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Initiation and Termination of Electromyographic Activity in the Early Hemiparetic Wrist

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국문요약

초기 편마비 환자의 손목에서 근수축 개시 및 종료의 지연

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본 연구는 초기 편마비 환자의 손목에서 표면근전도 분석을 통해 근수축 개시 및 종료의 특성들을 알아보고, 임상적인 치료방법의 기초를 제안하고자 실시하였다. 연구대상자는 원주기독병원에 뇌졸중으로 입원한 환자 중 발병 후 3개월 미만인 13명과 원주시에 거주하는 대조군 7명이었다. 근수축 개시 및 종료의 지연은 표면근전도를 이용하여 손목굽힘근과 손목펴짐근에서 손목관절의 굽힘과 폄동작 시 3초의 근전도 신호음에 따라 가장 빠르고 강하게 최대 등척성 수축과 이완을 하여 신호를 수집하였다. 그 결과 편마비 환자의 환측은 건측과 대조군에 비해 손목관절 굽힘과 폄동작에서 근수축 개시및 종료가 유의하게 지연되었으며, 개시보다 종료가 더 유의하게 지연되었다. 따라서 초기 뇌졸중 환자의 근약화는 근육의 개시 및 종료의 반응시간 지연에 영향을 준다고

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볼 수 있다. 앞으로 운동조절과 연관되어 기능적인 회복을 유도할 수 있는 근수축의 민첩성 훈련과 근섬유 동원의 효율성을 증진시키기 위한 치료방법들이 연구되어야 할 것이다.

핵심단어: 근약화; 뇌졸중; 손목굽힘근; 손목펴짐근; 표면근전도.

Introduction

After a cerebral injury, which may follow a stroke or head trauma, many patients suffer a constellation of physical signs known collectively as the "upper motorneuron" syndrome (Gemperline et al, 1995). These signs include spastic hypertonia, muscular weakness, and impaired movement coordination.

When the functional grasp and release of upper limbs, there is a limitation, and this syndrome can see what is limited on motor recovery of their proximal upper limb than the distal upper limb relatively.

Agility and coordination are required in the initiation and termination of repetitive phasic muscle contraction in activities of daily living. Researchers studied to analyze a muscle contraction delay of an onset and offset in a wrist joint of the hemiparesis who used an electromyogram in order to evaluate the agility of muscle contraction until now (Angel, 1981; Chae et al, 2002; Hammond et al, 1988). When a wrist joint moves, cocontraction of agonist and antagonist related to spasticity occurred in the chronic hemiplegic patient who used an electromyogram and delyed muscle contraction of initiation and termination in the affected sides (Hammond et al, 1988).

The functional problems of the upper limbs after brain injury are more related to the physiologic change of the nervous system by muscle weakness and loss of agility than spasticity regarding recent studies, it was recognized that there were efficiency and relationship of motor unit recruitment (De Luca, 1993; Kupa et al, 1995; Toffola et al, 2001). However, because most of the existing studies was not classified since the injury of the stroke, it is hard to discriminate whether the cause of muscle weakness is due to atrophy that is peripheral nervous system by a progress of time or neurological damage of an initial central nervous system (Newham and Hsiao, 2001). So the functional evaluations were not able to provide the basic understanding of a nerve physiology.

The purpose of this study is going to recognize the agility of wrist muscle contraction in hemiplegia caused by early cerebrovascular accident (CVA). This may assume that an onset and an offset of muscle contraction becomes delayed by the decrease of phasic contraction and muscle weakness in a wrist with

hemiplegia. Therefore, this study observes muscular response time regarding motor control during the rehabilitation stage of early stroke patients. The agile and concentrated treatment strategy related the muscular reaction time will be used as good information.

Methods

Subjects

Thirteen stable patients with a history of unilateral CVA 9 to 87 days previously, and seven healthy control subjects with a similar balance of age and sex were tested. Inclusion criteria included 1) an interval of at least 3 months from stroke onset; 2) unilateral lesion; 3) manual muscle testing that is poor or above in wrist flexor and extensor; 4) ability to follow three second maximal isometric contraction command. 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.

Table 1. Clinical characteristics of patients (N=13)

Patients	Age(yr)	Gender	Time since injury(days)	Plegic side	FMA[a]	MAS[b](F/E)	MMT[c](F/E)	Diagnosis
1	75	M	46	Lt	57	0/0	G/G	BH
2	68	F	24	Rt	56	0/0	G/G	MI
3	66	F	30	Lt	54	1/1	F+/F+	TH
4	54	M	87	Rt	62	0/0	G/G	MI
5	55	F	24	Lt	22	0/0	P+/P	MI
6	54	F	22	Lt	46	1/1	F-/F+	BH
7	53	M	80	Lt	38	1+/1+	F-/F	PI
8	70	F	64	Lt	17	0/0	F-/P+	MI
9	51	M	58	Rt	62	0/0	F-/F+	TI
10	59	M	9	Lt	60	0/0	G/G	MI
11	64	F	34	Rt	48	0/1	F-/F+	MI
12	64	F	26	Rt	46	0/0	F+/F-	BH
13	65	M	10	Rt	60	0/0	G/G	PI
Mean	61.4		39.5		48.3			

[a] Fugl-Meyer motor assessment

[b] Modified Ashworth scale

[c] Manual muscle testing

E: extensor, F: flexor, BH: basal ganglia hemorrhage, MI: middle cerebral artery infarction PI: pontine infarction, TH: thalamic hemorrhage, TI: thalamic infarction

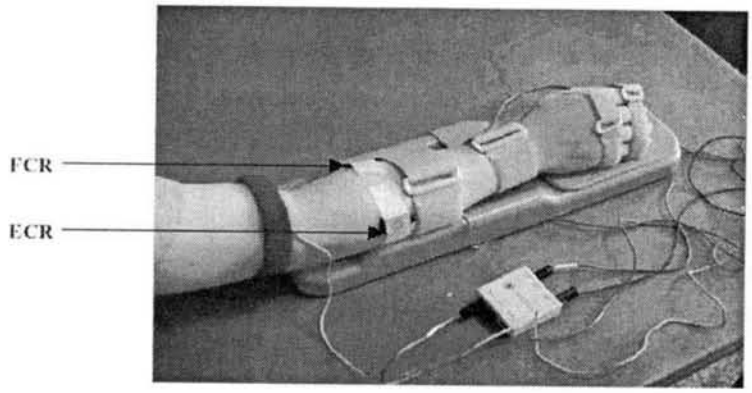

Figure 1. The 'forearm based skateboard' and the placement of the electrodes on the flexor carpi radialis and extensor carpi radialis muscles

Instruments

The surface electromyography (sEMG) signal was detected with an active parallel-bar electrode (bar size: 1 mm by 10 mm, located 10 mm apart differential electrode (DelSys)[1]. The electrodes were placed on the flexor carpi radialis and extensor carpi radialis muscle belly (Cram et al, 1998) (Figure 1). The EMG signals were digitally band-pass filtered at 20~450 Hz and notch filtered at 60 Hz to reduce noise. A sampling frequency of 1000 Hz was used. After collection, the data were transferred to a personal computer for data reduction.

The Acqknowledge 3.72 program[2] was employed to set up the required parameters and to store the EMG signal as computer files.

Procedures

Subjects were instructed to contract the wrist flexor or extensor as forcefully and quickly as possible against the confinement of the apparatus in response to an audible beep, and to relax the muscle as quickly as possible as soon as the beep terminated. For wrist flexion, all subjects were asked to respond to audible beeps consisting of three trials of 3-s contractions. The trials were presented in a balanced random order in order to minimize subject anticipation, The procedure was repeated for wrist extension. Delay in recruitment of the EMG signal was defined as the time interval between the onset of the audible beep and the onset of the EMG signal (Figure 2).

Delay in termination of the EMG signal was defined as the time interval between the offset of the audible beep and the offset of the EMG signal. The onset of the EMG activity was defined by com-

1) Delsys Inc. Boston, MA. U.S.A.
2) Biopac systems Inc. CA. U.S.A.

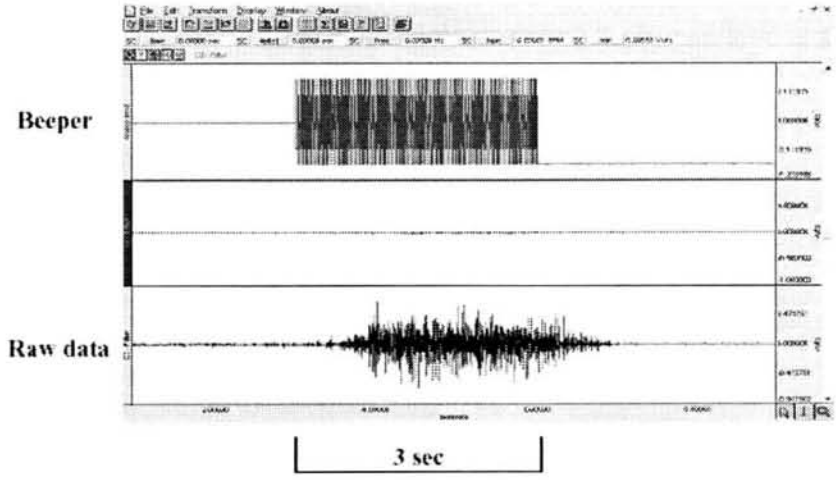

Figure 2. EMG raw data during wrist isometric contraction of the patient #11

puter-based onset and offset determination techniques (ICCs=.932/.977). This exhibits significantly higher reliability (Chung et al, 2003). The parameters were evaluated by low pass software filter (50 Hz) and the other parameters evaluated were the number of samples assessed in the sliding window (25 ms) and the magnitude of the deviation from the baseline required to indicate the threshold (3 SD). Processing was done using MatLab' signal processing toolkit[3].

Statistical analysis

An independent t-test was used to examine the differences between sides (flexion and extension, onset time and offset time) and paired t-test of with in the sides. All statistical analyses were performed using SPSS 10.0 for windows. A p value level of <.05 was used as the level of significance.

Results

The results of study were as follows. The onset and offset of muscle contraction were significantly delayed on the more paretic than nonparetic and control sides (Table 2). Offset was significantly more delayed than the onset on the paretic sides (Figure 3).

Discussion

Slower initiation of movements has been shown in performance of simple reaction time tasks by persons with unilateral cerebral damage (Benton and Joynt, 1959; DeRenzi and Faglioni, 1965; Howes and Boller, 1979). Surface elecromyographic studies have documented slower muscle re-

3) Math Works Inc. MA. U.S.A.

Table 2. Comparison of delay in contraction onset and offset of the wrist flexor and extensor muscles between side

Sides	Wrist flexion		Wrist extension	
	Onset delay	Offset delay	Onset delay	Offset delay
Control	.43(.08)	.46(.08)	.40(.07)	.44(.10)
Nonparetic	.48(.10)	.55(.16)	.45(.08)	.52(.06)*
Paretic	.52(.10)*†	.75(.11)*†	.57(.18)*†	.70(.14)*†

All values in the cells are mean (SD): second

*Significantly different from control sides with independent t-test ($p<.05$)

†Significantly different from nonparetic sides with paired t-test ($p<.05$)

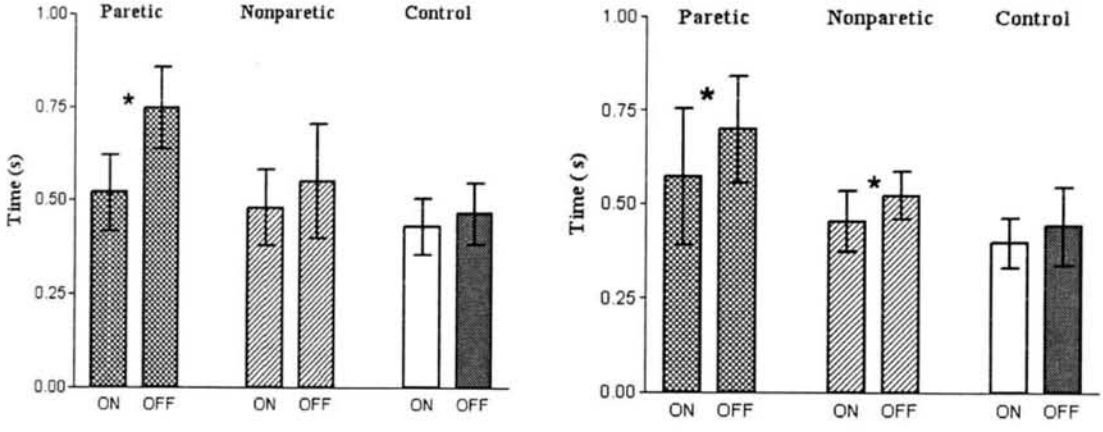

Figure 3. Comparison of delay in contraction onset and offset of the wrist flexor and extensor muscles with in the sides

Values of the bar are mean±SD. ON: onset delay, OFF: offset delay

*Significant difference between onset delay and offset delay ($p<.05$).

sponsiveness (Angel, 1981; Nakamura et al, 1978).

We found a significant delay in initiation and termination of muscle contraction of the hemiparetic wrist compared to nonparetic and control wrist (Table 2). Angel (1981) showed that the latency from a visual signal to onset of EMG activity in arm muscles was longer in four of eight hemiparetic subjects. Nakamura and associates (1978) found that the onset of EMG activity was longer for the hemiparetic biceps during both elbow flexion and forearm supination. Sahrmann and Norton (1977) noted delayed recruitments of agonist contraction in the hemiparetic upper limb.

The classical explanation for the paresis

advanced by Hughlings Jackson is that there is a loss of descending excitation from major descending pathways to the spinal cord (Lassek, 1970). The final motor output among stroke survivors can be modulated by changes in descending and propriospinal excitatory and inhibitory inputs into the spinal interneurons and alpha motoneurons as well as neuroplastic changes consequent to brain injury (Dobkin, 1996; Nudo et al, 2001). Co-contraction of antagonist muscles is a widely recognized clinical problem and was expected in paretic forearms (Hammond et al, 1988; Sahrmann and Norton, 1977). Whereas spasticity hardly appeared in initial hemiparetic patients in this study, it seems that primary cerebral cortex dysfunction causes specific impairments in processing. This presented to be contributed to neurological deficit that is the reorganization of the corticospinal tract, change of characteristic motor unit and activation of spinal interneuron (Adams et al, 1990; Bohannon and Walsh, 1992; Colebatch et al, 1986; Newham and Hsiao, 2001).

We found a significant delay in termination times compared to initiation times in paretic agonist muscles. Subject presented longer than in control wrist flexor and extensor muscles (Figure 3). Consistent with prior studies, we found a significant delay in termination of muscle contraction of the hemiparetic upper limb compared to the nonparetic upper limb (Beneck et al, 1983; Dewald et al, 1995; Sahrmann and Norton, 1977). This is attributed to loss of supraspinal inhibitory influence on the normal interneuronal pool (Ghez, 1991), spasticity, abnormal co-contraction of antagonist and agonist muscles, and abnormal co-activation of synergistic muscles (Chae et al, 2002). The main effect is on the lateral reticulospinal tract that is the most nearly corticospinal tract that the loss of inhibitory delivery (Delwaide and Young, 1985). So a reduction in corticospinal input may also result in increased dependence on undamaged vestibulospinal, reticulospinal, and tectospinal pathways not a spasticity and abnormal co-contraction (Delwaide and Young, 1985).

Conclusion

This study also suggests that since muscle weakness of early stroke patients affects the functional delay of muscle contraction in upper limbs, further studies must be focused on treatment to improve muscle agility and muscle fiber recruitment efficiency that can induce the functional recovery correlated to motor control.

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