Spasticity Evaluation of Hemiparetic Limbs in Stroke Patients before Intervention by Using Portable Stretching Device and EMG

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Abstract

The aim of this study is to utilize the portable muscle tone measurement device and surface EMG for investigating the spasticity on the elbow flexors before BOTOX® intervention. The reactive torque and reactive EMG (biceps brachii and triceps brachii muscles) of elbow joint induced at different stretching velocities (1/3, 1/2, 1 and 1.5 Hz) in limited range (-30 to +30 degrees, 90 degrees of elbow flexion was defined as zero degree) were recorded simultaneously in 2 weeks before BOTOX® treatment. The velocity-dependent viscous component (Bω) of elbow flexor and the EMG threshold defined as the angle at which the sustained EMG activity surpassed two folds of standard deviation of rest EMG prior to stretch were used for evaluating the severity of spasticity. In current study, we demonstrated that the viscoelastic parameter as well as EMG threshold could be used for quantifying the degree of spasticity. These parameters would be useful for quantifying the effects of BOTOX treatment in stroke patients.

***Keywords*:** Spasticity, Muscle tone measurement system, EMG, Elbow

Introduction

Spasticity, a complicate symptom, is usually seen in patients with upper motor neuron dysfunction, such as cerebral vascular accidents (CVA), spinal cord injuries (SCI), multiple sclerosis, etc., and defined as a velocity-dependent increase in the resistance to imposed movement [1,2]. The mechanism of spasticity is commonly thought as exaggerated stretch reflex. Stretch reflex is important for movement control, in normal condition, which incorporates with other spinal reflex by cortex (pre-motor and supplementary areas) to maintain its human balance and posture. However, there is lack of inhibitory signal from cortex via dorsal reticulospinal tract and lateral corticospinal tract in upper motor neuron dysfunction patients in which the alpha motoneuron pool at the segmental level is hype-excitable and results in exaggerated reflex [3, 4]. This problem hinders the functional improvement of these spastic patients. Spasticity may interfere with voluntary motor function in patients with residual muscle power. In addition, excessive muscle tone frequently causes difficulties in daily activities. Muscle pain or discomfort as well as contracture would occur at the joint surrounded by spastic muscles [5-6].

To alleviate the spasticity, some interventions such as medication, surgery, physical therapy and surface electrical stimulation have been commonly used. However, the treatment effects of these methods are still limited [2, 7]. Recently, Botulinum toxin type A (BTX-A) has been used for reducing the lower extremity spasticity in cerebral palsy children because of its localized effect [8, 9]. BTX-A could reduce spasticity only in the injected muscles so that it would not result in systemic side effects, such as whole body weakness or sensation loss. BTX-A reduces the spasticity by producing chemical denervation from avoiding the release of acetylcholine at the neuromuscular junction [8-9]. BOTOX® is commercially available medicine purified from BTX-A for clinical use.

Although BTX-A is now widely accepted as being an effective antispastic agent in a number of randomized and open studies, evidence supporting its clinical efficacy is not convincing. Recent studies have been investigated the treatment effect of BTX-A for spasticity suppression in upper limbs for adult stroke patients [4, 10-13]. However, current approaches often take functional outcome measures, self-report questionnaire, muscle force or range of motion as the semi-quantitative assessment methods. However, there is

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Table 1. Summary of the subjects' clinical data

Subject	Age	Gender	Months post-injury	Affected side	Modified Ashworth score	Brumstrom stage
A	56	M	18	Right	1	3
B	39	F	10	Right	1	5
C	64	F	7	Left	1	3
D	64	F	7	Right	2	3
E	63	F	16	Right	3	3
F	58	M	26	Right	2	3
G	61	M	120	Left	1	5
H	65	F	7	Left	3	4
I	60	M	57	Left	2	4
J	54	F	65	Left	3	4
K	70	M	16	Right	2	3
L	39	F	48	Right	2	5
M	50	F	6	Left	1	3

lack of study utilizing quantitative assessment techniques for evaluating the treatment of BTX-A in spasticity. By applying sensitive and quantitative evaluation methods, the optimal dose of BTX-A effective treatment can be established [4].

In our previous studies, we have developed a portable device for quantifying the velocity-dependent property of spasticity on upper limbs [14]. This study was to investigate muscle tone in stroke patients before BOTOX® intervention from biomechanical approach via portable measurement system as well as the neurophysiological evaluation from electromyography (EMG). By measuring the muscle tone and EMG, this study shall provide a quantitative base for evaluating the treatment for spasticity reduction.

Methods

Subjects

13 chronic stroke patients (onset > 6 months) with hemiparesis participated in this study. The age ranged from 39 to 70 years (mean (SD), 57.5(9.8)). All patients were diagnosed and evaluated by a qualified physiatrist. Each subject had no other contraindication and possessed sufficient cognition to understand the whole procedures and to be cooperative. There was no contracture around the elbow joint seen in these stroke patients. In addition, patients with Brunstrom stage above scale three, who are the candidates for BOTOX® injection, were included. The summary of subjects' data was listed in Table 1. The muscle tone, EMG, as well as scale-based evaluations before BOTOX® intervention, were collected as the baseline.

Instrumentation

The portable muscle tone measurement system developed in our previous study [14] was used to measure the response of stretch reflex. This measurement system was designed to measure two important signals during sinusoidal stretch of elbow joint, which are reactive resistance and stretch velocity. The measurement system consists of two major parts, including air-bags fixed on ventral and dorsal sides of wrist for sensing reactive torque and light-weight angular rate sensor (ARS-C142, Watson Industries, WI, USA) mounted on the dorsal side of the middle forearm for measuring stretch velocity. During the stretching procedure, the joint trajectory can be also derived from the integration of angular velocity. In addition, two surface EMG electrodes (MA100, Motion Lab Systems, LA, USA) were used to record the activity of targeted muscles during passive sinusoid movement. The reactive resistance, angular rate and EMG signals were sampled at 1000 Hz via an analogue-to-digital converter (NI,) with 12-bit resolution. The gain of amplifier for EMG was 380. All the systems were controlled by LabVIEW (National Instrumentation, Austin, TX), and the off-line data processing were under Matlab (The MathWorks) for further analysis.

Experimental Procedures

The assessment was first conducted at 2 weeks before BOTOX® injection. Further experiment will be performed 14 days, 2 months, and 5 months after intervention. In this study, we just showed the biomechanical and neurophysiological responses before intervention. All the tests were assessed by the same investigator who is a qualified physical therapist.

Before experimental assessment, the stroke patients were assessed by the investigator using modified Ashword Scale (MAS, score: 0, -1, +1, 2, 3, 4). The MAS scores were compared with the data from biomechanical and neurophyiological approaches later. During stretching experiment, the subjects were asked for lying down comfortably on the bed with slight abduction of upper arm and their elbow was restricted in constant range, +30 to -30 degrees, by using an elbow brace. The zero degree was defined as when elbow is at 90 degrees of flexion, and positive direction stands for extending from zero degree. Besides, the EMG electrodes were placed on the primary flexor and primary extensor of the elbow: biceps brachii (BB) and triceps brachii (TB). During the whole experiment, the subjects were requested to relax their upper limb.

The stretch reflexes were elicited by rotating the elbow sinusoidally. Four different stretch velocities (1/3 Hz, 1/2 Hz, 1 Hz and 3/2 Hz) were imposed on the elbow manually. Between each trial, the subject was rest for 30 seconds. With the assist of metronome and range limiter (elbow brace), the investigator can easily apply a regular sinusoidal displacement stretch after

a brief instruction. The selection of highest stretch frequency (1.5 Hz, with peak velocity about 280 deg/sec) used in this study is based on the criteria that the stretch velocity is higher enough to elicit the stretch reflex in spastic limb but not to cause human interaction due to discomfort in test subjects [14].

Data Analysis

For biomechanical data, our analytic approach in estimating velocity-dependent viscous component is based on the relationship between externally imposed joint displacements and the corresponding joint resistance. During a stretch, the measured reactive resistance results from inertial (I), viscous (B), elastic (K) contributions and initial constant offset. The offset in reactive resistance can be removed the baseline of the measured resistance. Thus, the dynamic equation of movement can be written as:

$$T(t) = I\ddot{X}(t) + B\dot{X}(t) + KX(t) \quad (1)$$

where X(t) is joint displacement, $\dot{X}(t)$ and $\ddot{X}(t)$ denote the joint angular velocity and acceleration, respectively. Because we took sinusoid movement as the assessed method, we could substitute Asin(ωt) for X(t). The equation (1) can be rewritten as:

$$T(t) = A[(K - I\omega^2)\sin(\omega t) + B\omega\cos(\omega t)]$$
$$= A\sin(\omega t+\theta)\,[(K-I\omega^2)^2 + (B\omega)^2]^{1/2} \quad (2)$$

where $\theta=\tan^{-1}[(B\omega)/(K-I\omega^2)]$. After FFT of the recorded torque data, we could find the different θ (phase lag) at different stretch angular frequency (ω). Utilizing known reactive torque value, angular frequency (ω) and derived phase lag (θ), the viscous components (Bω) could be estimated. With four viscous components (BW1/3, BW1/2, BW1 and BW3/2), one viscosity B was derived for each subject. Please refer to [14] for detailed information.

The raw EMG was fed through notch filter and bandpass (10 Hz-400 Hz) filter for eliminating 60 Hz noise and motion artifacts respectively. The filtered EMG was rectified and lowpass filtered to produce linear envelope (LE) [17]. The LE form of EMG signals was processed for determining the threshold. The threshold of EMG activity was defined as the angle at which the sustained EMG activity surpassed two folds of standard deviation of rest EMG prior to stretch. The threshold of EMG at varied stretch velocities was normalized to be the percentage of total range.

Results

The baseline conditions including biomechanical and neurophysiological data of thirteen chronic stoke patients recruited for this study were recorded.

Biomechanical data

With varied viscous components (Bω1/3, Bω1/2, Bω1 and Bω3/2), one viscosity B was derived for each subject. Figure 1 shows the grouped data with the same MAS value at different stretching frequencies and viscosity. The line with triangle, the line with square and the line with diamond stand for the spastic muscle property at grouped subjects with MAS=1, MAS=2 and MAS=3 respectively.

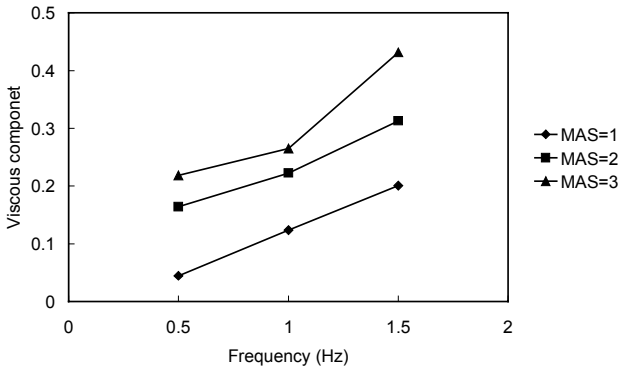

Figure 1. Viscous component (Bω) in subjects with varied severities of spasticity.

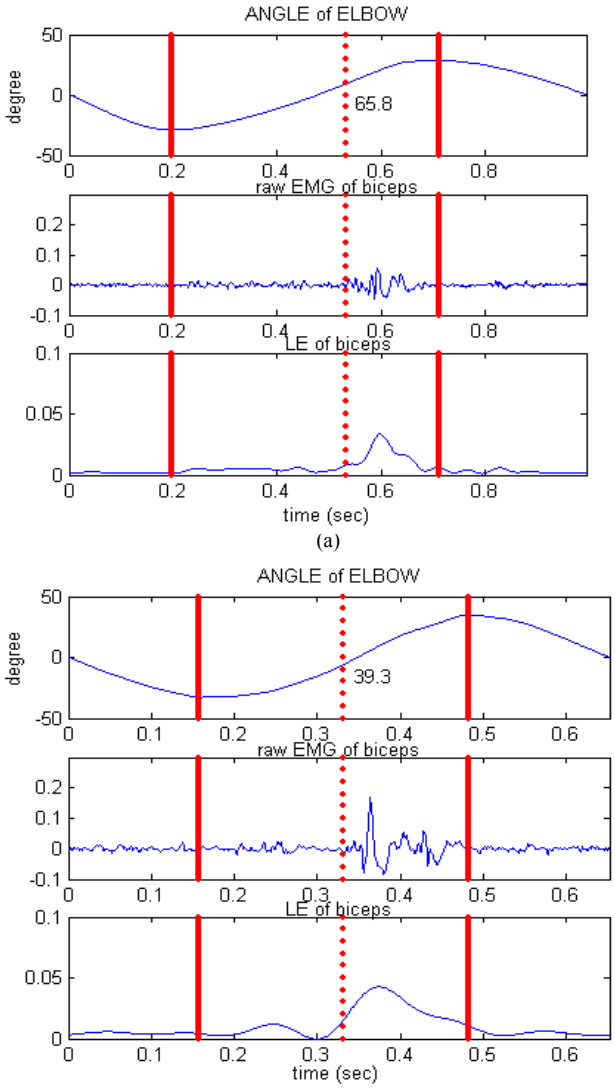

Figure 2. The elbow joint angle (top traces) was used to determine the EMG firing threshold. The middle and bottom traces are the raw EMG and EMG LE for biceps brachii muscle, which was stretched during extension inside two vertical lines. The firing threshold is marked as dotted line. The values shown in top traces are the percentage of full range for the detected firing threshold.

Table 2. Threshold of EMG firing at varied frequencies for all stroke patients.

Subject	MAS	1/3 Hz	1/2 Hz	1 Hz	3/2 Hz
A	1	—	—	96.3	90.8
B	1	—	—	—	81.2
C	1	—	—	59.7	57.3
D	2	99.3	96.1	80.6	67.6
E	3	—	—	54.3	50.0
F	2	—	—	76.2	59.9
G	1	—	—	77.8	79.5
H	3	52.6	41.9	49.1	42.4
I	3	—	83.3	55.1	63.6
J	2	—	70.4	70.5	67.0
K	2	99.2	85.5	73.7	—
L	2	—	80.7	84.7	75.8
M	1	73.3	78.2	72.2	71.9

— denotes not-available data which were resulted from no sufficient stretching velocity (at 1/3 Hz and 1/2 Hz).

Table 3. Correlation analysis between measured parameters (BW, B, EMG threshold) and modified Ashworth scale (MAS).

Variables	Bω1/3	Bω1/2	Bω1	Bω3/2	B	EMG 1	EMG 3/2
Correlation coefficient	0.9001	0.9774	0.9743	0.9998	0.9985	-0.9380	-0.9732

EMG 1 and EMG 3/2 denote the threshold of EMG firing for 1 Hz and 3/2 Hz.

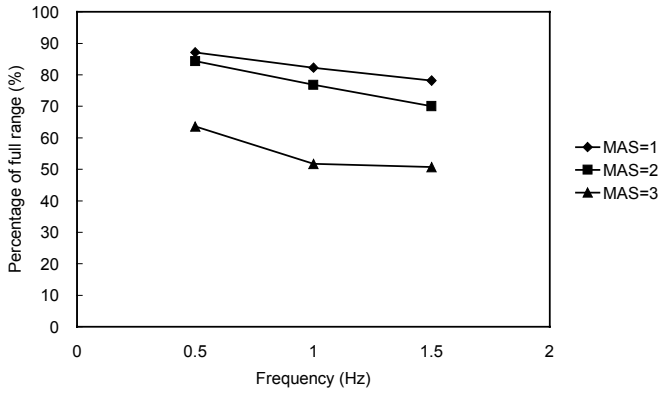

Figure 3. The trend of threshold decreased with increases of stretching frequencies and severity of spasticity. In this plot, the zero percentage is the end position of limited elbow flexion and one hundred percentages indicate the end position of limited elbow extension.

EMG data

In the continuous sinusoid movement, we divided the EMG data into extension and flexion phases, as shown in the vertical lines in Figure 2. The zero position is defined as 90 degrees of elbow flexion; the negative and positive values represent the corresponding flexion and extension movements of upper limb. Table 2 summarizes the EMG firing onsets at four stretching frequencies in subjects with different MAS scales. However, we could not observe clear firing pattern in lower stretch velocity and thus those without clear firing were not included in Figure 3. To observe the EMG firing threshold, Figure 3 summaries the EMG firing thresholds in terms of the percentage of full range of motion. We could find that the decreased trend of firing threshold with respective to the increase of stretch frequency. In addition, the subjects with severe spasticity (high MAS scale) exhibit lower firing threshold.

In order to assess the strength of the relationship between all variables with MAS, the least-squares regression lines were be obtained. And then the correlation coefficients were found from R-square. As expected, the MAS scales were highly positive correlated to biomechanical data (Bω and B) and highly negative correlated to the threshold of EMG firing.

Discussion

The aim of this paper is to record the baseline data of the stroke patients with excessive muscle tone and provide quantitative biomechanical and electrophysiological assessments before BTX-A intervention. Recent research has shown a renewal of interest in evaluating spasticity by biomechanical approach [15]. In our study, biomechanical approach and neurophysiological testing were both taken to assess the condition of spasticity. Similar approach was taken in [12, 16]; however, less consistent results in EMG observations were shown. Other research indicated the changes of EMG and reactive torque were not obvious [12]. We believe that the insensitivity might originate from the suitability of measurement device. In our current approach, a portable spasticity measurement system was adopted which is rather reliable and the measurement device, especially suitable for clinical trials. Besides, we determined the firing threshold of EMG by from EMG LE instead of the raw EMG waveform, amplitude or duration; we could clearly define the onset time of EMG firing. Comparing to amplitude and duration of EMG, onset time determination was more obvious and convenient to analyze.

In biomechanical approach, the viscosity of spastic muscle could be derived from the measured stretch position and reactive torque at varied frequencies. As shown in Figure 1, viscous component value increased with the increase of stretching frequency and was higher in subjects with server spasticity. Besides, we found that the subjects with the same MAS value would show different viscosity (B). It might be due to the inter-subject variability as well as the inadequacy for the scale-based of MAS. In addition, viscous component (Bω) was more obvious at the higher stretch frequencies and correlated very well with severity of spasticity (Table 3). These observations coincided well with our previous results [14].

As indicated in the Figure 2(a) and 2(b) that evident EMG activities can be observed in biceps brachii muscle (the middle vertical dotted line denoting the EMG onset point) when the elbow flexor was stretched during extension. When the muscle was imposed by external stretching, stretch reflex would be elicited and EMG activation might be seen in stretched muscle. Comparing the EMG activation stretching at 1 Hz (Figure 2(a)) with that of 1.5 Hz stretching frequency (Figure 2(b)), we could observe that the EMG activities were more evident and thus the threshold was lower at higher stretching frequency. Figure 3 reveals that the threshold of EMG firing correlated well with the severity of spasticity. It is commonly known that the threshold decreased when the stretching frequency increased and was lower in subjects with higher MAS scale. In our study, the threshold of EMG firing was lower at higher stretch frequencies, which was consistent to the velocity-dependent properties of spasticity. However, the stretching EMG was not evident when the stretch velocity is low or when the degree of spasticity is not severe. Under these circumstances, the utilization of EMG threshold becomes infeasible. It was also known that the 1/3 Hz and 1/2 Hz were not suitable for assessing the subject with mild spasticity, such as whose MAS was 1, because the stretching velocity was too slow to activate muscle spindle. In Pisano's previous study [16], the 100° /s velocity was used as a cut-off value that discriminates between patients and controls. The limited range for elbow movement was 60° , at 1 Hz stretching frequency with an equivalent stretch velocity of 120° /s which is sufficient to elicit stretch reflex. We believe that the EMG threshold can only be used as supportive evidence in addition to the biomechanical measurements.

The changes of the threshold of EMG firing were in accordance with the changes of viscosity value (B) at different subjects with varied MAS values. The trends for threshold of EMG firing and for viscosity value were consistent. It is believed that the Bw and B values could provide more quantitative parameters for assessing the severity of spasticity. In the future, these parameters will also be used to quantify the treatment effect of Botox injection for spasticity reduction.

Conclusion

Quantitative evaluation of the severity of spasticity is essential for the intervention of spasticity. For BTX-A intervention, owing to reliable quantitative evaluation, the effect would be assessed preciously and the suitable dosage might be decided and commended. On the basis of our current results, we can quantify the severity of spasticity in terms of biomechanical as well as electrophysiological assessment in each subject before BTX-A intervention. With these quantitative evaluation technology for spasticity, we could investigate the effect of intervention for spasticity suppression, such as BOTOX® as well as the design of therapeutic plan for each individual with varied degrees of spasticity.

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References

[1] P. Brown, "Pathophysiology of spasticity", *J. Neurol. Neurosurg. Psychiatry*, 57: 737-777, 1994.

[2] G. Sheean, " The pathophysiology of spasticity", *Eur. J. Neurol.*, 9: 3-9, 2002.

[3] B. B. Bhakta, "Management of spasticity in stroke", *British Medical Bulletin*, 56: 476-485, 2000

[4] A.M.O Bakheit, A.F. Thilmann, A.B. Ward, et al, "A randomized, double-blind, placebo-controlled, dose-ranging study to compare the efficacy and safety of three doses of botulinum toxin type A (Dysport) with placebo in upper limb spasticity after stroke", *Stroke*, 31: 2402-2406, 2000.

[5] A.M.O. Bakheit, D.A.H. Badwan, D.L. McLellan, "The effectiveness of chemical neurolysis in the treatment of lower limb spasticity", *Clin. Rehil.* 10: 40-43, 1996.

[6] N.H. Mayer, "Clinicophysiologic concepts of spasticity and motor dysfunction in adults with an upper motoneuron lesion", *Muscle & Nerve*, *Suppl.*, 6: S1-S13, 1997.

[7] L. Vodovnik, B. R. Bowman, and P. Hufford, "Effects of electrical stimulation on spinal spasticity", *Scand. J. Rehab. Med.*, 16: 29-34, 1984.

[8] E. C. Davis, M. P. Branes, "Botulinum toxin and spasticity", *J. Neurol. Neurosurg. Psychiatry*, 69: 143-149, 2000.

[9] M. F. Brin, "Botulinum toxin: chemistry, pharmacology, toxicity, and immunology", *Muscle & Nerve*, *Suppl.,* 6: S1-S23, 1997.

[10] A. M. O. Bakheit, S. Pittock, A. P. Moore, et al, "A randomized, double-blind, placebo-controlled study of the efficacy and safety of botulinum toxin type A in upper limb spasticity in patients with stroke", *Eur. J. Neurol.*, 8: 559-565, 2001.

[11] A. Brashear, M. F. Gordon, E. Elovic, V. D. Kassicieh, et al.,"Intramuscular injection of botulinum toxin for the treatment of wrist and finger spasticity after a stroke", *N. Engl. J. Med.*, 347: 395-399,2002.

[12] A. D. Pandyan, P. Vuadens, F. M. J. van Wijck, et al., "Are we underestimating the clinical efficacy of botulinum toxin (type A)? Quantifying changes in spasticity, strength and upper limb function after injections of BOTOX® to the elbow flexors in a unilateral stroke population", *Clin. Rehibil.*, 16: 654-660, 2002.

[13] B. B. Bhakta, J. A. Cozens, M. A. Chamberlain, J. M. Bamford, "Impact of botulinum toxin type A on disability and carer burden due to arm spasticity after stroke: a randomized double blind placebo controlled trial", *J. Neurol. Neurosurg. Psychiatry*, 69: 217-221, 2000.

[14] H.M. Lee, J.J. Chen, M.S. Ju, C.C. Lin, P. P. Poon, "Validation of Portable Muscle Tone Measurement Device for Quantifying Velocity-Dependent Properties in Elbow Spasticity", *J. electromyogr. kinesiol.*, 2004.

[15] D. G. Kamper, B. D. Schmit, W. Z. Rymer, "Effect of muscle biomechanics on the quantification of spasticity", *Ann. Biomed. Eng.*, 29: 1122-1134, 2001.

[16] F. Pisano, G. Miscio, C. D. Conte, D. Pianca, E. Candeloro, R. Colombo, "Quantitative measures of spasticity in post-stroke patients", *Clin. Neurophysiol.*, 111: 1015-1022, 2000.

[17] L. Q. Zhang, R. Shiavi, M.A. Hunt, J.J. Chen, "Clustering analysis and pattern discrimination of EMG linear envelopes", *IEEE Trans. Biomed.* Eng., 38: 777-784, 1991.