

Measuring muscle tone with isokinetic dynamometer technique in stroke patients

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Summary

Study aim: Increased muscle tone, a common consequence of stroke, has neural and non-neural components. Spasticity is related to the neural component. Non-neural resistance arises from passive stiffness. This study was designed to assess the feasibility of using isokinetic dynamometry to evaluate wrist flexor muscle spasticity in stroke patients.

Materials and methods: Twenty-six patients with hemiplegia in the subacute phase of stroke participated in this study. An isokinetic dynamometer was used to stretch wrist flexor muscles at four velocities of 5, 60, 120 and 180°/s on both the paretic and non-paretic sides. Peak torque at the lowest speed (5°/s) and reflex torque at the three higher speeds were quantified. Peak torque at the lowest speed was attributed to the non-neural component of muscle tone, and was subtracted from the torque response at higher velocities to estimate reflex torque (spasticity). Data from the two sides were compared.

Results: There was no significant difference in peak torque between the paretic (2.47 ± 0.22 N·m) and non-paretic side (2.41 ± 0.28 N·m) at the lowest velocity of 5°/s ($p=0.408$). However, compared to the non-paretic side, the paretic side showed higher reflex torque ($p<0001$), and reflex torque increased rapidly with increasing velocity ($p < 0.05$).

Conclusion: The isokinetic dynamometer distinguished spasticity from the non-neural component and showed higher reflex torque on the paretic side compared to the non-paretic side. This instrument is potentially useful to assess the efficacy of therapeutic interventions aimed at modifying spasticity.

Keywords: Hemiplegia – Muscle tone – Torque – Reflex– Stiffness

Introduction

Stroke is the third leading cause of death, and is the number one cause of long-term disability in the adult population [32]. The incidence of stroke has increased dramatically among younger adults, with 31% of all strokes occurring in people younger than 65 years [5]. Despite comprehensive training, most stroke survivors endure upper extremity impairment [27] and 70% of them have impaired hand motor function [6]. Although several positive and negative symptoms contribute to the resulting motor dysfunction, there is a substantial focus on spasticity, as one of the positive symptoms [25]. Nearly half of all patients with stroke develop moderate to severe spasticity

in their upper limbs, especially in distal muscles [37, 39]. Spasticity may appear within a week after the stroke [24]. A higher prevalence of spasticity has been reported in patients with high levels of motor impairment [3]. Uncontrolled spasticity may lead to serious complications including muscle contracture, reduced range of motion, abnormal posture and disturbed motor performance [17]. Spasticity is characterized by velocity-dependent excessive resistance of the muscle to passive stretching [18]. Increased resistance to passive stretch has neural and non-neural components [15]. The neural component is induced by stretch reflex activity, and is velocity dependent, whereas non-neural resistance arises from passive stiffness due to changes in the intrinsic properties of the muscle itself that are not usually velocity dependent [15]. Measurements of

spasticity should thus be aimed at the neural component rather than the non-neural component [22].

The contribution of spasticity and passive stiffness to increased muscle tone is variable in the post-stroke period. Spasticity develops and peaks at 1–3 months after the stroke, and the muscular component may increase over time, thus contributing to increased muscle tone at 6 months post-stroke [17]. Reflex activity is significantly reduced after 1 year or longer [36]. The ability to differentiate spasticity from passive stiffness is important in evaluating the effectiveness of different treatments and choosing the best option for each patient. For example, treatments such as botulinum toxin target spasticity, whereas treatments such as casting or stretching target passive stiffness [38].

Clinically, spasticity is usually assessed with the Modified Ashworth Scale (MAS), in which the examiner estimates muscle resistance by moving a joint based on an ordinal scale [2]. However, its validity and reliability as a measure of spasticity have been questioned [7, 29]. In addition, the MAS cannot distinguish between neural and non-neural components [40]. Because the MAS does not take into account the speed dependence of spasticity [31], this scale can be effectively used to assess resistance to passive movement rather than spasticity [29]. The Modified Tardieu Scale (MTS), another clinical scale for spasticity assessment, considers velocity-dependent characteristics of spasticity [30], but it is affected by the operator's skill in manually imposing angular velocity [23]. Therefore, its reliability has been questioned [12].

To overcome the limitations of clinical approaches, a variety of neurophysiological and biomechanical techniques have been developed. One of the proposed instrumental tests for assessing spasticity is isokinetic dynamometry (ID). This tool enables the investigator to standardize both velocity and angle of motion, and to objectively record the amount of force generated by the muscles. Moreover, operation and interpretation of the results are simple, and the procedure can be applied to a variety of muscles [16]. ID measures the amount of resistive torque generated by the muscle at different constant angular velocities [35]. Earlier studies that have tried to determine whether ID is appropriate for quantifying spasticity have mostly focused on patients with chronic stroke, and have reported spasticity to be speed dependent in elbow flexor and knee extensor muscles [4, 10, 35]. However, there is a lack of evidence regarding the measurement of spasticity with ID in patients with subacute stroke. In this context it is useful to recall that spasticity is more prominent in the subacute phase of stroke, and is the main determinant of motor impairment, which can lead in turn to increasing muscle tone in the chronic phase of stroke due to immobilization [18]. In addition, it has been shown that improvements in spasticity are more effective in improving function in subacute

patients than in chronic patients [21]. An additional consideration is that spasticity of the wrist flexor muscles is a critical problem in many stroke patients [28].

Therefore the aim of the study was to determine the feasibility of using ID to quantify wrist flexor muscle spasticity in patients with subacute stroke. We hypothesized that ID would distinguish between spasticity and the non-neural contribution to muscle tone.

Material and methods

A cross-sectional study was undertaken in a single session to measure the degree of wrist flexor spasticity in survivors of a subacute stroke. The study protocol was approved by the ethics committee of Shiraz University of Medical Sciences. Written informed consent was obtained from all participants prior to the measurements.

Participants

The participants were 83 patients with stroke who were admitted to neurorehabilitation clinics affiliated with Shiraz University of Medical Sciences in Shiraz, Iran, and who were screened with inclusion and exclusion criteria (Fig. 1). The inclusion criteria were: age 18 years or older, hemiplegia secondary to a single hemorrhagic or ischemic stroke, time elapsed since the stroke less than 6 months, a MAS score ≥ 1 for muscle tone in the wrist flexor muscles in the paretic arm, and the ability to sit safely and comfortably on a chair. Participants were excluded if they had other neurological conditions, cognitive impairment or deficits in language perception that might interfere with their ability to understand the instructions, and a history of anti-spasticity medication including nerve blockers or botulinum toxin injection. A total of 26 patients (13 men, 13 women) with a mean age of 51.38 ± 12.64 years (range 28–73 years), mean height of 167.42 ± 5.66 cm (157–178 cm) and mean weight of 68.77 ± 7.19 kg (range 60–83 kg) participated in this study. The mean time elapsed since the stroke was 98.77 ± 34.26 days (range 35–147 days). 16 patients had left-side hemiplegia and 10 patients had right-side hemiplegia. The type of stroke was ischemic in 23 patients and hemorrhagic in 3 patients. Mean Fugl-Meyer Assessment (FMA) score for upper limb motor impairment was 22.54 ± 9 (range 4–38). Grip strength as measured with an electronic dynamometer and expressed as a percentage of grip strength on the non-paretic side was 8.37 ± 4.5 newtons (range 0–18.28 N). Mean post-stroke rehabilitation consisted of 47.77 ± 21.81 sessions (range 10–90 sessions).

Procedures

Spasticity in wrist flexor muscles was objectively measured with a Biodex system 4 (Biodex Medical System,

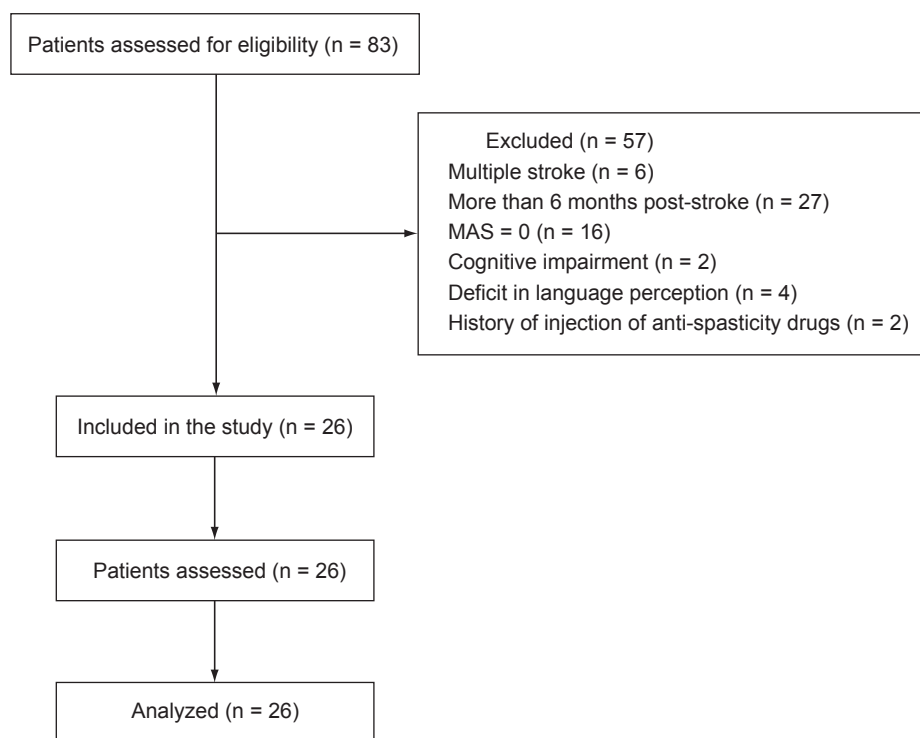


Fig. 1. Flow chart of study implementation. MAS, Modified Ashworth Scale

Shirley, NY, USA) isokinetic dynamometer. The participants were asked to sit on an isokinetic chair and were secured on the chair with a 3-point belt. Both paretic and non-paretic sides were tested in random order. The participant's forearm on the test side was pronated and rested on a flat surface attached to the chair. The shoulder was positioned at about 0° to 5° flexion and 10° to 15° abduction. The elbow was positioned at 90° flexion. The participants gripped the wrist attachment of the dynamometer. Because of the grip strength deficit in the paretic hand,

the participants' fingers on the paretic side were strapped around the handle with a glove in order to ensure that only their wrist joint was free to move (Fig. 2) [26]. The center of rotation of the dynamometer was visually aligned with the axis of the wrist joint (an imaginary line from the ulnar process to the radial styloid process). The wrist joint was passively moved in a total range of 120° from 60° wrist flexion to 60° wrist extension. Four velocities of 5, 60, 120 and 180°/s were used with three trials at each velocity. Slow velocity (5°/s) passive movement was used

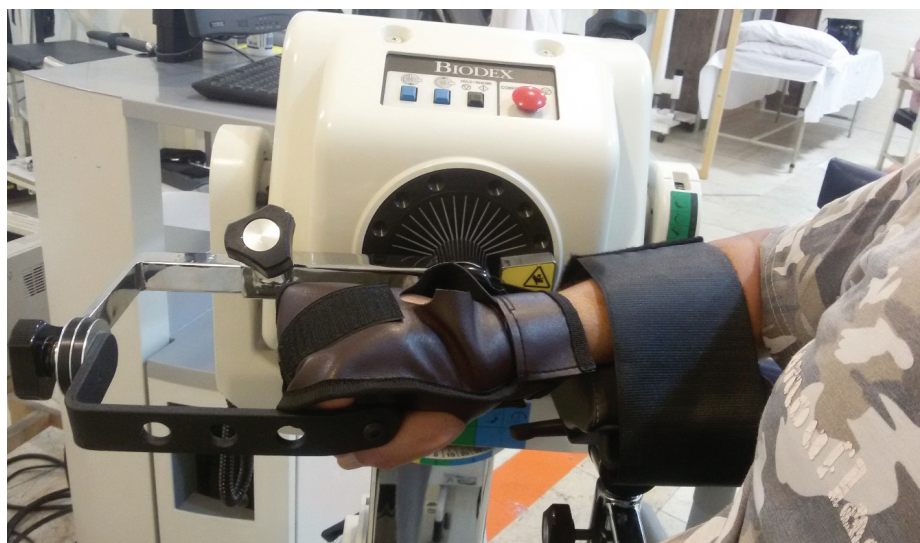


Fig. 2. Measurement of wrist flexor muscle spasticity with isokinetic dynamometry in the paretic hand

Table 1. Comparison of resistive torque (N·m) between the paretic and non-paretic side

Velocity	Paretic side	Non-paretic side	Mean difference (95%CI)	p value
5°/s	2.47 ± 0.22	2.41 ± 0.28	0.06 (−0.08–0.21)	0.408
60°/s	0.88 ± 0.39	0.01 ± 0.13	0.86 (0.7–1.02)	<0.001
120°/s	1.08 ± 0.43	0.03 ± 0.11	1.04 (0.87–1.23)	<0.001
180°/s	1.24 ± 0.40	0.04 ± 0.12	1.2 (1.03–1.37)	<0.001

Values are means ± standard deviation; CI – Confidence interval.

to minimize the reflex response during passive movement [1, 9]. Then the faster movement velocities (three trials per velocity) were tested in 3 different sequence orders to minimize any bias associated with an order effect: (a) 5°/s, 60°/s, 120°/s, 180°/s; (b) 5°/s, 180°/s, 120°/s, 60°/s; and (c) 5°/s, 120°/s, 60°/s, 180°/s [10, 22]. An interval of 30 s was allowed between sets of trials to avoid adaptation and minimize the influence of stretch history on the responses to subsequent stretches, and the time interval between each set of different velocities was 60 s [1, 10]. The participants were instructed to remain relaxed and not to assist or resist the passive movement of their hand. For familiarization and warm up, the wrist was moved through the range of motion 3 times at a velocity of 5°/s. After 5 min, the test movements were performed. Resistance to passive movements during wrist extension was recorded as resistive torque in newton meters (N·m). For each participant, peak resistive torque was calculated across all three trials for each velocity.

Torque at the slowest velocity movement determined passive stiffness of the wrist flexor muscles (non-neural component). It has been demonstrated that velocities less than 25°/s do not trigger stretch reflexes; however, at higher velocities the stretch reflex is easily activated

[34]. Spasticity or stretch reflex torque was calculated by subtracting the response torque at 5°/s from the responses recorded at 60, 120 and 180°/s. These higher velocity movements were used to evaluate the velocity dependence of spasticity [9, 1]. Reflex torque has been reported to be a reliable and clinically acceptable parameter to quantify spasticity [13].

Statistical analysis

All analyses were done with SPSS version 17 (SPSS, Inc., Chicago, IL, USA). Normal distribution of the data was verified with the Shapiro–Wilk test. Descriptive statistics (mean ± standard deviation) were compiled and reported for all data. Resistive torque (at 5°/s) and reflex torque (at 60, 120 and 180°/s) were compared between the paretic and non-paretic side at each movement velocity with the paired t-test. Mean differences were calculated and are reported with their 95% confidence intervals (95% CI). The effect of velocity increments on reflex torque on the paretic and non-paretic side was analyzed with repeated measures one-way analysis of variance (ANOVA). Statistical significance was set at $p < 0.05$.

Results

There was no significant difference in peak torque between the paretic side (2.47 ± 0.22 N·m) and non-paretic side (2.41 ± 0.28 N·m) at the slowest velocity of 5°/s ($p = 0.408$). However, reflex torque was higher at all velocities on the paretic side compared to the non-paretic side ($p < 0.001$) (Table 1). Repeated measures ANOVA showed a significant effect of velocity 60, 120, 180°/s $\times$ side paretic, non-paretic on reflex torque ($F = 17.03$; $p < 0.001$, partial eta squared = 0.8), indicating significant differences in the changes in reflex torque during velocity increments between the paretic and non-paretic side (Fig. 3). Reflex torque increased significantly as velocity increased to each successive angular velocity on the paretic side ($p < 0.05$). However, on the non-paretic side, no significant changes in reflex torque were observed as velocity increased ($p = 0.248$) (Fig. 3).

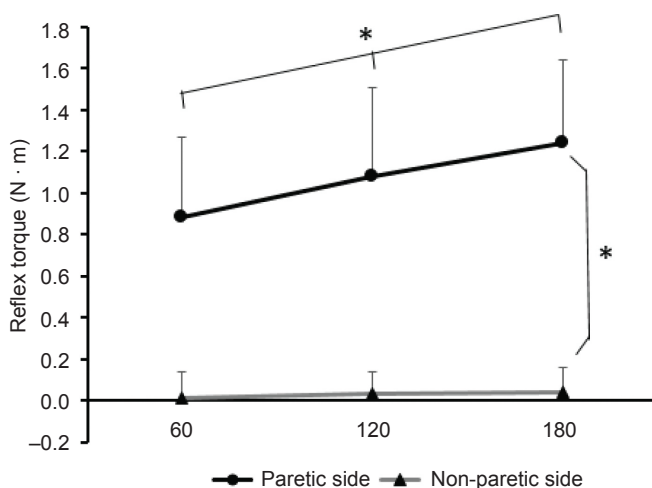


Fig. 3. Changes in reflex torque with increasing velocity. Error bars represent standard deviations. * – Significant difference

Discussion

This study examined spasticity in the wrist flexor muscles with a biomechanical technique based on ID. The computer-assisted device enables investigators to standardize both velocity and range of motion, and quantify spasticity by measuring the amount of resistive torque generated by the participant's muscles. Our results demonstrated no significant increase in passive wrist stiffness on the paretic side compared to the non-paretic side ($p = 0.408$). However, the paretic side showed higher reflex torque than the non-paretic side at all velocities ($p < 0.001$). In addition, there was a significant increase in reflex torque associated with increasing velocities on the paretic side ($p < 0.05$).

The lack of significant differences in passive stiffness between the paretic and non-paretic sides supports the view that passive stiffness increases with time after stroke. As described earlier, spasticity develops and peaks at 1–3 months after a stroke, and the non-neural component may increase with time, thus contributing to increased muscle tone at 6 months post-stroke [17]. Previous studies in patients with chronic stroke have reported inconsistent results. Given et al. used an instrumental device to quantify spasticity bilaterally in the ankle plantar flexors and elbow flexors in stroke patients with hemiparesis, and found higher passive stiffness in the ankle plantar flexors in the affected limb than the contralateral limb but no significant difference between sides in the elbow flexors [11]. To quantify wrist flexor spasticity, Gaverth et al. used a neuroreflexor instrument that distinguishes between spasticity and the non-neural component by applying different constant velocities, and reported no significant difference in passive stiffness between the paretic side in stroke patients and healthy people [22]. Another study that measured finger flexor spasticity found that passive stiffness was no greater in participants with stroke than in control participants [14]. It has been suggested that passive stiffness is influenced by several factors including limb size, the amount of intramuscular connective tissue, muscle fiber type and cross-sectional area [11, 16]. The greater passive stiffness in ankle plantar flexors has been attributed to histological characteristics of these muscles, which are rich in connective tissue and slow-twitch fibers [11]. In light of the present results and previous findings, we speculate that upper limb resistance to muscle stretching after stroke is mediated primarily by neurological rather than muscular components. However, further studies are required to better characterize the post-stroke changes in passive stiffness in both upper and lower limb muscles.

Higher reflex torque on the paretic side compared to the non-paretic side showed that spasticity was the predominant cause of increased passive resistance. The contribution of the stretch reflex to increased muscle tone is

maximal between 1 and 3 months after a stroke [17]. The mean post-stroke period in participants in the present study was 3 months. Increased stretch reflex gain is the mechanism underlying spasticity. Cumulative evidence from animal and human studies supports the supraspinal origin of stretch reflex hyperexcitability [19]. More specifically, the loss of balanced inhibitory and excitatory descending reticulospinal projections after a stroke is the most plausible mechanism for post-stroke spasticity [20]. Our findings show that reflex torque was dependent on velocity, becoming greater with increasing velocities of passive stretching. Speed dependence is one of the defining characteristics of spasticity, and is attributed to stretch reflex activity, which increases in intensity at higher velocities [40]. This characteristic has also been reported in other studies that tested ID at different velocities [4, 34, 35]. In contrast, Kim et al. examined ankle plantar flexor spasticity with ID in chronic stroke patients [16], and reported no increase in resistive torque associated with increasing velocity after testing five consecutive passive movements at velocities of 60, 120, 180 and 240°/s. Methodological differences between the present study and that of Kim et al. may account for the difference in findings. Kim and colleagues reported resistive torque without calculating reflex torque. Subtracting resistive torque from the slow velocity in the present study eliminated components of passive stiffness. A further potential explanation for the differences between studies is that Kim and colleagues used five consecutive passive motion tests without a rest between trials. In this connection, another earlier study reported the adaptation of reflex torque responses after repeated passive movements [33].

Our findings are clinically useful because wrist flexor muscle spasticity is easily and objectively quantified. The present results thus suggest that ID is a tool with potential applications in clinical practice, in particular to objectively evaluate changes in spasticity in response to different treatments.

The findings of this study should be interpreted with due consideration of its limitations. Electromyography was not used to confirm that reflex torque responses measured with ID resulted from muscle contraction induced by stretch reflex activation. In addition, our results are not generalizable to all adults with stroke. This study included only individuals with moderate to severe paresis in the upper limb as indicated by their MAS scores. Our participants were in the subacute phase of stroke (less than 6 months after stroke), and because the contribution of spasticity to increased muscle tone is maximal between 1 and 3 months after stroke onset, it would be informative to investigate spasticity at shorter post-stroke time points. A final limitation is that spasticity was evaluated here only in the wrist flexor muscles. Future studies should measure spasticity with ID in other patient populations and other muscle groups.

Our findings show that isokinetic dynamometry is a potentially useful tool to quantify spasticity in the wrist flexor muscles in patients with subacute stroke.

Conflict of interest: Authors state no conflict of interest.

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