 (−5)	0	8	Independent	Independent	T-cane	SHB
B	Male	75	Cerebral infarction	R	VI	5	1	10	Independent	Independent	T-cane	no use
C	Male	70	Cerebral infarction	L	IV	−5	1	10	Supervision	Supervision	T-cane	SLB
D	Male	75	Cerebral hemorrhage	L	IV	−10	2	23	Independent	Independent	T-cane	no use
E	Male	75	Cerebral hemorrhage	R	VI	5	1	12	Independent	Independent	T-cane	no use
F	Male	71	Cerebral infarction	L	IV	0 (−20)	0	11	Supervision	Supervision	T-cane	SHB
G	Male	81	Cerebral hemorrhage	L	IV	−5 (−30)	3	17	Supervision	Light assistance	T-cane	no use
H	Male	63	Cerebral hemorrhage	R	IV	−10	3	9	Independent	Independent	no use	no use
I	Female	64	Cerebral hemorrhage	R	IV	−20 (−25)	3	12	Independent	Independent	T-cane	(SHB)
J	Female	70	Cerebral hemorrhage	R	IV	−10	2	5	Independent	Independent	T-cane	SHB
K	Male	81	Cerebral infarction	L	VI	5	1	23	Independent	Independent	T-cane	no use

SUBJECTS AND METHODS

Subjects who were unable to understand the oral instructions because of severe high order brain dysfunction diagnosis, those who had orthopedics disorders or disorders that affected their walking, and those who were unable to understand the oral instructions or had difficulty in walking were excluded. Presence of paralysis was confirmed by the Achilles tendon reflex. The period from stroke onset was an average 12.7 years; 5 subjects had cerebral infarction, and 6 had cerebral hemorrhage; and 5 subjects had right hemiplegia and 6 had left hemiplegia. All of the subjects received instruction in advance, and answered a questionnaire. The measurements were carried out after obtaining approval for this study and the collection of basic information. This study was performed after obtaining approval from the ethic screening committee of the International University of Health and Welfare (authorization number: 10-130(2)). The characteristics of the subjects are shown in Table 1.

A three dimensional motion analysis system (VICON Inc. with 9 infrared cameras) and force plates (AMTI Corp) were used to measure the subjects' walking.

Elastic supporters of 20 cm (Health Point Corp.) were used to compress the soleus muscle on the hemiplegic side. Pressure was monitored by a pressure measuring instrument, PREDIA (Molten Corp.). The infrared reflective markers were put on 15 points: the bilateral acromion, hip joint, knee joint, external condyle of the fibula, head of the fifth metatarsal bone, and the epicondylus lateralis humeri. Measurements of the ground reaction force and the three dimensional motion analysis system were synchronized and recorded on a personal computer at a sampling frequency of 100 Hz.

The principal action was free walking over 6 m. The subjects performed walking three times under three different conditions: barefoot, and with two levels of pressure applied to the hemiplegic lower leg. The pressures were 30 and 50 mmHg. The free walking was prompted by the request: "Please walk at your normal speed".

Visial3D.V4 (C-motion company) software was used for kinematic, and dynamic motion analyses. The markers coordinate data were filtered with a 6–18 Hz Butterworth filter, and ground reaction force, three joint angles of the lower limbs, moment and power were calculated using six degrees of freedom models. Based on observational gait analysis by the Rancho Los Amigo method, the walk was divided into initial contact (IC), loading response (LR), mid-stance (MS), terminal stance (TS) and pre-swing (PS) phases, and the angles of the ankle, knee joint, and hip joint, moment, and the maximum and minimum power were calculated for each phase. In addition, walking speed, steps of the paralyzed and non-paralyzed sides, step time and ground position of the center of gravity were calculated using the vertical component of the reaction force as a distance factor.

For statistical processing, one-way analysis of variance with repeated measures was performed, and the gait parameters at each pressure strength were compared using the multiple comparison Bonferroni method of SPSS Ver.16. Significance was accepted for values of $p<0.05$.

RESULTS

The results of the gait parameters of each pressure strength are shown in Table 2 .

Compared with no pressure, the knee joint flexion angle in IC with a pressure of 30 mmHg applied to the paralyzed side soleus muscle significantly decreased ($F=2.563$, $p<0.05$), and the power of the knee extension in LR significantly increased ($f=2.887$, $p<0.05$). Significant differences were not found among the conditions for any of the other indices.

DISCUSSION

By calculating and comparing distance factors and kinematic and dynamic motion parameters at the time of walking, and the changes in these parameters with pressure applied to the subjects' lower legs it was revealed that, the knee joint flexion angle in IC significantly decreased when a pressure of 30 mmHg was applied to the paralyzed side leg, and the power of the knee extension in LR significantly increased.

At IC, for healthy persons, the ankle is in the cadaver

Table 2. Average, standard error and statistical results of gait parameters at the different pressures

Parameter of the walk	0 mmHg	30 mmHg	50 mmHg	p-value	0 mmHg	30 mmHg	50 mmHg
Spatiotemporal							
Walking speed (m/s)	0.37 (0.070)	0.39 (0.075)	0.39 (0.080)				
Loading response time (s)	0.49 (0.103)	0.55 (0.165)	0.55 (0.140)				
Single stance time (s)	1.00 (0.105)	0.99 (0.095)	0.93 (0.126)				
Pre-swing time (s)	0.48 (0.087)	0.51 (0.097)	0.53 (0.102)				
Non-paretic step time (s)	0.80 (0.117)	0.87 (0.185)	0.88 (0.152)				
Paretic step time (s)	1.00 (0.110)	0.99 (0.100)	1.00 (0.120)				
Non-paretic step length (%)	0.25 (0.040)	0.25 (0.041)	0.26 (0.046)				
Paretic step length (%)	0.28 (0.037)	0.30 (0.037)	0.29 (0.040)				
COG							
Loading response (Max)	0.05 (0.017)	0.05 (0.017)	0.05 (0.015)				
Loading response (Min)	−0.01 (0.015)	−0.01 (0.016)	−0.01 (0.013)				
Pre-swing (Max)	0.05 (0.018)	0.04 (0.017)	0.05 (0.016)				
Pre-swing (Min)	0.05 (0.018)	0.04 (0.017)	0.05 (0.016)				
Stance (Max)	0.06 (0.019)	0.06 (0.018)	0.06 (0.017)				
Stance (Min)	−0.02 (0.015)	−0.02 (0.016)	−0.02 (0.013)				
GRF							
Loading response (Max)	0.05 (0.011)	0.05 (0.010)	0.05 (0.011)				
Loading response (Min)	−0.04 (0.012)	−0.04 (0.013)	−0.04 (0.013)				
Pre-swing (Max)	−0.01 (0.015)	−0.02 (0.016)	−0.01 (0.013)				
Pre-swing (Min)	0.05 (0.013)	0.05 (0.012)	0.05 (0.013)				
Stance (Max)	0.04 (0.019)	0.04 (0.020)	0.04 (0.021)				
Stance (Min)	−0.02 (0.023)	−0.02 (0.022)	−0.02 (0.024)				
Ankle							
Angle at initial contact (Deg)	−0.47 (1.927)	−0.78 (2.039)	0.85 (1.977)				
Peak plantar flexion angle in loading response (Deg)	2.85 (1.641)	2.86 (1.780)	4.17 (1.500)				
Max morment in loading response (Nm/kg)	0.50 (0.076)	0.53 (0.080)	0.53 (0.084)				
Max power in loading response (W/kg)	0.06 (0.024)	0.06 (0.022)	0.06 (0.023)				
Plantar flexion angle in loading response (Deg)	−4.62 (1.301)	−5.55 (1.342)	−3.43 (1.356)				
Min morment in loading response (Nm/kg)	−0.006 (0.001)	−0.007 (0.001)	−0.006 (0.001)				
Min power in loading response (W/kg)	−0.24 (0.054)	−0.30 (0.064)	−0.26 (0.072)				
Peak dorsiflexion in stance (Deg)	6.60 (1.175)	6.67 (1.195)	6.90 (1.038)				
Max morment in stance (Nm/kg)	0.81 (0.077)	0.82 (0.078)	0.90 (0.105)				
Max power in stance (W/kg)	0.69 (0.148)	0.73 (0.166)	0.85 (0.221)				
Plantar flexion angle in stance (Deg)	−6.55 (1.428)	−7.15 (1.842)	−6.16 (1.537)				
Min morment in stance (Nm/kg)	−0.01 (0.001	−0.01 (0.002)	−0.01 (0.001)				
Min power in stance (W/kg)	−0.35 (0.064	−0.39 (0.079)	−0.50 (0.127)				
Peak plantar flexion in pre-swing (Deg)	5.70 (1.065)	5.78 (1.056)	5.52 (0.839)				
Max morment in pre-swing (Nm/kg)	0.80 (0.076)	0.80 (0.078)	0.89 (0.105)				
Max power in pre-swing (W/kg)	0.68 (0.150)	0.73 (0.167)	0.85 (0.222)				
Min angle in pre-swing (Deg)	−5.69 (1.427)	−6.20 (1.880)	−5.38 (1.569)				
Min morment in pre-swing (Nm/kg)	−0.001 (0.005)	−0.008 (0.002)	0.025 (0.031)				
Min power in pre-swing (W/kg)	−0.19 (0.057)	−0.23 (0.067)	−0.34 (0.134)				
Knee							
Angle at initial contact (Deg)	12.93 (2.244)	12.12 (2.236)	13.12 (2.213)	0.013		*	
Peak flexion in loading response (Deg)	14.73 (2.423)	14.46 (2.520)	15.16 (2.580)				
Max morment in loading response (Nm/kg)	0.09 (0.034)	0.09 (0.035)	0.09 (0.033)				
Max power in loading response (W/kg)	0.16 (0.055)	0.16 (0.056)	0.16 (0.049)				
Min angle in loading response (Deg)	7.41 (2.691)	7.40 (2.754)	7.71 (2.855)				
Min morment in loading response (Nm/kg)	−0.39 (0.095)	−0.38 (0.083)	−0.42 (0.099)				

Table 2. Continued

Parameter of the walk	0 mmHg	30 mmHg	50 mmHg	p-value	0 mmHg	30 mmHg	50 mmHg
Min power in loading response (W/kg)	−0.20 (0.059)	−0.25 (0.065)	−0.24 (0.058)	0.033		*	
Peak extension in stance (Deg)	25.38 (3.541)	24.64 (3.222)	25.77 (3.442)				
Max morment in stance (Nm/kg)	0.13 (0.034)	0.15 (0.034)	0.14 (0.036)				
Max power in stance (W/kg)	0.36 (0.088)	0.32 (0.076)	0.40 (0.089)				
Min angle in stance (Deg)	0.79 (2.427)	0.77 (2.120)	1.84 (2.562)				
Min morment in stance (Nm/kg)	−0.52 (0.093)	−0.50 (0.084)	−0.55 (0.095)				
Min power in stance (W/kg)	−0.38 (0.097)	−0.42 (0.113)	−0.42 (0.113)				
Peak flexion in pre-swing (Deg)	24.85 (3.604)	24.24 (3.224)	25.52 (3.459)				
Max morment in pre-swing (Nm/kg)	0.10 (0.025)	0.11 (0.025)	0.10 (0.023)				
Max power in pre-swing (W/kg)	0.33 (0.094)	0.28 (0.080)	0.37 (0.093)				
Min angle in pre-swing (Deg)	1.47 (2.489)	1.34 (2.138)	2.67 (2.620)				
Min morment in pre-swing (Nm/kg)	−0.41 (0.092)	−0.40 (0.082)	−0.43 (0.088)				
Min power in pre-swing (W/kg)	−0.18 (0.054)	−0.18 (0.048)	−0.17 (0.051)				
Hip							
Flexion at initial contact (Deg)	12.78 (3.202)	13.18 (3.039)	14.76 (2.451)				
Peak extension in stance (Deg)	17.05 (2.029)	16.81 (2.250)	17.55 (1.995)				
Max morment in loading response (Nm/kg)	0.60 (0.085)	0.60 (0.095)	0.60 (0.088)				
Max power in loading response (W/kg)	0.39 (0.104)	0.40 (0.093)	0.44 (0.104)				
Min angle in loading response (Deg)	5.51 (2.432)	5.80 (2.288)	6.03 (2.464)				
Min morment in loading response (Nm/kg)	−0.15 (0.042)	−0.15 (0.054)	−0.13 (0.033)				
Min power in loading response (W/kg)	−0.16 (0.048)	−0.17 (0.057)	−0.12 (0.035)				
Max angle in stance (Deg)	17.06 (2.030)	16.82 (2.250)	17.55 (1.995)				
Peak extensor moment in stance (N.m/ (kg.m))	0.68 (0.149)	0.67 (0.146)	0.68 (0.137)				
Max power in stance (W/kg)	0.47 (0.116)	0.47 (0.110)	0.53 (0.134)				
Min angle in stance (Deg)	−7.91 (1.627)	−8.22 (1.390)	−8.09 (1.642)				
Peak flexion moment in stance (N.m/ (kg.m))	−0.31 (0.051)	−0.33 (0.049)	−0.32 (0.051)				
Min power in stance (W/kg)	−0.25 (0.060)	−0.25 (0.058)	−0.22 (0.056)				
Peak flexion in pre-swing (Deg)	0.26 (1.819)	0.56 (1.793)	−0.21 (1.724)				
Max morment in pre-swing (Nm/kg)	0.07 (0.061)	0.05 (0.052)	0.06 (0.057)				
Max power in pre-swing (W/kg)	0.25 (0.065)	0.25 (0.061)	0.27 (0.078)				
Min angle in pre-swing (Deg)	−7.91 (1.627)	−8.21 (1.390)	−8.09 (1.642)				
Min morment in pre-swing (Nm/kg)	−0.29 (0.053)	−0.31 (0.050)	−0.31 (0.052)				
Min power in pre-swing (W/kg)	−0.18 (0.056)	−0.18 (0.062)	−0.18 (0.062)				

Compared with no pressure, the knee joint flexion angle in IC with a pressure of 30 mmHg applied to the paralyzed side soleus muscle significantly decreased ($F=2.563$, $p<0.05$), and the negative power of the knee joint in LR significantly increased ($f=2.887$, $p<0.05$). Significant differences were not found among the conditions for any of the other indices

position, the knee is in an extended position, and the hip joint is flexed at 30 degrees to stabilize the knee and provide smooth forward movement[3]. It has been reported that ankle joints of a subject with cerebrovascular disorder often present plantar flexion accompanied with grounding of the entire area of the sole, or grounding of the distal foot, and smooth grounding of the foot as an axis of the heel does not occur[4]. Furthermore, it has been reported that the knee becomes unstable due to functional decline of the ankle and hip joints and often adopts hyperextended or flexed positions[4]. In comparison with the other conditions, the 30 mmHg pressure applied to the triceps surae decreased the gonycampsis angle, and gait improved because the legs were supported in the extended position. As for its mechanism, it is possible that pressure on the triceps surae suppressed spasticity, restoring the arthrodesis function of the normal leg position resulting stable grounding with the knee extended. The soleus stabilizes the ankle, and stabilizes the knee in the extended position[5]. Since the arthrodesis function occurred because of suppression of spasticity, and in addition spasticity of the gastrocnemius that acts on knee flexion was suppressed because spasticity of the soleus was suppressed, knee extension occurred.

In LR, healthy subjects show an impact absorption reaction at the ankle and knee. Impact is absorbed by limiting flexion of the knee by eccentric contraction of the quadriceps femoris[3]. The increase in the power of the knee extension lower leg pressure of 30 mmHg indicates there was an improvement in the impact absorption function and maintenance of load stability on the knee. Pressure on the

lower leg suppresses movement in the ankle plantar flexion direction due to the spasticity, and the heel rocker function, that is important for forward movement but barely occurs in a subject with cerebrovascular disorder, occurred. As a result, improvement was seen in the important impact absorption mechanism through eccentric contraction of the knee extension muscle that occurs in response to the heel rocker function. Furthermore, contraction of the quadriceps femoris and gluteus maximus which are the knee extension muscles physically unimpaired subjects pushes up the pelvis and trunk. It has been reported that in subjects with cerebrovascular disorders, the heel rocker action does not arise in IC to LR; therefore, the center of gravity is not lifted, and forward rotation of the body with the ankle as the axis in the early period of MS is obstructed[4]. In this study, variation in the center of gravity (COG) was not observed, though knee extension at the time of IC with pressure on the triceps surae, and improvement in the muscle power of the quadriceps femoris at the time of LR made body movement to the upper front direction easier. The improvement in center of gravity movement to the upper front direction during IC to LR was an improvement that beneficially influenced the subsequent gait action, preventing knee hyperextension and hyperflexion after MS. Therefore, detailed verification of the movement speed of the center of gravity when applying pressure to the leg is a future study topic.

It is known that the H-wave, which indicates spinal cord motor nerve excitability, is usually suppressed more in the standing position than in the sitting and abdominal positions and that pre-synaptic inhibition is enhanced during walking[6]. However, it has been reported that the spinal cord motor nerve excitability of subjects with cerebrovascular disorder is excited even more in the standing position than in the sitting position, requiring greater suppression of excitation in quiet standing and in gait[7]. Similarly, suppression probably did not occur when walking at the time when suppression of the spinal cord motor nerve excitability was required. This study clarified that 30 mmHg pressure at this time, similar to the orthosis which has plantar flexior breaking, suppresses spasticity in IC and LR of the gait cycle, helping to extend the knee joint and increase extension muscle strength. This result suggests the possibility of applying pressure to suppress spasticity during gait training for subjects with cerebrovascular disorders.

In this study, subjects with relatively high degrees of autonomy in walking were recruited. Since it has been reported that subjects with cerebrovascular disorders and a low degree of autonomy need greater plantar flexion braking for knee locking, and that subjects with a high degree of walking autonomy show improvement with small improvements in small plantar flexion braking, indicating that application of pressure is effective for subjects with cerebrovascular disorders who have a high degree of walking autonomy. Further, we could not verify the facilitation of optional joint motion of antagonist muscles by electromyography. A future study should verify the effects of spasticity suppression by electromyography for the motions that require high control of the ankle. Furthermore, it will be necessary to investigate subject data in greater detail to determine which of rehabilitation, medical disease, or peripheral problems affect the spasticity conditions of subjects.

The soleus is an antigravity muscle, and therefore it is usually works to control posture. In particular, it activates in the preliminary stage of gait before motion in the standing position. At this time, the reflex function is also adjusted by high order nerves so that the attempted motion can be achieved successfully in the presented situation, and the action incorporated into the motion. In addition, the reflex function causes muscle tonus before the voluntary contraction to some extent, and optional muscle contraction to make muscle tone effective as soon as possible[8]. Myotatic reflex of the crural muscle is a reflex response that maintains muscle length, and is continuously involved in voluntary movement adjusting reflex activity to the motion[9]. This adjustment was clarified in a study that reported that the H/M values indicating soleus spinal cord motor nerve excitability vary depending on postural change in the dorsal position, standing position and unilateral leg load position and gait[10]. However, subjects with cerebrovascular disorder and spinal cord injury whose high order nerves are affected have insufficient reflex control, and it is thought that adjustment not only at the time of rest but also before motion does not occur making it difficult to suppress control in particular. In this study, the effect of pressure on spasticity suppression at rest and during walking was verified. A future study will attempt to verify the pressure effect at the times of postural changes that require greater spasticity suppression and at the time of the preliminary stage of motion to determine the appropriate muscle pressures for spasticity suppression to assist physical therapists in actual treatments that will lead to more effective physical therapy.

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