Published in final edited form as:

Clin Biomech (Bristol, Avon). 2017 December ; 50: 110–113. doi:10.1016/j.clinbiomech.2017.10.013.

Assessing muscle compliance in stroke with the Myotonometer

Xiaoyan Li^{a,*}, Henry Shin^a, Ya Zong^a, Sheng Li^a, Ping Zhou^{a,b}

^aDepartment of Physical Medicine and Rehabilitation, University of Texas Health Science Center at Houston, and TIRR Memorial Hermann Research Center, Houston, TX, USA

^bGuangdong Work Injury Rehabilitation Center, Guangzhou, China

Abstract

Background: This study explores changes of the intrinsic biomechanical property in the biceps brachii muscle after a hemispheric stroke using the Myotonometry technique.

Methods: Nineteen subjects with chronic hemiplegia participated in the study. Myotonometer was used to measure tissue displacement when compression force was applied at 8 levels from 2.45 N to 19.6 N. Muscle displacement and compliance were determined and averaged over multiple trials.

Findings: Statistical analysis indicated a significant decrease in muscle displacement and compliance in the spastic muscles compared with the contralateral side (muscle displacements: spastic: 4.51 (0.31) mm, contralateral: 5.74 (0.37) mm, $p < 0.005$; compliance: spastic: 0.17 (0.011) mm/N, contralateral: 0.22 (0.014) mm/N, $p < 0.005$). Correlation analysis, however, did not show any association between clinical assessments and myotonometric measurement ($p > 0.1$).

Interpretation: Alterations of muscle compliance in the spastic side reflect changes in the contractile or intrinsic mechanical properties after a stroke. Findings of the study have demonstrated high sensitivity and effectiveness of the Myotonometer in assessing muscle compliance changes.

Keywords

Stroke; Muscle compliance; Myotonometer; Spasticity; Area under the curve (AUC)

1. Introduction

Spasticity or hypertonia, characterized by the velocity-dependent resistance to passive stretch, has been commonly observed in a number of neurological disorders including cerebral palsy, multiple sclerosis, spinal cord injury, and stroke (Burke, 1988; Lance, 1980; Leonard et al., 2001; Mirbagheri et al., 2001; Sanger et al., 2003; Sinkjaer et al., 1993). The origins of spasticity remain controversial, possibly from hyper-active stretch reflexes, abnormal intrinsic properties of the contractile apparatus, or mechanical (viscoelastic) properties of the passive tissues (Katz and Rymer, 1989; Mirbagheri et al., 2001; Sinkjaer and Magnussen, 1994).

*Corresponding author at: TIRR Research Center, 1333B Moursund St, Houston, TX 77030, USA. Xiaoyan.li@uth.tmc.edu (X. Li).

Quantification of spasticity involves subjective and objective measurements which provide important information on the pathophysiological changes of nervous system and muscle tissues after an injury. The Modified Ashworth Scale (MAS) is frequently used in clinics, which consists of a 6-point ordinal scale to differentiate levels of spasticity (Bohannon and Smith, 1987). The scale of MAS, however, is subjective and tends to cluster in the lower ranges, leading to compromised interrater repeatability, accuracy and reliability (Aarrestad et al., 2004; Blackburn et al., 2002; Leonard et al., 2001). Objective evaluation of spasticity involves biomechanical and electrophysiological measurements which record joint torques, voluntary muscle activities, and muscle responses at rest or during contractions. H reflex, the ratio of H:M, and F wave are typical electrophysiological measures for assessing motoneuron excitability and spasticity (Argyriou et al., 2010; Huang et al., 2006; Pizzi et al., 2005).

Conventional biomechanical techniques utilize a servo-controlled motor to measure joint resistance torque with respect to angular displacement (Katz and Rymer, 1989; Mirbagheri et al., 2001; Rydahl and Brouwer, 2004; Toft et al., 1991). By stretching the muscle at a constant velocity within a preset range and inducing perturbations or electrical stimulations, it is possible to separate total mechanical stiffness into intrinsic and reflex components (Rydahl and Brouwer, 2004; Sinkjaer et al., 1988). Those components are interdependent and may individually contribute or co-contribute to muscle stiffness. Given that equipment setup and data collection are complicated the potential applications of the techniques in clinical facilities are restricted.

Myotonometry provides an alternative and more compact means to assess muscle stiffness or compliance (inverse of stiffness). Presently, there are two representative types of devices that quantify muscle compliance. One is Myoton, which applies a brief mechanical impulse to the skin and records the oscillations of muscle responses (Chuang et al., 2012). The other type is Myotonometer, which measures the amount of muscle deformation to a number of perpendicularly applied forces (Leonard, 1994; Leonard et al., 2001). Myotonometer is used to examine muscles in a relaxed position or under an isometric contraction (Bizzini and Mannion, 2003; Hung et al., 2010; Leonard et al., 2003; Marusiak et al., 2010). It requires minimal setup time and can achieve high intra- and inter-rater reliabilities (Aarrestad et al., 2004; Leonard et al., 2003). Therefore, it may have promising applications for evaluation of muscle conditions in the clinical setting.

Both myotonometric devices have been used to examine muscle compliance in spastic muscles in previous studies. The findings, however, are different (Chuang et al., 2012, 2013; Leonard et al., 2001; Rydahl and Brouwer, 2004). For example, substantial changes of the elasticity and stiffness in hand flexors were observed in stroke using the Myoton device (Chuang et al., 2012). On the other hand, no significant changes of compliance are reported between the spastic and healthy groups using the Myotonometer (Leonard et al., 2001; Rydahl and Brouwer, 2004). According to the literature, muscle displacement occurs after the full compression of subcutaneous layers (Rydahl and Brouwer, 2004). Previous studies focus on the overall tissue displacement without distinguishing subcutaneous and muscle displacements (Leonard et al., 2001).

In this study, we applied a two-layer spring model to evaluate the muscle displacement and the overall tissue displacement (Horikawa et al., 1993). We identified significant decrease of muscle compliance in the spastic side. The change of muscle compliance, however, was not correlated with the MAS.

2. Methods

2.1. Subjects

Nineteen subjects (10 Female, 9 Male) with chronic hemiplegia participated in the study. All subjects survived from one incidence of stroke at least 6 months prior to the study and were free of any other known neurological disorders. The average age was 62 (10) years (mean (standard deviation)) and the time course since stroke varied from 6 months to 13 years. All subjects signed consent approved by the Institutional Review Board of University of Texas Health Science Center at Houston prior to any experiments. The MAS test was performed on the spastic elbow flexors after the experiment. Clinical assessments and demographic information of subjects are presented in Table 1.

2.2. Experiments

Subjects were seated comfortably in a chair, and instructed to maintain an upright sitting posture during the experiment. In particular, the shoulder was slightly abducted and the elbow was laid on a height-adjustable table in 90-degree flexion. The Myotonometer (Neurogenic Technologies, Missoula, MT, USA) is a hand-held electronic device that consists of an inner metal probe, an outer plastic sleeve, and a linear array of force transducers. The probe and the plastic sleeve are on the same surface when no compressing force is applied. Prior to the test, the probe and the sleeve were placed over the muscle bulk perpendicular to the bone. During the experiment, the experimenter exerted continuous compression to the muscle until a sound was heard, which indicated the 19.6 N of force was met. The resistance that the muscle generated against probe penetration was sampled at 8 force levels from 2.45 N to 19.6 N at increments of 2.45 N. The corresponding tissue displacement was measured as the distance between the inner probe and the outer plastic sleeve. Each trial takes approximately 1 s and each muscle was tested 8 times in the experiment. The test was performed bilaterally over the biceps brachii muscles and all experiments were conducted by the same experimenter.

2.3. Data analysis

Offline analysis of the Myotonometer data was conducted in MATLAB® (MathWorks, Natick, MA). Results of the Myotonometer recording from a representative participant are presented in Fig. 1.

As seen in the figure, tissue displacement changes nonlinearly with force. For example, when compression force increases from 2.45 N to 5.51 N, tissue displacement changes from 8.54 mm to 10.5 mm in the spastic side. As compression force continues to increase from 5.51 N to 19.6 N, the second part of tissue displacement changes from 10.5 mm to 12.47 mm (Fig. 1). The different slopes of displacement-force may suggest two different types of tissues (i.e. subcutaneous tissue and muscle) (Horikawa et al., 1993; Sakai et al., 1995).

Therefore, a two-segment model was used to approximate the nonlinear displacement-force relation. As illustrated, the turning point of the two segments occurs around 7.35 N or lower (Fig. 1).

Muscle displacement in this study was assumed to occur when resistance ranged from 9.8 N to 19.6 N. The area under the curve of the muscle (AUC_muscle) was defined as the sum of the muscle displacement subtracted by the displacement at 9.8 N

($AUC_{\text{muscle}} = \sum_{i=9.8N}^{19.6N} \text{dist}_i - \text{dist}_{9.8N}$). In addition, segmented regression analysis was applied to test the linear relation between muscle displacement and force (from 9.8 N to 19.6 N). Once confirmed, muscle compliance was derived from the slope of the muscle displacement-force function. The area under the curve of all tissues (AUC_all) was calculated as the sum of tissue displacement at all force levels with respect to the baseline displacement at 2.45 N ($AUC_{\text{all}} = \sum_{i=2.45N}^{19.6N} \text{dist}_i - \text{dist}_{2.45N}$). The area under the curve for the subcutaneous tissues (AUC_sub) was calculated as:

$$AUC_{\text{sub}} = \sum_{i=2.45N}^{7.35N} \text{dist}_i - \text{dist}_{2.45N}$$

2.4. Statistical analysis

All data were screened for outliers and normality of distribution. No significant deviations were found. Paired *t*-test was applied to compare the differences between spastic and contralateral muscles for Myotonometer variables (AUC_all, AUC_sub, AUC_muscle, and muscle compliance). In addition, Pearson correlation analysis was used to assess linear correlations between time course of stroke and Myotonometer variables. Due to the ordinal nature of the MAS, Spearman ρ coefficients were computed to examine whether the MAS was correlated with Myotonometer variables. All data are presented in the format of mean (standard error) unless specified. Statistical significance was defined as $p < 0.05$.

3. Results

The relation of resistance and tissue displacement was illustrated in Fig. 1 from a representative subject. Tissue displacement ranged from 8.54 mm to 12.47 mm in the spastic biceps brachii and from 9.09 mm to 15.14 mm in the contralateral side with the resistance increasing from 2.45 N to 19.6 N. The areas under the curve for all tissues (AUC_all) were 20.28 mm and 29.55 mm in the spastic and contralateral sides respectively. The areas for subcutaneous tissue were 3.92 mm and 4.88 mm in the spastic and contralateral muscles. The areas of the muscle (AUC_muscle) were 3.55 mm and 6.95 mm in the spastic and contralateral muscles, respectively. Regression analysis confirmed a significant linear relation between muscle displacement and resistance from 9.8 N to 19.6 N for all muscles. Bilateral muscle compliance were 0.14 mm/N and 0.26 mm/N for the spastic and contralateral sides, respectively (spastic: $r^2 = 0.99$, $p < 0.001$; contralateral: $r^2 = 0.98$, $p < 0.002$).

Comparison of the AUC_muscle and muscle compliance between the spastic and contralateral muscles indicated a significant reduction of values in the spastic muscles (Fig. 2). AUC_muscle: spastic: 4.51 (0.31) mm, contralateral: 5.74 (0.37) mm, $p < 0.005$. Muscle

compliance: spastic: 0.17 (0.011) mm/N, contralateral: 0.22 (0.014) mm/N, $p < 0.005$). Comparison of AUC for all tissues showed similar results (AUC_all: spastic: 27.70 (1.71) mm, contralateral: 33.07 (1.45) mm, $p < 0.01$). No significant differences between two sides were observed in the AUC for subcutaneous tissues (AUC_sub: spastic: 5.05 (0.31) mm, contralateral: 5.48 (0.27) mm, $p = 0.1$).

Clinical data is presented in Table 1. Spearman ρ tests did not confirm any significant correlations between the MAS and Myotonometer measures. Similarly, the Pearson test did not find any linear relation between duration of stroke and Myotonometer variables ($p > 0.1$).

4. Discussion

This study used the Myotonometry technique to explore changes of mechanical properties of the muscle subsequent to a hemiplegic stroke. Significant decreases of muscle compliance and displacements were observed in the spastic muscles as compared with that of the contralateral side. Such changes, however, were not associated with the clinical assessments including MAS or time course of stroke.

4.1. Modifications in muscle compliance

This study reported significantly reduced muscle compliance in resting spastic muscles. Modifications of muscle mechanical properties were also found in a previous study that examined the elasticity and stiffness of hand flexors in stroke using Myoton device (Chuang et al., 2012). Higher passive stiffness (or increased hardness, reduced compliance) was observed in patients with Parkinson disease or with tension-type headaches (Marusiak et al., 2010; Sakai et al., 1995). In the tension related headaches, abnormal hardness was interpreted as circulatory congestion and swelling of the muscle tissue caused by sustained contractions (Sakai et al., 1995). The mechanisms underlying reduced compliance in spastic muscles, however, remain unclear (Aarrestad et al., 2004). According to the study that compared Myotonometer measurements with conventional stretching findings, the altered compliance in the spastic muscle presumably reflects changes in contractile or intrinsic mechanical properties rather than the passive stiffness or reflex stiffness of the muscle (Rydahl and Brouwer, 2004). The mechanical modifications of muscle are likely affected by structural changes in the muscle such as fiber atrophy, changes in composition of different muscle fiber types, fascicle length, pennation angle, and etc. (Chuang et al., 2012; Gao et al., 2009).

On the other hand, insignificant changes of compliance between the spastic and healthy muscles were also observed (Leonard et al., 2001). The conclusion was drawn from comparison of overall tissue displacements at single force levels. In our study, muscle displacement was separated from the overall tissue displacement, which minimized the influence of subcutaneous displacement and may account for the different findings in our study and the previous study.

Myotonometer measures tissue displacement as a function of perpendicular compressing force. In general, larger tissue displacement appears in the more superficial tissues before

muscle is compressed. The exact force for full compression of the subcutaneous tissues is unknown. According to a previous report, 2.45 N is sufficient to fully compress the subcutaneous layer of the lower limb muscles (Rydahl and Brouwer, 2004). In our study the force is around 7.35 N or lower. Note that estimation of the connective compliance was not performed in this study due to the limited force levels.

4.2. Clinical relevance

Correlations between the Myotonometer measurements and the MAS scores were previously reported in contracted muscles with spasticity (Leonard et al., 2001; Rydahl and Brouwer, 2004). Such correlations, however, were not observed in our study. According to the literature, tissue displacement-force relation may demonstrate differently depending on whether the muscle is relaxed or contracted (Hung et al., 2010; Leonard et al., 2001; Rydahl and Brouwer, 2004). The difference of our study and previous findings could lie in that muscle compliance in our study was evaluated in the resting state whereas previous findings were obtained from voluntary contractions. In addition, a majority of the MAS scores were < 2 (15 out of 19 subjects) in this study, which made it difficult to discriminate levels of spasticity in individuals and may partially lead to the insignificant results of the study.

4.3. Limitation of the study

In this study, examination of muscle compliance was based on subjects who had mild to moderate spasticity with only one incidence of stroke. Since there are many factors that influence muscle tone including the location of brain lesion, severity of the stroke, and body position (Chuang et al., 2013), current conclusions may limit the generation of the findings to patients with more severe spasticity or having multiple strokes. Also, the result of reduced muscle compliance was based on tests of the biceps brachii muscles. As Myotonometer is designated to evaluate superficial muscles, it remains unknown whether deep muscles such as brachialis, another prime contributor to elbow flexion, have similar changes after a stroke.

4.4. Conclusions

To summarize, this study assessed mechanical changes in muscles and identified reduced muscle compliance in the spastic side. Correlation analysis, however, did not confirm any significant relation between clinical assessments and Myotonometer measures. Future work that integrates the Myotonometer and conventional biomechanical techniques to quantify both passive and reflex torques in stroke may provide better understanding of clinical applications of Myotonometer in spastic muscles.

Acknowledgement

This work was supported by the National Institutes of Health (Grant #: R01NS080839), the Guangzhou Science and Technology Program under Grant 20170403003 and the Memorial Hermann Foundation.

References

- Aarrestad DD, Williams MD, Fehrer SC, Mikhailenok E, Leonard CT, 2004 Intraand interrater reliabilities of the Myotonometer when assessing the spastic condition of children with cerebral palsy. *J. Child Neurol.* 19 (11), 894–901. [PubMed: 15658795]

- Argyriou AA, Karanasios P, Makridou A, Makris N, 2010 F-wave characteristics as surrogate markers of spasticity in patients with secondary progressive multiple sclerosis. *J. Clin. Neurophysiol* 27 (2), 120–125. [PubMed: 20505376]
- Bizzini M, Mannion AF, 2003 Reliability of a new, hand-held device for assessing skeletal muscle stiffness. *Clin. Biomech. (Bristol, Avon)* 18 (5), 459–461.
- Blackburn M, van Vliet P, Mockett SP, 2002 Reliability of measurements obtained with the modified Ashworth scale in the lower extremities of people with stroke. *Phys. Ther* 82 (1), 25–34. [PubMed: 11784275]
- Bohannon RW, Smith MB, 1987 Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys. Ther* 67 (2), 206–207. [PubMed: 3809245]
- Burke D, 1988 Spasticity as an adaptation to pyramidal tract injury. *Adv. Neurol* 47,401–423. [PubMed: 3278524]
- Chuang LL, Wu CY, Lin KC, 2012 Reliability, validity, and responsiveness of myotonometric measurement of muscle tone, elasticity, and stiffness in patients with stroke. *Arch. Phys. Med. Rehabil* 93 (3), 532–540. [PubMed: 22222143]
- Chuang LL, Lin KC, Wu CY, Chang CW, Chen HC, Yin HP, Wang L, 2013 Relative and absolute reliabilities of the myotonometric measurements of hemiparetic arms in patients with stroke. *Arch. Phys. Med. Rehabil* 94 (3), 459–466. [PubMed: 22960277]
- Gao F, Grant TH, Roth EJ, Zhang LQ, 2009 Changes in passive mechanical properties of the gastrocnemius muscle at the muscle fascicle and joint levels in stroke survivors. *Arch. Phys. Med. Rehabil* 90 (5), 819–826. [PubMed: 19406302]
- Horikawa M, Ebihara S, Sakai F, Akiyama M, 1993 Non-invasive measurement method for hardness in muscular tissues. *Med. Biol. Eng. Comput* 31 (6), 623–627. [PubMed: 8145589]
- Huang CY, Wang CH, Hwang IS, 2006 Characterization of the mechanical and neural components of spastic hypertonia with modified H reflex. *J. Electromyogr. Kinesiol* 16 (4), 384–391. [PubMed: 16253519]
- Hung CJ, Hsieh CL, Yang PL, Lin JJ, 2010 Relationships between posterior shoulder muscle stiffness and rotation in patients with stiff shoulder. *J. Rehabil. Med* 42 (3), 216–220. [PubMed: 20411215]
- Katz RT, Rymer WZ, 1989 Spastic hypertonia: mechanisms and measurement. *Arch. Phys. Med. Rehabil* 70 (2), 144–155. [PubMed: 2644919]
- Lance JW, 1980 Symposium synopsis In: Feldman RG, Koella WP (Eds.), *Spasticity:Disordered Motor Control*. Year Book, Chicago, pp. 485–494.
- Leonard CT, 1994 Motor behavior and neural changes following perinatal and adult-onset brain damage: implications for therapeutic interventions. *Phys. Ther* 74 (8), 753–767. [PubMed: 8047563]
- Leonard CT, Stephens JU, Stroppel SL, 2001 Assessing the spastic condition of individuals with upper motoneuron involvement: validity of the myotonometer. *Arch. Phys. Med. Rehabil* 82 (10), 1416–1420. [PubMed: 11588747]
- Leonard CT, Deshner WP, Romo JW, Suoja ES, Fehrer SC, Mikhailenok EL, 2003 Myotonometer intra- and interrater reliabilities. *Arch. Phys. Med. Rehabil* 84(6), 928–932. [PubMed: 12808553]
- Marusiak J, Kisiel-Sajewicz K, Jaskolska A, Jaskolski A, 2010 Higher muscle passive stiffness in Parkinson's disease patients than in controls measured by myotonometry. *Arch. Phys. Med. Rehabil* 91 (5), 800–802. [PubMed: 20434620]
- Mirbagheri MM, Barbeau H, Ladouceur M, Kearney RE, 2001 Intrinsic and reflex stiffness in normal and spastic, spinal cord injured subjects. *Exp. Brain Res.* 141 (4), 446–459. [PubMed: 11810139]
- Pizzi A, Carlucci G, Falsini C, Verdesca S, Grippo A, 2005 Evaluation of upper-limb spasticity after stroke: a clinical and neurophysiologic study. *Arch. Phys. Med. Rehabil* 86 (3), 410–415. [PubMed: 15759220]
- Rydahl SJ, Brouwer BJ, 2004 Ankle stiffness and tissue compliance in stroke survivors: a validation of Myotonometer measurements. *Arch. Phys. Med. Rehabil* 85(10), 1631–1637. [PubMed: 15468023]
- Sakai F, Ebihara S, Akiyama M, Horikawa M, 1995 Pericranial muscle hardness in tension-type headache. A non-invasive measurement method and its clinical application. *Brain* 118 (Pt 2), 523–531. [PubMed: 7735892]

- Sanger TD, Delgado MR, Gaebler-Spira D, Hallett M, Mink JW, Task Force on Childhood Motor D, 2003 Classification and definition of disorders causing hypertonia in childhood. *Pediatrics* 111 (1), e89–97. [PubMed: 12509602]
- Sinkjaer T, Magnussen I, 1994 Passive, intrinsic and reflex-mediated stiffness in the ankle extensors of hemiparetic patients. *Brain* 117 (Pt 2), 355–363. [PubMed: 8186961]
- Sinkjaer T, Toft E, Andreassen S, Hornemann BC, 1988 Muscle stiffness in human ankle dorsiflexors: intrinsic and reflex components. *J. Neurophysiol* 60 (3), 1110–1121. [PubMed: 3171659]
- Sinkjaer T, Toft E, Larsen K, Andreassen S, Hansen HJ, 1993 Non-reflex and reflex mediated ankle joint stiffness in multiple sclerosis patients with spasticity. *Muscle Nerve* 16 (1), 69–76. [PubMed: 8423835]
- Toft E, Sinkjaer T, Andreassen S, Larsen K, 1991 Mechanical and electromyographic responses to stretch of the human ankle extensors. *J. Neurophysiol* 65 (6), 1402–1410. [PubMed: 1875249]

Fig. 1.

Myotonometric measurement from a representative subject. Tissue displacement was averaged over eight trials and marked (dots: spastic, open circles: contralateral). The grey areas indicated the range of one standard deviation from the mean values.

Fig. 2.
Comparisons of Myotonometer variables between the spastic and contralateral muscles.

Table 1

Subjects' demographic and clinical data.

ID	Paretic	Age (year)	Gender	MAS	Duration (month)
1	L	58	F	1 +	6
2	L	77	M	0	16
3	L	58	F	1	88
4	L	71	M	0	76
5	R	61	F	1	91
6	R	53	M	1 +	52
7	L	65	M	0	51
8	R	52	F	1 +	86
9	R	69	M	1 +	75
10	R	63	F	1	41
11	R	79	M	1	64
12	L	62	F	0	164
13	R	67	F	1	101
14	R	37	F	2	88
15	L	65	M	1 +	24
16	L	71	M	1 +	60
17	R	49	F	3	17
18	L	55	F	2	82
19	R	67	M	3	21

MAS: Modified Ashworth Scale; Duration: months since stroke.