

1 **Effects of Knee Extensor Muscle Fatigue on Gait Ability in Patients with**  
2 **Chronic Stroke.**

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9 **Abstract**

10 **Purpose** The purpose of this study were how to effect gait ability to chronic stroke patients who were taken  
11 knee extensor fatigue and were understood and offered the way of effective therapy to stroke patients in  
12 clinical trials. **Methods** The subject of this study were 16 chronic stroke patients (9 males, 8 females), and 16  
13 healthy volunteers( 5 males, 11 females) and their average duration of symptoms were 4.9 years at S hospital  
14 in Sungnam city. Stroke patient group age was 55.67, health group age was 59.13. Both stroke patient group  
15 and healthy group were measured using maximal voluntary isometric contraction(MVIC) for leading knee  
16 extensor fatigue. Isometric contraction was maintained for 10 seconds. And each isometric contraction was  
17 taken rest 5 seconds one time for inducing muscle fatigue. The end point was measured decreasing 3 times  
18 continuously which the value of MVIC was less 50%, and then the number of knee extensor to induce muscle  
19 fatigue gait ability were measured after taking knee extensor fatigue. **Results** There was a significantly  
20 differences that Stroke patients group was shown that the paretic side knee extensor fatigue was represented  
21 faster than health control group ( $p<0.05$ ). The gait ability were represented drastically decreased knee extensor  
22 fatigue in Stroke patients group's non paretic side. **Conclusion** Therefore, localized muscle fatigue by gaiting  
23 abnormal gait pattern to chronic stroke patient was affected the disability of gait capacity. And abnormal gait  
24 pattern was brought secondly increasing number of falling down. These factors were considered to stroke  
25 rehabilitation.

26 **Key Words** Knee extensor, Paretic, Nonparetic, Muscle fatigue, Stroke

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## I. INTRODUCTION

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36 Stroke is an acquired disease that causes considerable disability for more than a few months despite active rehabilitation. It is  
37 a classic cause of paraplegia. Hemiplegic patients have damaged posture control because of strokes. Motion, sensory, and high  
38 central cognitive defects due to a stroke cause loss of function. Most patients have limitations throughout their community  
39 activities due to independent walking disorders, causing secondary physical disability problems.

40 Secondary physical disorders include asymmetrical posture and abnormal walking patterns, which can also lead to pain, muscle  
41 weakness, stiffness, sensory information disturbance, and abnormal strain.

42 The ideal walk for normal people should be a symmetrical weight, bearing on the body with a continuous, repetitive,  
43 symmetrical motion, and a symmetrical walking cycle and walking speed. The cycle, velocity, step length, and stride length are  
44 important for stroke patients. However, the characteristic walk of a hemiparetic patient due to stroke shows asymmetric weight  
45 load, slow walking cycles, and walking speed. These asymmetrical support times affect walking speed and walking cycles.<sup>1)</sup>

46 The walking speed of a stroke patient is related to the differences in step length, step width, swing phase, and stance phase. As a  
47 result, walking in hemiplegic patients results in a decrease in the time of the paralysis side and an increase in the time of the  
48 non-paretic side swing time period.<sup>2)</sup> In addition to this, it interferes with the selective movement control ability in gait and  
49 causes a decrease in voluntary muscle activation and muscle recruitment ability. Therefore, most hemiplegic patients support  
50 the weight on the paretic side because of unstable standing and abnormal gait.<sup>1-2)</sup> Therefore, according to the asymmetric gait  
51 pattern, patients have a longer time in the stance phase of the paretic side, resulting in inefficient energy consumption and slow  
52 walking. It is reported that muscle fatigue and tension are reduced due to excessive weight support of the non-paretic side.<sup>2-4)</sup>

53 Muscular fatigue is the gradual decrease in the ability of the acute muscles to exert force because of exercise; there is decrease  
54 in muscle strength and mental function as well as lack of exercise.<sup>5)</sup> Skeletal muscle fibers are classified according to the rate of  
55 contraction reaction to electrical stimulation. They are classified into slow twitch oxidative fibers (I type) and fast twitch  
56 glycolytic fibers (IIb type). Fast twitch glycolytic fibers have a fast muscle contraction rate and tire easily.<sup>6-7)</sup> Muscle fatigue is  
57 known to reduce motor control capacity.<sup>8)</sup> Reduced motor control capacity may be caused by decreased ability to maintain  
58 muscle strength due to fatigue. As a result, muscle fatigue causes a decrease in proprioceptive sensation.<sup>9-10)</sup> Also, Muscle  
59 fatigue cause impair the proprioceptive and kinesthetic properties of joints by increasing the threshold of muscle spindle  
60 discharge. Recent studies have reported that vestibular information, visual information, and neck somatosensory are also  
61 closely related to postural control. As a result, incorrect somatosensory information due to muscle fatigue causes fluctuations in  
62 postural control.<sup>9-11)</sup> Studies on muscle fibers and position senses were previously conducted.<sup>12)</sup>

63 Parijat and Lockhart(2008) made a study on young subjects and reported that localized muscle fatigue of the proximal knee  
64 extensor negatively affected the position of the knee joint, and it caused a change in the gait pattern by interfering with the

65 movement of the joint.<sup>13)</sup>

66 There are many studies that have focused on gender, age, and focal localized muscle fatigue for normal subjects, but there are  
67 a lack of studies on the effects of fatigue on the paretic and the non-paretic side of the knee extensor muscles in stroke patients.  
68 The purpose of this study was to investigate the effect of the knee extensor on the gait pattern in hemiplegic patients and to  
69 provide understanding and improved therapeutic efficacy of stroke patients in clinical practice. This study followed the ethical  
70 principles of the Declaration of Helsinki, and all patients gave written informed consents before participating in this study. We  
71 approved the approval of the Institutional Life Research Ethics Committee of Yongin University  
72 (2-1040966-AB-N-01-20-1611-HSR061-8).

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## II. Material and Methods

### 74 1. Subjects

75 In this study, 16 stroke patients and 16 control subjects participated in S hospital in Seongnam City. The study subjects were  
76 32 persons who agreed to participate in this study.

77 The inclusion criteria for the stroke patient group were as follows: (1) Sub acute stage patients (6 months to 1 year after  
78 onset), (2) No damage to position sense, (3) No cognitive deficits (MMSE-K score of  $\geq 24$  points), (4) No joint deformity and  
79 orthopedic disease, (5) Understanding and informed consent to this study

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### 81 2. Procedure

82 This study was performed on the stroke paretic side, the non-paretic side, the normal control group dominant side, and the  
83 normal control group non-dominant side. The order was randomly assigned.

84 This study was designed to evaluate the maximum voluntary isometric contraction of the knee extensor using the Primus  
85 dynamometer (Baltimore Therapeutic Equipment, USA, 2006). The subjects were seated on a Primus dynamometer with  $10^\circ$   
86 backward sitting, sitting with the hip joint at  $80^\circ$  and the knee joint at  $60^\circ$  ( $0^\circ =$  full extension) in an upright position. During  
87 maximal voluntary isometric contraction, the upper body and thigh were immobilized in a velcro. The knee joint was aligned  
88 with the axis to perform knee flexion at  $60^\circ$ .<sup>14)</sup> The maximum isometric contraction force measurement was performed by  
89 isometric contractions of the knee extensor four times for five seconds to determine the maximum voluntary contraction force  
90 and to provide auditory and visual feedback for the subject's continued efforts in the measurement. In order to eliminate the  
91 effect of muscle fatigue during the measurement, a 2-minute rest period was given for each contraction.<sup>15)</sup> The measured values  
92 were recorded using Newton-meters. When the maximum value exceeded 10% in four measurements, the remaining values  
93 were excluded and the average value was selected as the maximum voluntary isometric contraction value.

### 94 3. Muscle fatigue measurement

95 The subjects were in the same posture as for the maximum voluntary isometric contraction force measurements. The isometric  
96 contraction of the knee extensor was continuously sustained for ten seconds, and the maximum isometric contraction was  
97 followed by a 5-second rest period to induce muscle fatigue. The end point of the isometric contraction was until the maximum  
98 isometric contraction decreased to less than 50% at three consecutive times. Gait ability tests were done immediately after  
99 muscle fatigue set in [14-15]. The subjects used a wheelchair to minimize the recovery of the knee extensor after muscle fatigue  
100 and to ensure the safety of the subject.<sup>16)</sup>

### 101 4. Gait ability measurement

102 GaitRite (CIR system Inc, USA, 2008) was used for the gait ability test. After the patient walked in front of the gait board, he

103 was guided to walk at the most comfortable walking speed by a verbal sign, "Walk comfortably", on the gait board. The  
104 GaitRite is equipped with a sensor on an electronic gait plate with a length of 3 m, a width of 61 cm, and a height of 0.6 cm.  
105 The time and spatial variables during walking on the mat was evaluated using a computer.  
106 Information on collected temporal and spatial variables was processed with GAITRite GOLD, Version 3.2b (CIR system Inc,  
107 USA, 2007) software.

#### 1085. Data analysis

109 In this study, SPSS 20.0 program for Windows was used. Differences between pre- and post-test were analyzed by paired t-test  
110 and one-way ANOVA was performed to compare differences between the groups. When there were significant differences,  
111 Tukey's Honest Significant Difference test was performed. Statistically significant levels were = to 0.05.

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### III. Results

114 The subjects were 16 chronic stroke patients (9 males and 8 females) and 16 normal peoples (5 males and 11 females). In  
115 the stroke patients, four was tested on the right side and 12 on the left side. The mean duration of the disease was 4.9 years. The  
116 mean age was 55.67 years in the patient group and 59.13 years in the control group. According to this study, the score of the  
117 Functional Ambulation Profile was reduced from the initial assessment of 74.31 points to 69.69 points after fatigue on the  
118 paretic side of the patient's knee. For the non-paretic side fatigue, it decreased to 69.75 points ( $p < .01$ ) (Table 1). In the normal  
119 control group, the initial evaluation of 96.75 points was reduced to 94.56 points after non-dominant side fatigue. In addition to  
120 this, after fatigue on the dominant side, the initial evaluation of 94.81 points decreased by 1.94 points. These results showed  
121 statistically significant differences in the normal controls ( $p < .01$ ) (Table 2). After non-paretic side knee extensor fatigue, the  
122 velocities decreased significantly from 68.10 to 62.29, indicating a significant difference ( $p < .05$ ). There was no significant  
123 difference after paretic side knee extensor fatigue (Table 1). In the normal control group, there was no significant difference  
124 between before and after fatigue.

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### IV. Discussion

127 The purpose of this study was to investigate the effects of knee extensor fatigue on the gait ability of chronic stroke patients,  
128 particularly hemiplegic patients. Fatigue of the knee extensor was performed using a Primus dynamometer (Baltimore  
129 Therapeutic Equipment, USA, 2006), which is a frequently used induction method for voluntary fatigue induction tests.<sup>17)</sup>  
130 Stroke patients reported muscle weakness on the contralateral limb due to cerebral cortical damage. The maximum voluntary  
131 isometric contraction of the knee extensor significantly decreased the torque value in both paralyzed and non-paralyzed subjects  
132 compared to normal subjects.<sup>14, 18)</sup>

133 This suggests that voluntary activation failure of the knee extensor occurs in both sides, confirming the theory that neurological  
134 damage directly affects skeletal muscle.<sup>14)</sup> Voluntary activation failure may be due to a motor unit recruitment failure, or it may  
135 be due to a decrease in the rate of firing of the active units.<sup>16)</sup> The skeletal muscles of hemiplegic patients due to stroke have  
136 slow contraction and relaxation and the paralyzed limb still moved slower than normal after 6 months. In addition to this, stroke  
137 patients have decreased oxidative metabolism, resulting in decreased exercise capacity and muscle endurance. Resistance to  
138 muscle fatigue weakens functional mobility.<sup>14)</sup> This is because stroke patients take two to three times longer than normal to  
139 produce strength when walking.<sup>4)</sup> Stroke patients also cannot maintain effective walking speeds on their own. As a result of this,  
140 high energy consumption and poor muscle endurance affect functional performance ability. Dean et al. (2001) described  
141 endurance shortages in 14 patients with chronic stroke, which suggests that stroke patients with similar body characteristics can

142 walk only about 50% of the normal walking distance. In stroke patients, 80% of body weight was supported on the non-paretic  
143 side leg, and when standing, the paretic side lower limb supported less than 50% of the total body weight.<sup>3)</sup> The gait  
144 characteristics of these stroke patients were twice as long as in the normal control group in the double support phase, which  
145 required more time for the patients to maintain balance. Thus, the short stance phase time of the paralyzed leg during gait is  
146 because it causes muscle fatigue of the non-paretic lower limb muscle. These factors can lead to more balance and walking  
147 disorders. Therefore, asymmetrical gait characteristics of stroke patients lead to overuse of the non-paretic side lower limb; this  
148 easily induces muscle fatigue. In this study, we found that the non-paretic knee extensor muscle fatigue showed a decrease in  
149 walking ability as compared to the paretic side knee extensor muscle fatigue (Table 1).

150 According to Dean Order, the walking support time in the single support phase of the patients with hemiplegia decreased in  
151 both the paretic side and non-paretic side (biceps femoris and femoral rectus,  $p < .05$ ) as compared with the normal control  
152 group. Co-contraction time of biceps femoris and rectus femoris increased significantly in stroke patients ( $p < .05$ ).<sup>19)</sup> As a result  
153 of this, stroke patients used more double support time than single support time during walking and the propulsive forces on the  
154 paretic side and the non-paretic side reduction caused abnormal gait.<sup>19)</sup> The results of this study showed that hemiplegic patients  
155 had greater gait disturbance on the non-paretic knee extensor muscle fatigue because of the longer time required for double  
156 support phase as compared to normal patients (Table 1). The purpose of this study was to investigate the effect of artificial local  
157 knee extensor fatigue on walking in hemiplegic patients. This is not a natural fatigue of everyday life, so it is difficult to  
158 generalize research results. The results of this study were limited because all the chronic stroke patients were not grouped into  
159 recovery stages (acute, subacute, and chronic). In addition, this study was limited to interpretation of patients with evthing  
160 stroke patients because it was performed in patients with chronic stroke without being grouped into recovery stages (acute,  
161 subacute, and chronic).

162 After having a stroke, a patient has abnormal posture control and gait disturbances due to impaired motor and sensory  
163 function. This leads to increased postural sway, asymmetrical weight support, impaired ability to move weight, and persistent  
164 abnormal gait patterns. Gait training leads to localized muscle fatigue because of the non-paretic side. Especially, Fatigue at the  
165 knee extensor led to postural control impairment in the frontal plane. Therefore, localized muscle fatigue on the non-paretic  
166 side may result in decreased balance ability and gait disturbance of the patient, and abnormal gait pattern may lead to secondary  
167 risk of falls. In the future, it will be necessary to consider these factors in stroke gait learning.

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	Paretic		Paretic Post-fatigue				Non paretic		Non paretic			
	Pre-fatigue						Pre-fatigue		Post-fatigue			
	M±SE		M±SE		t	p	M±SE		M±SE		t	p
Velocity (cm/sec)	68.10	± 6.9	63.44	± 6.34	1.721	.106	68.10	± 6.9	62.29	± 6.57	2.861	.012*
Cadence (step/min)	92.48	± 4.05	91.83	± 3.81	.386	.705	92.48	± 4.05	89.69	± 2.92	2.130	.050
Functional Amb. (score)	74.31	± 4.59	69.69	± 4.42	2.432	.028*	74.31	± 4.59	69.75	± 4.61	3.047	.008**

215 Table 1. The gait ability in patients before and after fatigue knee extensor

217 Table 2. The gait ability in healthy control before and after fatigue knee extensor

	Pre-fatigue		Non dominant				Pre-fatigue		Dominant			
			Post-fatigue						Post-fatigue			
	M±SE		M±SE		t	p	M±SE		M±SE		t	p
Velocity (cm/sec)	107.34	± 3.01	101.84	± 3.18	1.832	0.087	107.34	± 3.01	106.93	± 4.08	0.15	0.883
Cadence (step/min)	113.46	± 2.45	110.66	± 2.92	1.26	0.227	113.46	± 2.45	112.63	± 2.29	0.428	0.675
Functiona l Amb. (score)	96.75	± 0.74	94.56	± 0.63	4.68	0.000**	96.75	± 0.74	94.81	± 0.68	7.766	0.000**

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