한국전문물리치료학회지 제 14권 제 4호
PTK Vol. 14 No. 4 2007.

Characteristics of Initiation and Termination of Tibialis Anterior Contraction in Adults With Hemiplegia: A Preliminary Study

Yi-jung Chung, Ph.D., P.T
Dept. of Physical Therapy, College of Health Welfare, Sahmyook University
Jung-ah Lee, M.Sc., P.T.
Dept. of Rehabilitation Therapy, The Graduate School, Yonsei University
Won-seob Shin, M.Sc., P.T.
Seung-heon An, M.Sc., P.T.
Eun-woo Lee, B.H.Sc., P.T.
Kyoung-sim Jung, B.H.Sc., P.T.
Dept. of Physical Therapy, The Graduate School, Sahmyook University

Abstract

The purpose of this study was to investigate the relationship between delays in initiation and termination of tibialis anterior contraction through surface electromyographic (sEMG) analysis in adults with hemiplegia and healthy subjects and clinical assessment of lower-limb mobility. EMG activity of 6 long-term survivors of stroke and 5 healthy subjects was recorded during maximal isometric ankle dorsiflexion in 3 seconds beeper signals. It must be done as fast and forcefully as possible. Lower limb mobility was assessed with Modified Emory Functional Ambulation Profile (mEFAP). Delay in initiation and termination of muscle contraction was significantly prolonged in the affected lower limb relative to the unaffected limb. Termination of muscle contraction in the hemiplegic lower limb was significantly delayed than the initiation on the affected sides. Delay in initiation and termination of muscle contraction correlated significantly with a few range of mEFAP. Abnormally delayed initiation and termination of muscle contraction may contribute to hemiparetic lower limb mobility in hemiparetic patients. Consequently, this study showed that abnormal delay of initiation and termination of muscle contraction may contribute to hemiparetic lower limb mobility in adults with hemiplegia. Further studies are needed to demonstrate a treatment effect.

Key Words: Stroke; Surface electromyography; Tibialis anterior.

Introduction

The mortality rate for strokes in 2005 was second to neoplasms at 64.3 per 100,000 people and 31,297 people were death. The prevalence of strokes tends to increase with age and increases rapidly over 50 (Ministry of Health & Welfare, 2007).

In over 50%, physical or functional disabilities remain after the stroke, of these 48% suffer mild paralysis, 22% gait disability, 12~18% language disability, 24~53% need help to execute daily chores, 32% suffer from depression (Sacco et al, 1997). There will be more needed studies about strokes because it occurs more often to the elderly as well as increases in life expectancy.

Bobath (1990) reported that ataxia was one of the neurological symptoms from strokes. Ataxia is the state of decreased coordination among agonist, antago-

Corresponding author: Won-seob Shin *suby96@naver.com*

nist, synergist and fixator, and is a significant movement disorder showing loss of specific sensations, the involuntary movement. Compared to muscles controlled from normal innervations, because of upper motor neuron lesions, spasticity would appear along with weakness and muscular stiffness arising from neuromuscular system changes, motor unit decrease, and nerve reinnervation, and decrease of firing rate (Fridén and Lieber, 2003; Hara et al, 2000). It is important to treat the functional movement disabilities from the changes of the muscle itself and the damages of movement-order transmitting systems after a stroke (Chae et al, 2002). According to previous stroke mobility studies, functional disabilities ranging from muscle weakness to loss of quickness outnumbered those from spasticity after brain injury (Hammond et al, 1988; Newham and Hasio, 2000; O'Dwyer et al, 1996).

With EMG studies, it was proven that there were initiation and termination delays from muscular contractions on the affected upper limb and the differences between the affected and the unaffected arose from recruitment pattern changes and a decrease of fire ratio (Chae et al, 2002; Hammond et al, 1988). However, comparisons for upper limb functional movement evaluations still lack classifying the movement injuries and quantifying the effect of treatments because the lack of studies for quickness of lower limbs.

According to the recent study of Marigold et al (2004), falling down and instability are caused from the initiation delay on the affected side having an effect on the ankle position. Chae et al (2006) study also supported that the reaction time for the tibialis anterior contraction in the lower limb hemiparesis, there appeared to be initiation and termination delays on the affected side.

Therefore, the purpose of this study was to confirm the findings of previous studies. This study was firstly intended to evaluate the initiation and termination delays of muscle contractions from the affected, the affected both for hemiplegia patients, and normal control. Secondly, to assess the correlation between the characteristics of muscle activity from damage and the evaluation of movement deficit.

Methods

Subjects

Six stable patients with a history of unilateral CVA (cerebral vascular accident) 76 to 375 days previously in the National Rehabilitation Center in Seoul, and five healthy control subjects with a similar balance of age and sex were tested. Inclusion criteria included 1) an interval of at least 2 months from stroke onset; 2) unilateral lesion; 3) manual muscle testing is poor or above in tibialis anterior muscle; 4) ability to follow three second maximal isometric contraction command. Patients who having the following conditions were excluded: those who had experienced hemiplegia on the opposite hemisphere, had other neurological problems, and had hemi-neglect, ipsilateral homonymous hemianopsia, and loss of sensation. Control groups were excluded if they reported previous injury or current orthopedic problems in their body. Characteristics of the subjects are summarized in Table 1.

Instrumentations

The surface electromyography (sEMG) signal was detected with an active parallel-bar electrode (bar size: 1 ㎜ by 10 ㎜, located 10 ㎜ apart differential electrode[1]). The electrodes were placed on the tibialis anterior muscle belly (Cram et al, 1998) (Figure 1). The EMG signals were digitally band-pass filtered at 20~450 ㎐ and notch filtered at 60 ㎐ to reduce noise. A sampling frequency of 1000 ㎐ was used. After collection, the data were transferred to an personal computer for data reduction. The Acqknowledge 3.72 program (Biopac systems Inc., CA, U.S.A.) was employed to set up the required parameters and to store the EMG signal as computer files.

Procedures

We used EMG to measure times of initiation and

1) Delsys Inc., Boston, MA, U.S.A.

Table 1. Clinical characteristics of patients (N=6)

Patients	Age (yrs)	Gender	Time since injury (days)	Plegic side	FMA[a]	MAS[b]	MMSE-K[c]	Diagnosis
1	62	Female	375	Lt	17	0/0	26	BI[d]
2	67	Male	204	Lt	22	0/2	28	MI[e]
3	34	Male	76	Rt	34	0/0	24	BH[f]
4	50	Male	146	Lt	27	0/0	30	BI
5	33	Male	101	Lt	25	0/1	30	TH[g]
6	46	Male	81	Rt	26	0/0	30	BH
Mean	48.7		163.8		25.2		28.0	

[a]FMA: Fugl-Meyer motor assessment.
[b]MAS: Modified Ashworth scale of ankle dorsiflexion.
[c]MMSE-K: Mini Mental State Examination-Korean.
[d]BI: basal ganglia infarction.
[e]MI: middle cerebral artery infarction.
[f]BH: basal ganglia hemorrhage.
[g]TH: thalamic hemorrhage.

termination in isometric contraction of tibialis anterior muscle. Subjects were seated on the N-K exercise

Figure 1. The placement of the electrodes and posture of the measurement.

unit with seat back tilted to 75° knee flexed to 30° and ankle plantarflexed to 30°. We elected not to force the neutral ankle position, in order to minimize baseline electromyographic activity in subjects with hypertonia of the gastrocnemius muscle. Subjects were to respond to beeps to dorsiflex against the ankle apparatus and to relax the muscle as beeps were removed as fast as possible. 3-second contraction trials were measured three times and done for both stroke involved side and the opposite. The trials were enforced with the break time randomly for subjects not to expect the initiation time, and subjects and tested side were randomized during the trials. Delay in recruitment of the EMG signal was defined as the time interval between onset of the audible beep and onset of the EMG signal. The parameters evaluated low pass software filter (50 Hz) and the other parameters evaluated were the number of samples assessed in the sliding window (25 ㎳) and the magnitude of the deviation from the baseline required to indicated the threshold (3 SD). Processing was done using MatLab′ signal processing toolkit[2)].

Lower-Limb Motor Impairment and Mobility Assessment

The mEFAP was used to evaluate the executive capability for the specific tasks on the hemiparetic limb. The mEFAP is intended to measure the walking time in 5 common environments with or without using assistance. There are 5 specific tasks: (1) a 5 m walking on a hard floor (2) a 5 m walking on the carpet (3) standing up from a chair, a 3 m walking, and back to the seat, (4) passing by the standardized

2) Math Works Inc., MA, U.S.A.

obstacle, and (5) walking up and down the 5 stairs. Subjects can use other assistant devices except ankle foot orthosis (AFO). All procedures were performed under supervision. The adjusted score with the assistant devices was used for the study. the reliability and validity of the mEFAP were evaluated with the proper methods (Baer and Wolf, 2001).

Statistical Analysis

Wilcoxon signed-rank test was used to examine the differences in delay of initiation and termination of electromyographic signal between the affected and unaffected sides. Mann-Whitney test was used to examine the differences between control and each side. To assess the relationship between electromyographic timing parameters and clinical measures, delays in initiation and termination of the affected side were normalized with respect to the unaffected side. Specifically, normalized delay in the affected side was defined as: (delay in the affected limb-delay in the unaffected limb)/delay in the unaffected limb (Chae et al, 2006). The relationship between the electromyographic timing parameters and mEFAP was assessed using the Spearman's rank order correlation coefficient. All statistical analyses were performed using SPSS 10.0 for windows. An p-value level of <.05 was used as the level of significance.

Results

Electromyographic Parameters

The mean and SD of delays in initiation and termination of electromyographic activity between the affected, unaffected, and control limbs are shown in Table 2. Initiation and termination of muscle contraction were significantly delayed on the more affected than unaffected and control sides (Table 2). Termination of tibialis anterior muscle was significantly delayed than the initiation on the affected sides (p<.05).

Electromyographic Parameters and Clinical Measures

The mEFAP scores is shown in Table 3. Spearman's rank order correlation coefficients relating delays and clinical measures are shown in Table 4. Delay in initiation of tibialis anterior muscle contraction correlated significantly with hard floor component and total score of the mEFAP. And delay in termination of tibialis anterior muscle contraction

Table 2. Comparison of delay in contraction onset and offset of the tibialis anterior muscles between side (Unit: sec)

Sides	Tibialis anterior		
	Initiation delay	Termination delay	p
Control	389.00±59.04[a]	441.60±78.01	.14
Unaffected	405.08±114.37	610.75±246.58	.17
Affected	463.44±100.98†‡	1226.50±703.94†‡	.03*

[a]Mean±SD.
† Significantly different from control sides with Mann-Whitney test (p<.05).
‡ Significantly different from unaffected sides with Wilcoxon signed-rank test (p<.05).
*Significant difference between initiation delay and termination delay (p<.05).

Table 3. mEFAP scores (Unit: sec)

Measures	Mean±SD
5 m walk: hard floor	13.36±9.27
5 m walk: carpet	16.15±11.65
Timed Up and Go	17.00±12.14
Obstacle course	32.08±30.36
5 stairs	18.24±14.25
Total	96.83±76.42

Table 4. Spearman's rank order correlation coefficient relating delays and clinical measures

Clinical measures	Initiation	Termination
5 m walk: hard floor	-.94**	.54
5 m walk: carpet	-.77	.83*
Timed Up and Go	-.77	.83*
Obstacle course	-.65	.37
5 stairs	-.71	.26
mEFAP total	-.89*	.66

*p<.05.
**p<.01.

correlated significantly with carpet and Timed Up and Go (TUG) components.

Discussion

Normal motor process is ordered by motor strategy of the primary motor cortex and regulated by the posterior parietal cortex and premotor area (Ghez, 1991). However, muscles ordered and regulated by the nervous system often are not functional due to the lowering of higher cortex intentions, malfunction in transmitting nervous signals, muscular fiber injury which directly perform, or other peripheral dysfunction (Gandevia et al, 1995). Especially for stroke patients with EMG, the stroke-involved side has a more delayed time of initiation and termination during muscle activation (Chae et al, 2002; Chae et al, 2006; Chung et al, 2003a; Hammond et al, 1988; Smith et al, 1998), muscle weakness (Chae et al, 2002), and increase of fatigue (Hu et al, 2006).

Keele (1968) had measured the responding time of activation initiated by visual signals to study the regulatory process between the neurological center and lower motor processes prior to responding passively. This method was used to measure the response time of muscle to evaluate when blue circle appears on the monitor. However, in recent studies, the signal sound was used to differentiate initiation of the muscle activation and the termination time (Chae et al, 2002; Chae et al, 2006; Chung et al, 2003b; Hammond et al, 1988). Chae et al (2006) especially showed that there were no effects on the difference of time because there were delays of both 3-seconds and 5-seconds in studying the initiation and termination of tibialis anterior muscle.

Our study compared the impaired and non-impaired side, initiation time had a 58 ㎳ difference, termination time 616 ㎳. For stroke patients, problems caused by motor-neurological regulatory center injuries have an effect no only on the direct activation time on the impaired side but also a delay before initiating action (Smith et al, 1998). Chae et al (2006) explained that due to the difference of initiation delay of 53 ㎳ and termination of 337 ㎳, there were differences between movement order and regulation on the impaired side. Also like on the results, because of 205 ㎳ difference bewteen the delay of initiation time and the delay of termination time in the impaired side, termination had more delay time difference. Chae et al (2006), too, showed that there were more delay time difference in termination, which were 53 ㎳ and 337 ㎳.

For mechanisms explaining the delay in termination time, there are an increase of excitation on alpha motor neurons (Delwaide, 1993), the supraspinal inhibition of interneuron pool (Ghez, 1991), increase and decrease of Renshow's inhibition (Delwaide, 1993), decrease of corticospinal input which is only dependent on uninjured vestibulospinal, tectospinal pathway (Dewald and Beer, 2001; Dewald et al, 1999), and redistribution of cortical pathway to the spinal segmental circuit connecting to more unfocused descending inputs (Kamper and Rymer, 2001).

There were no significant differences between a normal person and the intact side of the subject. However ipsilateral pathways of uncrossed corticospinal tract initiates from the cortex, internal capsule, and the anterior side of the brain stem, the medial corticospinal tract extends only into the thoracic cord sending branches to synapse that control neck, shoulder, and trunk muscles (Lundy-Ekman, 2002). Because of the little effects of the not-impaired lower limb in this study, compared to the not-impaired side to normal, there might be no mechanism effect differences.

However, in selecting subjects, there were much smaller 96.83±76.42 ㎳ compared to mFFAP total time 303.0±307.1 ㎳ on Chae et al (2006) because of selecting good-conditioned subjects who were evaluated by mean of lower limb FMA was 25.2 of total 34. Due to good-conditioned subjects, there were little effects on the delay between initiation and termination, there were significant results on only a few tests. For further studies, other factors influencing the movement must be considered. In previous stud-

ies, not only was there a delay between initiation and termination of movements by the problem of neurological regulation and transmission, but there were the changes of muscle fibers on the hemiplegic side as the characteristic change of the muscle itself. The diameter of muscle fibers decreased after the subacute phase, the selective type II muscle fibers atrophy, and then muscle weakness occurred (Dietz et al, 1986; Scelsi et al, 1984).

Due to muscle paralysis and weakness, stroke patients cannot produce proper power to regulate their posture or to initiate and adjust movements. Within 6 months, the number of movement-unit lessened to 50% after the event, there was no effective strength because of firing rate reductions (Bobath, 1990; Kirker et al, 2000).

In this study, there were significant impaired lower limb delays from the malfunctioning of the supraspinal contral, not being controlled well of postural reflex from various downward information, and the change of the muscle itself from the stroke. Also compared to the unimpaired side, there are delays in postural control on the impaired side because of reduction of neurologic transmission speeds (Cruz-Martinez, 1983).

In the Marigold et al (2004) study, when subjects were asked to change to the forward platform translation from the erect position, the initiation delay of subjects falling tibialis anterior muscles were 23 ㎳ difference, compared 139.2±1.6 ㎳ to subjects not falling 116.9±19.6 ㎳. Forster and Young (1995) recorded that 73% patients fell after suffering a stroke. Disrupted timing of muscle activation also contributes to the movement problems in people with upper motor neuron syndrome. Initiation of movement is delayed, the rate of force development is slowed, muscle contraction time is prolonged, and the timing of activation of antagonists relative to agonists is disrupted in people with upper motor neuron syndrome (Sharmann and Norton, 1977; Young and Mayer, 1982). The lower the initiation difference through therapeutic intervention, the lower causes of falling would be.

Like above, studying each muscle for delay of initiation and termination, and investigation the correlation with movement can approach to the therapeutic destination for each muscle of a person. In this study, we studied the difference and correlation of delay times. In the future, there would be much needed studies of therapeutic intervention practically reducing the delay of initiation and termination and of improving the movement and the quality of life for the stroke survivors.

Conclusion

This study has shown that there were differences of initiation and termination delays on tibialis anterior muscle contraction between the affected and unaffected. The clinical relation of tibialis anterior muscle delays are needed to study for therapeutic intervention which reduced the delay of tibialis anterior muscle contraction that can induce the functional recovery correlated motor control.

References

Baer HR, Wolf SL. Modified emory functional ambulation profile: An outcome measure for the rehabilitation of poststroke gait dysfunction. Stroke. 2001;32(4):973-979.

Bobath B. Adult Hemiplegia: Evaluation and treatment. 3rd ed. London, Heinemman Medical Books, 1990.

Chae J, Yang G, Park BK, et al. Muscle weakness and cocontraction in upper limb hemiparesis: Relationship to motor impairment and physical disability. Neurorehabil Neural Repair. 2002;16(3):241-248.

Chae J, Yang G, Park BK, et al. Delay in initiation and termination of muscle contraction, motor impairment, and physical disability in upper limb hemiparesis. Muscle Nerve. 2002;25(4):568-575.

Chae J, Quinn A, El-Hayek K, et al. Delay in initiation and termination of tibialis anterior contraction in lower-limb hemiparesis: Relationship

to lower-limb motor impairment and mobility. Arch Phys Med Rehabil. 2006;87(9):1230-1234.

Chung YJ, Cho SH, Kwon OY, et al. Initiation and termination of electromyographic activity in the early hemiparetic wrist. Journal of the Korean Academy of University Trained Physical Therapists. 2003a;10(4):95-103.

Chung YJ, Cho SH, Lee JH, et al. Reliability of the onset time determination during maximal isometric contraction in surface EMG. Journal of the Korean Academy of University Trained Physical Therapists. 2003b;10(1):51-62.

Cram JR, Kasman GS, Holtz J. Introduction to Surface Electromyography. Gaithersburg, Aspen, 1998.

Cruz-Martinez A. Electrophysiological study in hemiparetic patients. Electromyography, motor conduction velocity, and response to repetitive nerve stimulation. Electromyogr Clin Neurophysiol. 1983;23(1-2):139-148.

Delwaide PJ. Human reflex studies for understanding the motor system. Phys Med Rehabil Clin N Am. 1993;4:669-686.

Dewald JP, Beer RF. Abnormal joint torque patterns in the paretic upper limb of subjects with hemiparesis. Muscle Nerve. 2001;24(2):273-283.

Dewald JP, Beer RF, Given JD, et al. Reorganization of flexion reflexes in the upper extremity of hemiparetic subjects. Muscle Nerve. 1999;22(9):1209-1221.

Dietz V, Ketelsen UP, Berger W, et al. Motor unit involvement in spastic paresis. Relationship between leg muscle activation and histochemistry. J Neurol Sci. 1986;75(1):89-103.

Forster A, Young J. Incidence and consequences of falls due to stroke: A systematic inquiry. BMJ. 1995;311(6997):83-86.

Fridén J, Lieber RL. Spastic muscle cells are shorter and stiffer than normal cells. Muscle Nerve. 2003;27(2):157-164.

Gandevia SC, Allen GM, McKenzie DK. Central fatigue. Critical issues, quantification and practical implications. Adv Exp Med Biol. 1995;384:281-294.

Ghez C. The control of movement. In: Kandal ER, Schwartz JH, Jessell TM, eds. Principles of Neural Science. Norwalk, Appleton & Lange, 1991:533-563.

Ghez C. Voluntary movement. In: Kandal ER, Schwartz JH, Jessell TM, eds. Principles of Neural Science. Norwalk, Appleton & Lange, 1991:609-625.

Hammond MC, Fitts SS, Kraft GH, et al. Co-contraction in the hemiparetic forearm: Quantitative EMG evaluation. Arch Phys Med Rehabil. 1988;69(5):348-351.

Hammond MC, Kraft GH, Fitts SS. Recruitment and termination of electromyographic activity in the hemiparetic forearm. Arch Phys Med Rehabil. 1988;69(2):106-110.

Hara Y, Akaboshi K, Masakado Y, et al. Physiologic decrease of single thenar motor units in the F-response in stroke patients. Arch Phys Med Rehabil. 2000;81(4):418-423.

Health & Welfare White Paper. Ministry of Health & Welfare, 2007.

Hu XL, Tong KY, Hung LK. Firing properties of motor units during fatigue in subjects after stroke. J Electromyogr Kinesiol. 2006;16(5):469-476.

Kamper DG, Rymer WZ. Impairment of voluntary control of finger motion following stroke: Role of inappropriate muscle coactivation. Muscle Nerve. 2001;24(5):673-681.

Keele SW. Movement control in skilled motor performance. Psychol Bull. 1968;70(6 pt 1):387-403.

Kirker SG, Jenner JR, Simpson DS, et al. Changing patterns of postural hip muscle activity during recovery from stroke. Clin Rehabil. 2000;14(6):618-626.

Lundy-Ekman L. Neuroscience: Fundamentals for rehabilitation. 2nd ed. Saunders, 2002:188-197.

Marigold DS, Eng JJ, Timothy Inglis J. Modulation of ankle muscle postural reflexes in stroke: Influence of weight-bearing load. Clin Neurophysiol. 2004;115(12):2789-2797.

Newham DJ, Hsiao SF. Knee muscle isometric strength, voluntary activation and antagonist

co-contraction in the first six months after stroke. Disabil Rehabil. 2001;23(9):379-386.

O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. Brain. 1996;119(Pt 5):1737-1749.

Sacco RL, Benjamin EJ, Broderick JP, et al. American Heart Association Prevention Conference. IV. Prevention and rehabilitation of stroke. Risk factors. Stroke. 1997;28(7):1507-1517.

Sahrmann SA, Norton BJ. The relationship of voluntary movement to spasticity in the upper motor neuron syndrome. Ann Neurol. 1977;2(6):460-465.

Scelsi R, Lotta S, Lommi G, et al. Hemiplegic atrophy. Morphological findings in the anterior tibial muscle of patients with cerebral vascular accidents. Acta Neuropathol (Berl). 1984;62(4):324-331.

Smith LE, Besio WG, Tarjan PP, et al. Fractionated premotor, motor, and ankle dorsiflexion reaction times in hemiplegia. Percept Mot Skills. 1998;86(3 Pt 1):955-964.

Young JL, Mayer RF. Physiological alterations of motor units in hemiplegia. J Neurol Sci. 1982;54(3):401-412.

This article was received October 9, 2007, and was accepted November 9, 2007.