

1 **Effect of Facilitating Reciprocal Inhibition of Ankle Flexors on Muscle Activation**  
2 **Pattern in Sit to Stand Movement in Stroke -Case Study-**

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14**Purpose** The aim of this study was to determine the effects of facilitating reciprocal inhibition of ankle  
15dorsi-Plantar flexors on leg muscle activation patterns during the sit to stand movement. **Methods** Fifteen  
16patients with hemiplegia were randomly recruited from Y hospital. Subjects stood up from an armless plinth  
17with a comfortable and self-paced speed. Onset-offset times of leg muscles during the sit to stand movement  
18were measured using surface electromyograph and a force platform. **Results** The mean onset time of muscle  
19activation in paretic tibialis anterior (TA) was significantly earlier after treatment ( $p<.05$ ), and soleus (SOL),  
20which showed a significant delay ( $p<.05$ ) in paretic side. However, there was no significant difference between  
21pre- and post-treatment in rectus femoris (RF) and biceps femoris (BF) ( $p>.05$ ) in paretic side. There were  
22significant differences between pre- and post-treatment in onset time of both paretic and non-paretic leg muscle  
23( $p<.05$ ). The recruitment order of muscle activity in pre-treatment was in sequence of RF, SOL, then followed  
24by TA activation. In post-treatment, it was in order of RF activation, TA, BF and then SOL activation.  
25**Conclusion** It was found that the therapeutic exercise to facilitate the intrinsic muscle and antagonistic activity  
26of SOL in paretic foot and leg made significant effects on enhancing reciprocal inhibition for efficient postural  
27control.

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29**Key words** Ankle function, Muscle activation pattern, Reciprocal inhibition, Sit to stand movement

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47The sit-to-stand movement is a precondition for independent locomotion and activities of daily living.<sup>1)</sup> During  
48the sit-to-stand movement, a stable sitting posture is converted into a relatively unstable standing posture<sup>2)</sup> and it  
49is a transitional movement that connects sitting posture to walking. Healthy persons can easily and naturally  
50perform this movement but hemiplegic patients due to stroke show difficulties in performing this movement.<sup>3)</sup>

51In the lower limb muscle activation pattern in the sit-to-stand movement, the tibialis anterior (TA) muscle is  
52activated first to move the body's center of gravity forward and plays central roles for the timing and pattern of  
53ankle muscle activation.<sup>4, 5)</sup> Almost simultaneously with the activation of the TA, the rectus femoris (RF) muscle  
54is activated to work as a knee extensor to provide the stability of the knee and the hip joint. After the activation  
55of the TA and the RF, the biceps femoris (BF) muscle is activated. The BF maintains the stability of the knee  
56when the hip has been taken off the bed and helps the extension of the hip joint.<sup>6)</sup> The soleus (SOL) muscle is  
57activated last and plays the role of adjusting the speed of knee extension when the maximum ankle flexion is  
58converted into extension in the stage of the sit-to-stand movement and controlling the posture so that stable  
59standing postures can be maintained.

60 Normally, the TA and the SOL are reciprocally inhibited while flexion and extension are attempted.<sup>5)</sup>  
61Reciprocal inhibition refers to alternating occurrence of inhibition and facilitation of an agonistic and an  
62antagonistic muscle and this function does not properly operate in some pathological conditions. For instance, if  
63the activity of the SOL increases abnormally, muscle activity will become hard even if the TA is normal.<sup>7, 8)</sup>

64 A movement that is the most frequently used in functional movement assessment performed at clinics is the  
65sit-to-stand movement. The sit-to-stand movement is also used for therapeutic purposes such as muscle  
66strengthening, improvement of postural control, and functional activities. Therefore, the sit-to-stand movement  
67and the stand-to-sit movement are diversely performed in assessments and therapeutic approaches. However,  
68studies related to the foot investigated changes in the order and duration of muscle activation according to foot  
69positions in healthy persons and hemiplegic patients.

70 The purpose of this study was to investigate the effects of the exercises which are to active intrinsic foot  
71muscles and facilitate reciprocal inhibition of ankle muscles on the neurologic factors that determine muscle  
72balance during the sit-to-stand movement.

**741. Study subjects**

75 The subjects of this study were fifteen hemiplegic patients (8 males and 7 females) after stroke at Y hospital  
76who fully and sufficiently understood and agreed to the purpose of the study. After being evaluated, the patients  
77were provided with treatment intervention that facilitated reciprocal inhibition of the ankle dorsi-plantar flexors

78for 30 minutes. After the therapeutic intervention, the patients could take a rest in a supine posture for 10  
79minutes and reevaluation was performed thereafter. The inclusion criteria were as follows: 1) no past history of  
80respiratory, 2) no past musculoskeletal disease 3) no perceptual disturbance such as hemi-neglect, 4) capable of  
81independent sit-to-stand movements, and 5) at least 24 points in the Korean version of the mini-mental state  
82examination test (K-MMSE) (Table 1).

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84 Table 1. General characteristics of the subjects (N=15)

Variable		n(%)	Mean±SD
Age (yrs)		15	47.86±16.73
Sex	Male	9(60)	
	Female	6(40)	
Body weight (kg)			61.33±8.37
Body height (cm)			165.13±5.89
Type of stroke	Hemorrhage	8(53)	
	Infarction	7(47)	
Side affected	Right	9(60)	
	Left	6(40)	
Duration of stroke (mos)	1-12	10(67)	8.46±8.87
	Above 12	5(33)	
MBI score	Severe	5(34)	64.26±23.58
	Moderate	2(13)	
	Mild	8(53)	
Spasticity	G0-2	15	
Sensory function	No impairment	5(33)	
	Impairment	10(67)	

85Mean±SD, Mean±Standard Deviation; MBI, Modified Barthel Index

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## 87 2. Study method

88 Wireless surface electromyography (Aurion, Italy) was used to measure the onset times of muscle activation of  
89four muscles that act importantly in the sit to stand movements<sup>10</sup>, that is, the TA, SOL, RF, and BF (Fig 1-A). A  
90force platform from Kistler Co. was used to obtain kinetic data from movements occurred in sitting postures.  
91The data, obtained through the surface EMG and the force platform, were analyzed using a QTM system made  
92by Qualisys Co. EMG and force platform signals were synchronized using an analog-digital converter. (Fig 1-B,  
93C)

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96 Figure 1. Zero-Wire EMG(A), QTM system(B) and force platform(C)

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### 98) Study procedure

99 For the same study environments of the individual subjects considering their physical conditions, a  
 100 height-adjustable treatment bed without any armrest or backrest was used. Each subject placed two feet on the  
 101 force platform in parallel with each other at 10 cm apart based on the medial planes of the feet. As a start  
 102 posture, each subject maintains 10° flexion of the ankle and 100° flexion of the knee joint and placed the arms  
 103 on both sides. The subjects were instructed to stand up comfortably at the speed at ordinary times. The subjects  
 104 performed the sit to stand movement at the command 'Stand up' after a preparatory command to prepare. This  
 105 movement was repeated three times and the average value was obtained.<sup>10,11)</sup> After the baseline evaluation, the  
 106 subject took a rest for 10 minutes in a supine posture after the intervention, and reevaluation was performed in  
 107 the same method after the rest. (Figure 2). The start of the movement was defined as the section from the point  
 108 at which the vertical ground reaction force began to decrease to the point showed the minimum value. The  
 109 movement execution stage was defined as the section from the point at which the vertical ground reaction force  
 110 began to increase to the point reached the peak. The final stationary stage was defined to the point at when the  
 111 ground reaction force was stabilized. The stages from the start to the end of movement were normalized to  
 112 analyze the time of onset of muscle activation and ground reaction force data.<sup>10)</sup>

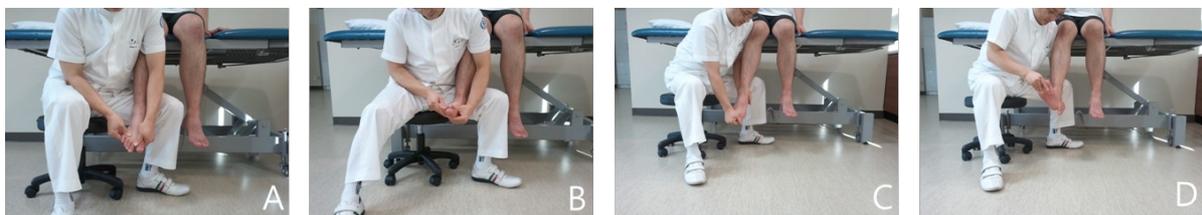
113



114 Figure 2. Data collection during sit to stand movement

### 1152) Therapeutic intervention

#### 116(1) Realignment of the feet and ankles and facilitation of muscle activation

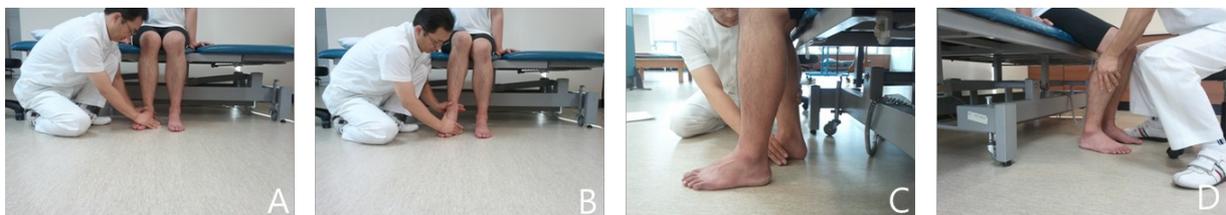


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1181 For selective recovery of the lengths of the TA and SOL and muscle activation, the treatment bed should be  
 119 high enough so that both feet are held away from the ground before beginning the treatment. As for the  
 120 position of the foot to be held, the medial side of the foot including the big toe should be maintained stable  
 121 with one hand and the lateral side of the foot including the little toe should be held with the other hand of  
 122 the physical therapist (A). Sensory input should be provided between the abductor digiti minimi muscle and  
 123 the metatarsal bone through movements by changes in the intrinsic muscles between the sole and toes (B).<sup>8)</sup>  
 1242 The therapist should hold the fore part of the foot with one hand and the heel of the foot with the other hand  
 125 (C). The therapist should make the subject lower the heel slowly toward the floor as the heel pushes down  
 126 with the eccentric contraction of the SOL in the sit-to-stand movement (D).

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#### 128(2) Movement composition and execution facilitation method



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1301 The treatment bed should be lowered so that the feet can be touched on the ground before starting the  
 131 treatment. The subject should sit on the treatment bed with the feet and heels placed flat on the floor. The  
 132 therapist should support the heel of a foot with one hand and hold the top of the foot with the other hand to

133 facilitate the eccentric contraction of the SOL (A). The therapist should reinforce the interaction between  
 134 the TA and SOL through repetitive lifting and putting down of the heel (B).  
 1352 The therapist should hold the top of the foot close to the ankle with one hand and hold the lower part of the  
 136 tibia with the other hand. The therapist should softly press the foot backward so that the heel gradually  
 137 comes into tight contact with the floor. The therapist should move the foot backward with the heel  
 138 maintained in contact with the floor to lengthen the SOL (C). The therapist should instruct the subject to  
 139 keep the heel in tight contact with the floor to achieve the maximum contact. The therapist should sit in  
 140 front of the subject and place both hands on the knee extensors of the subject and press the knee extensors  
 141 down toward the heel (D).  
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1444) **Statistical analysis**

145 The means and standard deviations of the general characteristics of the subjects were obtained through  
 146descriptive statistics and the normality of the measured data was identified through a 1-sample  
 147Kolmogorov-Smirnov analysis. Paired t-tests were conducted to analyze changes in muscle activities through  
 148the therapeutic intervention and the one-way repeated ANOVA was used to analyze the order of muscle  
 149activation of the lower limbs before and after the treatment. The data were statistically processed using the SPSS  
 15012.0 program and the significance level was set to 0.05.

151 **III. Results**

1521. Changes in onset time of muscle activity

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 154 Regarding changes in both lower limb muscle activity onset times between before and after the treatment,  
 155among paretic side lower limb muscles, the TA showed significantly earlier onset after the treatment (25.27%),  
 156while the SOL showed delayed onset (22.86%) ( $p < 0.05$ ). The RF and BF showed delays in average values, but  
 157the differences were not statistically significant ( $p > 0.05$ ). Non-paretic lower limb muscles showed no significant  
 158change in onset time of muscle activity between pre- and post-treatment ( $p > 0.05$ ).

159Table 2. Variations of muscle onset time between pre- and post-treatment during the sit to stand movement  
 160( $n=15$ )

161(Value= Mean±SD)

Muscle	Paretic Side			Non-paretic Side		
	Pre	Post	<i>t</i>	Pre	Post	<i>t</i>
TA	49.26±20.64	36.81±14.14	3.01*	16.08±12.01	21.02± 8.77	-1.50
SOL	44.00± 5.95	54.06±17.64	-2.28*	49.82±13.59	51.79±18.07	-0.44
RF	30.39± 8.06	35.93±10.80	-1.53	25.36± 7.44	29.45± 8.18	-1.20
BF	35.29±13.67	36.59±14.54	-0.34	38.72±10.31	38.67±12.11	0.01

162\*: $p < .05$ ; Mean±SD, Mean±Standard deviation

163Note: TA, tibialis anterior muscle; SOL, soleus muscle; RF, rectus femoris muscle; BF, biceps femoris muscle

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1652. Changes in recruitment order of lower limb muscle activity

166 Recruitment orders were significant between the paretic side TA and RF and between the paretic side SOL and  
167RF before the treatment ( $p < 0.05$ ). Therefore, regarding the recruitment order of paretic lower limb muscle  
168activity before the treatment, the RF was activated first followed by the SOL and TA in order of precedence. The  
169recruitment order of paretic muscle activity in the post-treatment was significant and the TA was activated first  
170followed by the SOL, the RF, and the BF ( $p < 0.05$ ). The recruitment order of non-paretic lower limb muscle  
171activity was statistically significant ( $p < 0.05$ ). The recruitment order between pre- and post-treatment were the  
172same and the TA was activated first followed by the RF, the BF, and the SOL

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Figure 3. Variations of the recruitment order of muscle activation between pre- and post-treatment during the sittostand movement.

#### IV. Discussion

174 Clinically, patients with neurological damage have a reduced ability to maintain a sitting posture without any  
175backrest or to shift their weight in sitting postures. Therefore, they become to use compensatory postural

176strategies. <sup>8)</sup> The use of compensatory postural strategies means making excessive movement patterns with  
177biased recruitment, that is, one-sided muscle contraction. According to Sahrman (1992), shortened muscles are  
178more easily mobilized than extended contralateral antagonists, resulting in increases in the muscle tension of the  
179shortened muscles. <sup>12)</sup> In a study conducted by Cheng et al. (2004), the TA showed no activation or very low  
180activity during the sit to stand movements of hemiplegic patients which is consistent with the results of another  
181study indicating that hemiplegic patients showed asymmetric muscle activities of two lower limbs due to  
182excessive non-paretic side TA activation. <sup>10)</sup>

183 Raine et al. (2009) reported that the shortening and weakening of the SOL as an antagonistic muscle restricted  
184dorsi-flexion resulted in excessive medial elevation. <sup>8)</sup> The ineffective ankle alignment as such was attributed to  
185the compensatory activation of the TA. The abovementioned results related to the TA are considered attributable  
186to the treatment method in this study that facilitated the activation of the lateral elevator while maintaining the  
187length of the muscle in order not to induce excessive contraction of the TA, which is a medial elevator, thereby  
188reinforcing the antagonist which is the SOL. The pre-treatment muscle activity of the paretic side SOL was 73%  
189of that of the non-paretic side SOL, but decreased to 43% after the treatment. This can be considered attributable  
190to the fact that the action potential of the uncontrolled excessive activity of the TA before the treatment was  
191reduced through the recovery of the antagonistic muscle and the synergist after the treatment.

192 Regarding lower limb muscle activity onset time, the paretic side TA showed significantly earlier activity onset  
193time after the treatment compared to that before the treatment while the SOL showed delayed activity onset time  
194with a statistically significant difference. In the study conducted by Goulart and Valls-Sole (2001) on the  
195reciprocal inhibition of the TA and SOL in healthy persons, it was reported that information coming in from the  
196periphery was suppressed for a certain time for the antagonism of the soleus muscle during the sit-to-stand  
197movements and that the foregoing facilitated the activation of the TA, which is an agonist, through ankle  
198stability. <sup>5)</sup> In normal study subjects, the SOL is activated last among lower limb muscles and is activated after  
199the hip taking off during the sit-to-stand movement. <sup>5)6)</sup>

200 The result indicating that the SOL is activated earlier than the TA before the treatment was similar to the  
201results of the study conducted by Camargos et al. (2009), in which changes in the muscle activity and muscle  
202activity onset time according to changes in foot positions during hemiplegic patients' sit-to-stand movement  
203were analyzed, indicating that the SOL was activated before hip taking off regardless of foot positions. <sup>13)</sup>  
204Therefore, the significant differences in the TA and SOL activity onset times can be said to mean the  
205enhancement of the ability for reciprocal inhibition of dorsi-flexion and plantar flexion.

206 The recruitment order of muscle activation was statistically significant on both paretic and non-paretic lower  
207limb muscles. The recruitment order of muscle activation in pre-treatment was the RF, SOL, and TA. Therefore,  
208the SOL was activated earlier than the TA. The order of muscle activation as such becomes to disturb the

209movement of the center of gravity toward the front of the feet and disturb the sufficient extension of the hip joint  
210with early extension of the knee joint.<sup>8)</sup> After the treatment, the TA and SOL showed significant differences in  
211the recruitment order. The delayed activation of the SOL can be regarded to contribute to postural control at  
212vertical acceleration and deceleration time, that is, during the extension of the lower limb joints with the  
213enhancement of the antagonism of the plantar flexor.

214Given the abovementioned results, the fact that the treatment method that facilitates the reciprocal inhibition of  
215dorsi and plantar flexion of the ankle in patients with hemiplegia due to stroke improves the muscle activity  
216patterns of lower limb muscles on both sides could be identified and it could be seen that such results would  
217affect improvement of the sit-to-stand movement efficiently.

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## V. Conclusion

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221Through the results of this study, it could be identified that the intervention used in this study reinforces the  
222muscle activity patterns of both side lower limb muscles and it could be concluded that such results would affect  
223stable postural control during the sit-to-stand movement. Therefore, we believe that the treatment would prevent  
224changes in the neurologic reciprocal inhibition of the feet and ankles and biomechanical alignments and can  
225provide therapeutic benefits to hemiplegic patients in need.

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## References

2281. Lomaglio MJ, Eng JJ. Muscle strength and weight-bearing symmetry relate to sit to stand performance in  
229individuals with stroke. *Gait Posture*. 2005;22:126-31.

2302. Mark MKY, Levin O, Mizrahi J, et al. Joint torques during sit-to-stand in healthy subjects and people with  
231parkinson's disease. *Clin Biomech*. 2003;18:197-206.

2323. Carr JH, Shepherd RB. *Neurological Rehabilitation: Optimizing Motor Performance*. Oxford,  
233Butterworth-Heinmann. 2003.

2344. Goulart F, Valls-Sole J. Patterned electromyographic activity in the sit to stand movement. *Clin Neurophysiol*.  
2351999;110(9):1634-40.

2365. Goulart F, Valls-Sole J. Reciprocal changes of excitability between tibialis anterior and soleus during the sit to  
237stand movement. *Exp Brain Res*. 2001;158(1):18-27.

2386. Khemlani MM, Carr JH, Crosbie WJ. Muscle synergies and joint linkages in sit to stand under two initial foot  
239positions. *Clin Biomech*. 1999;14:236-46.

2407. Gjelsvik B. The Bobath concept in adult neurology. Stuttgart: Thieme. 2008.
2418. Raine S, Meadows L, Lynch-Ellerington M. Bobath concept. Oxford: Wiley-Blackwell. 2009.
2429. Cheng PT, Liaw MY, Wong MK, et al. The sit to stand movement in stroke patients and its correlation with falling. Arch Phys Med Rehabil, 1998;79:1043-6.
24410. Cheng PT, Chen CL, Wong MK, et al. Leg muscle activation patterns of sit to stand movements in stroke patients. Am J Phys Med Rehabil, 2004;82:42-7.
24611. Chou SW, Wong AMK, Leong CP, et al. Postural control during sit to stand and gait in stroke patients. Am J Phys Med Rehabil, 2003;82(1):42-7.
24812. Sahrman SA. Diagnosis and treatment of movement impairment syndromes. St. Louis, MO: Mosby. 2002.
24913. Camargos AC, Rodrigues-de-Paula-Goulart F, Teixeira-Salmela LF. The effects of foot position on the performance of the sit to stand movement with chronic stroke subjects. Arch Phys Med Rehabil, 2009;90:314-9.