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Post stroke examination with evoked and voluntary surface EMG: A study with hypothenar muscles

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Abstract: By applying evoked and voluntary surface electromyography (EMG) recordings, motor unit changes in paretic hypothenar muscles of stroke survivors were examined. Eleven stroke subjects participated in the study. The maximum M waves and voluntary surface EMG signals at different muscle contraction levels were recorded from the hypothenar muscles bilaterally in each subject. The motor unit number index (MUNIX) values were estimated from the mathematical model describing the relation between the surface EMG signal and the ideal motor unit number count derived from M wave and surface EMG measurements. A decrease in both the maximum M wave amplitudes and the estimated MUNIX values was observed in paretic muscles of the stroke subjects compared with the contralateral muscles. But no significance was found. The ratio between the maximum voluntary EMG to compound muscle action potential was significantly deceased in paretic muscles compared with the contralateral side. These findings, in combination with previous simulation and experiment outcomes, provide evidence of muscle activation deficiency and complex neuromuscular changes post stroke, thus helping understand complicated determinants of paretic muscle weakness for stroke rehabilitation.

Key words: surface electromyography (EMG); stroke; paretic muscle; motor unit (MU)

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中风后小鱼际肌的诱发和自主表面肌电图研究

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摘要:利用诱发和自主表面肌电研究在中风后偏瘫病人的小鱼际肌的生理变化.通过对 11 个受测者患侧和正常侧的对比,发现患侧肌肉的复合动作电位和运动单位个数索引略为降低.正常侧的小鱼际肌最大自主收缩肌电与复合动作电位的比值显著大于患侧,表明患侧肌肉的激活能力明显不足.相比传统的针电极检测,通过无创的表面肌电实现了对中风后运动单位变化的研究,以促进对中风后肌无力的理解与康复.

关键词:表面肌电;中风;肌无力;运动单位

0　Introduction

Following a hemispheric stroke, many patients suffer a variety of disabling physical symptoms on the contralesional side of the body, often including weakness, which can be highly debilitating. Weakness can be the result of one or more mechanisms, including disuse, autonomic changes, pressure neuropathy, loss of central motoneuron trophic influences, and trans-synaptic motor neuron degeneration[1]. Regardless of the origins of weakness, it is very important to determine how a cerebral lesion might affect motor unit survival and function. This information is helpful to guide development of effective treatments for muscle weakness.

Electrophysiological studies have played an important role in assessing neuromuscular changes post stroke. For example, various forms of motor unit number estimation (MUNE)[2] have been used to detect spinal motor neuron loss and monitor disease progression in amyotrophic lateral sclerosis (ALS) and other neuromuscular diseases[3-7]. Although no significant difference was found in anterior horn cell numbers of the affected and unaffected sides in stroke[8-9], the MUNE studies have revealed varied degrees of reduction in motor unit number in the hemiparetic muscles[1,10-13].

The traditional MUNE methods involve estimates of single motor unit potential size. The procedures are usually based on either incremental nerve stimulation or intramuscular electromyogram (EMG) decomposition based spike triggered averaging. Both approaches are potentially laborious and time-consuming. A recently developed technique uses compound muscle action potential (CMAP) and the surface EMG interference patterns during voluntary muscle contraction to derive an index proportional to the number of motor units in a muscle[14]. This method is called motor unit number index (MUNIX), and is easy and quick to perform. Upon introduction of the method, it was effectively used to quantitatively assess the pathophysiology of motor neuron loss in ALS patients[14]. In recent years, the method has attracted increasing applications, primarily providing an objective measure of the ALS progression and also in other studies[15-19]. Recently, the method was also applied to the

paretic first dorsal interrsseous (FDI) muscles of stroke and spinal cord injury subjects[20-21].

The current study presents an examination of the paretic hypothenar muscle (which is actually a muscle group located at the ulnar side of the palm) using evoked and voluntary surface EMG recordings. The hypothenar muscle is superficial and aids with movements of the little finger. The MUNIX technique was applied to examine whether there was motor unit loss or muscle fiber shrinkage in paretic hypothenar muscles of stroke survivors. Alterations in capacity for muscle activation were also assessed. Such investigations of motor unit and muscle properties can help understand complicated determinants of stroke induced muscle weakness, thus facilitating design of appropriate therapy or rehabilitation strategies.

1 Methods

1.1 Subjects

Eleven subjects who sustained hemiparetic stroke participated in the study. All procedures were performed in accordance with the Declaration of Helsinki and approved by the local institutional review board. Prior to participation in the study, all subjects gave written informed consent. A physical therapist performed clinical assessment for each stroke subject, including the upper arm impairment measures using the Fugl-Meyer test[22] and the Chedoke-McMaster assessment[23] (Tab. 1).

Tab. 1 Demographic and clinical information of the stroke subjects

ID	gender	age	paretic	duration	Chedoke	FM
1	M	69	L	9	3	21
2	M	44	R	4	5	58
3	F	62	L	15	3	34
4	M	48	R	1	6	54
5	F	40	L	5	2	11
6	F	58	R	14	2	14
7	M	70	R	3	3	23
8	M	64	R	10	4	56
9	F	60	R	7	4	40
10	M	72	R	5	7	64
11	M	58	R	10	5	51

1.2 Experiments

Subjects were instructed to sit in a comfortable position with the hand and forearm rested in a vertical half supinated position on a height-adjustable table. The experiments were performed bilaterally on the hypothenar muscles of each stroke subject. The Sierra Wave EMG system (Cadwell Lab Inc, Kennewick, WA, USA) was used in this study. We followed standard motor nerve conduction studies to place the electrodes. Two 10 mm silver/silver chloride disc surface recording electrodes were used to record electrical activity from the hypothenar muscles. The active electrode was placed in the estimated motor point region. The reference electrode was placed at the distal phalanx of the pinky finger and the ground electrode was on the dorsum of the hand between the stimulus and recording sites.

The M wave was evoked with a cathode placed approximately 2 cm proximal to the wrist crease over the ulnar nerve. The duration of each stimulation single pulse was set to be 200 μs. The stimulation intensity started around 18 mA, increasing approximately 3～4 mA each step, until the maximum motor response was reached. Then the stimulation intensity was further increased by approximately 20% of the final intensity to prevent no further enlargement in the M wave peak-to-peak amplitude.

After the compound muscle action potential (CMAP) (i. e., the maximum M wave) recording, voluntary surface EMG signals were recorded from the hypothenar muscles while subjects generated isometric voluntary contractions in different force levels from minimal to maximal effort. The different force levels were recorded using a single trial with graded contraction consisting of interference EMG epochs. Subjects were offered substantial rest to avoid muscle and mental fatigue during the recording. Visual feedback of surface EMG was displayed on a computer screen. The M wave and surface EMG recordings were sampled at 6.4 kHz and 32 kHz, with a bandpass filter setting

at 3 Hz～2 kHz and 10 Hz～10 kHz, respectively. A notch filter was used for the surface EMG recording to remove the power line interference.

1.3 Data analysis

The maximum M wave and surface EMG interface pattern (SIP) at different contractions were used to compute the MUNIX[14]. For each level of SIP, we first calculated its ideal case motor unit count (ICMUC), defined as $\frac{\text{Maximum M Wave Power}\times\text{SIP Area}}{\text{Maximum M Wave Area}\times\text{SIP Power}}$. Then, we applied linear regression analysis to estimate the relationship between SIP area and ICMUC: $\text{ICMUC}=\beta(\text{SIP Area})^{\alpha}$, where parameters β and α were obtained from the regression. Finally, the MUNIX was defined as the value when SIP area was 20 mV·ms: $\text{MUNIX}=\beta(20)^{\alpha}$.

With available MUNIX values, the motor unit size index (MUSIX), which represents the average amplitude of the individual surface motor unit action potentials (MUAPs), was estimated by dividing MUNIX into the CMAP or maximum M wave amplitude[16]: $\text{MUSIX}=\frac{\text{Maximum M wave amplitude}}{\text{MUNIX}}$.

During the MUNIX calculation, very low amplitude surface EMG signals may give very high ICMUC values. Therefore, three criteria were imposed to accept a valid SIP epoch: ①SIP area＞20 mV·ms; ②ICMUC＜100; and ③SIP area/CMAP area＞1. In addition, CMAP amplitude less than 0.5 mV were removed from the analysis[16].

In addition to MUNIX analysis, we also calculated the ratio between the maximum voluntary EMG amplitude (averaged from 1 s period) and the maximum M wave amplitude. This ratio can help assess alterations in muscle activation capacity.

All the examined parameters were calculated bilaterally in hypothenar muscles of each stroke subject. One-way ANOVA was performed to assess the differences between the paretic and contralateral muscles.

2 Results

Maximal M wave and voluntary surface EMG signals at different contraction forces were recorded from bilateral hypothenar muscles of all stroke subjects. Fig. 1 demonstrates an example of the comparison of the MUNIX calculations from the paretic and contralateral muscles of one stroke subject. For this subject, the maximum M wave was 6.5 mV for the paretic muscle and 8.4 mV for the contralateral muscle. The maximum voluntary surface EMG generated by the paretic muscle was also much lower than that from the contralateral muscle. With the measured maximum M wave and different levels of voluntary surface EMG signals, this stroke subject showed a MUNIX value of 114 for the paretic hypothenar muscle, which was lower than the MUNIX value of 165 for the contralateral muscle. The MUSIX value was 56.7 μV for the paretic muscle and 50.9 μV for the contralateral muscle.

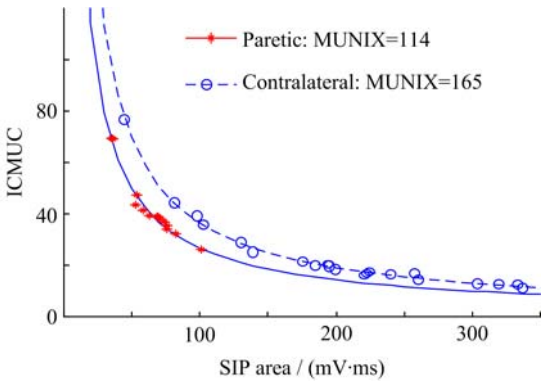

Fig. 1　MUNIX comparison between paretic and contralateral muscles of a stroke subject

Maximal M waves from the paretic and contralateral hypothenar muscles were averaged across all subjects. The averaged maximal M wave amplitude in the paretic muscle was (9.2±0.8) mV (mean±standard error, the same for the rest of the paper), and was (9.8±0.7) mV for the contralateral muscle ($p>0.05$). A good fit of exponential regression for the relationship between SIP area and ICMUC was found across all stroke subjects. We observed a slight decrease of MUNIX

values in the paretic hypothenar muscles when compared with the contralateral muscles. Eleven stroke subjects showed a MUNIX value of 152±13 for the paretic muscle, which was lower than the MUNIX value of 170 ± 10 for the contralateral muscle ($p>0.1$). The MUSIX value of the paretic hypothenar muscle averaged from all the stroke subjects was (61.5±3.1)μV, while the MUSIX value averaged from the contralateral muscles was (58.7±3.9)μV. ($p>0.5$).

Fig. 2 shows the correlation between maximal M wave and the MUNIX values of stroke subjects for both paretic and contralateral muscles. A positive correlation between the maximal M wave and MUNIX was observed in both muscles. There was no correlation between maximal M wave and the MUSIX measurement. We observed a significant decrease in the ratio of the maximum voluntary EMG amplitude to the CMAP amplitude in paretic muscles (0.02±0.003) compared with the contralateral muscles (0.06 ± 0.005) ($p<0.01$).

3 Discussion

This study presents an application of the MUNIX technique in examination of paretic hypothenar muscles of stroke survivors. Since the MUNIX measurement provides an index of the number of motor units in a muscle, the MUNIX values in different muscles may not match the actual motor unit numbers estimated using various MUNE methods. Recently, we have examined the sensitivity of the MUNIX technique to changes in motoneuron and muscle properties using motoneuron pool and surface EMG models[24]. Our simulation results indicate that MUNIX estimates can appropriately track variations in input motor unit number when keeping motoneuron pool and muscle parameters unchanged. Such MUNIX estimates are not sensitive to alterations in motor unit control properties (e. g., reduction of motor unit firing rate and compression of motor unit recruitment range) which may happen after stroke[25]. One limitation of the MUNIX estimates is that if the amplitude of each MUAP is reduced, then MUNIX estimates substantially underestimate the motor unit numbers in the muscle. The findings from the simulation emphasize the importance of a parallel MUSIX measurement in addition to MUNIX estimates, which may provide additional useful information.

The emphasis in the current study was to explore stroke survivors' paretic muscle changes in comparson with the contralateral muscles. Different from previous reports of traditional MUNE studies[1,10-13,26] as well as a recent study examining MUNIX changes in the FDI muscle[20-21], we did not observe a significant reduction of MUNIX values in the paretic hypothenar muscles compared with the contralateral muscles. We found that there was no significant change in MUSIX values between

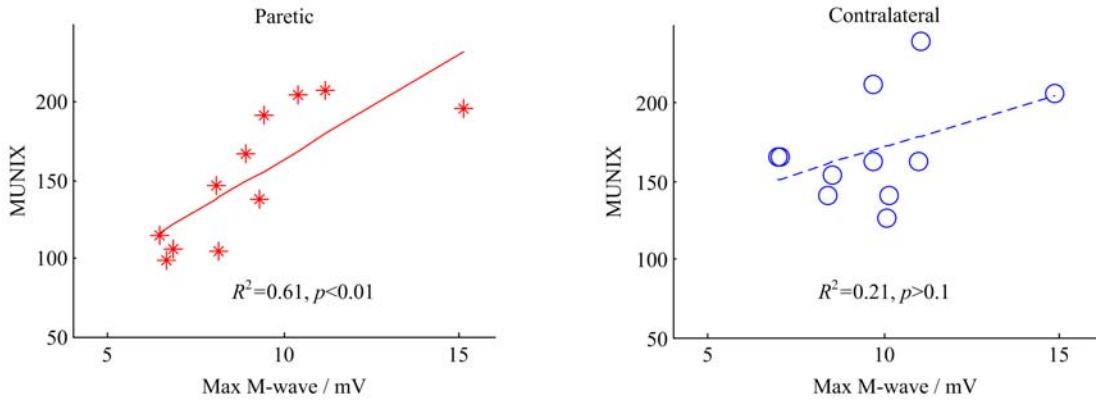

Fig. 2 Correlation between M wave amplitude and MUNIX for paretic and contralateral muscles of stroke subjects

paretic and contralateral hypothenar muscles which is consistent with the findings from the FDI muscles[20-21]. Meanwhile, we observed that there was a significant reduction in activation capacity of paretic muscles compared with the contralateral muscles, as demonstrated by the ratio of the maximum voluntary EMG to the CMAP. This might be due to different factors including the motor unit peak firing rate reduction[25] and partial paralysis. It is not clear how the impaired muscle activation capacity might influence MUNIX measurement. This remains a topic for further examination to help understand whether the MUNIX was overestimated in the paretic hypothenor muscles.

In conclusion, the present cross-sectional study using surface EMG recordings provides electrophysiological evidence of reduced muscle activation capacity post stroke. The surface EMG based examination requires minimum amount of electrical stimulation and is convenient to implement. The findings, together with our previous model and experimental studies, suggest complex neuromuscular alterations (motor unit control property changes, muscle fiber atrophy, muscle fiber denervation and reinnervation, etc.) post stroke. It is important to quantify the determinants of different factors contributing to motor impairment to promote appropriate medication or therapies for stroke rehabilitation.

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