JRSR 9 (2022) 156-161

Journal of Rehabilitation Sciences and Research

Journal Home Page: jrsr.sums.ac.ir

Original Article

The Relationship between Neural Properties, Mechanical Properties, Functional Outcomes, and Clinical Parameters in Spastic Chronic Stroke Patients: An Observational Study

Ehsan Ghasemi[1]*, PhD; Khosro Khademi-Kalantari[2], PhD; Fateme Bokaee[1], PhD; Alireza Akbarzadeh Baghban[3], PhD; Majid Ghasemi[4], MD

[1]Musculoskeletal Research Center, Isfahan University of Medical Sciences, Isfahan, Iran
[2]Department of Physiotherapy, Faculty of Rehabilitation, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[3]Department of Basic Sciences, School of Rehabilitation, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[4]Department of Neurology& Isfahan Neurosciences Research Center, Faculty of Medical Sciences, Isfahan University of Medical Sciences, Isfahan, Iran

ARTICLE INFO

Article History:
Received: 25/06/2021
Revised: 01/12/2021
Accepted: 21/05/2022

Keywords:
Neural properties
Mechanical properties
Functional outcome
Muscle spasticity
Stroke

Please cite this article as: Ghasemi E, Khademi-Kalantari K, Bokaee F, Akbarzadeh Baghban AR, Ghasemi M. The Relationship between Neural Properties, Mechanical Properties, Functional Outcomes, and Clinical Parameters in Spastic Chronic Stroke Patients: An Observational Study. JRSR. 2022;9(4):156-161.

ABSTRACT

Background: Subsequent to spasticity, which is a positive impairment of stroke, neural and mechanical changes often occur in paretic muscles, affecting muscle function. The aim of this study was to find more accurate indices, which could affect decisions about spasticity treatment by investigating the relationships among neural, mechanical, functional outcomes, and clinical parameters in spastic chronic stroke patients.
Methods: This cross-sectional study investigated 45 spastic chronic stroke patients. Clinical assessments were conducted using the Modified Modified Ashworth Scale (MMAS). Neural properties including H-reflex latency and H_{max}/M_{max} ratio were acquired. Mechanical properties including fascicle length, pennation angle, and thickness of spastic medial gastrocnemius muscle were evaluated. Functional outcomes were evaluated by the Timed Up and Go (TUG) test and Timed 10-Meter Walk Test (10-m WTT). Spearman's rank correlation analysis in SPSS version 22.0 was used to find correlations between parameters.
Results: A low negative correlation was determined between MMAS and H-reflex latency (r=-0.320, P=0.032). MMAS score had a low significant relationship with pennation angle (r=0.296, P=0.049) and thickness of muscles (r=0.389, P=0.008). However, no significant correlation was found between MMAS and functional outcomes.
Conclusion: Based on these findings, it is clear MMAS can partly identify changes in neural and mechanical properties of spastic muscles.

Introduction

Stroke is recognized as the most important cause of morbidity in adults [1]. Spasticity is a positive impairment of stroke with a highly variable prevalence, estimated between 4% and 42.6% [2]. Lance (1980) defined spasticity as "a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflex with exaggerated tendon jerks, resulting from the hyperexcitability of the stretch reflex" [3]. Spasticity also can arise from intrinsic transformations in muscles or reflex properties [4, 5].

Various studies have distinguished the quantification of mechanical and neural properties of spastic muscles in stroke patients [6]. Spasticity can be advantageous at times for some functional movements; nevertheless, it is still one of the most important impairments, limiting motor functions in many stroke patients [7]. Subsequent to spasticity, paretic muscles may undergo

*Corresponding author: Ehsan Ghasemi, Department of Physiotherapy, School of Rehabilitation, Isfahan University of Medical Sciences, Isfahan, Iran. **Tel:** +98 31 37925009; **Fax:** +98 31 36687270
Email: eghasemi@rehab.mui.ac.ir

morphological changes, affecting muscle function [8]. The skeletal muscle architecture, including the fascicle length, pennation angle, and thickness of muscles, may be affected by adjustments in the mechanical properties of muscles [9, 10].

One approach for evaluating muscle injury following stroke is the use of clinical scales; however, these scales cannot identify specific changes in the structure of the muscles [11]. Moreover, there are debates regarding the reliability of clinical scales in adults with central nervous system injuries [12]. As a result, quantitative methods have been developed to investigate spasticity. For instance, ultrasonography, as a noninvasive method, has recently been used to investigate muscle/tendon function in stroke [6, 13]. However, only few ultrasonic studies have examined spastic muscles in stroke patients [9, 11]. With this background in mind, a better understanding of the association between instrumental and clinical features of muscle spasticity may aid clinicians in selecting the appropriate therapeutic approach for improving spasticity treatment.

The main aim of this research was to analyze the relationship between the neural and mechanical properties of spastic medial gastrocnemius muscle and clinical parameters in spastic chronic stroke patients. The secondary aim was to examine the association between functional outcomes and clinical features of spasticity in chronic stroke patients.

Methods

Participants

In this cross-sectional study, a descriptive analysis through convenience sampling was performed on forty-five participating subjects with hemiparesis post-stroke. G*power 3.1.9.2 software was used to calculate sample size. The power was set at 0.8 and the alpha error at 0.05, and the effect size was set to 0.4 based on the pilot study. Participants were recruited from rehabilitation centers in the city of Isfahan in 2017, with the use of announcements to inform patients about this study. Inclusion criteria comprised: (1) at least 3 months since stroke onset; (2) spasticity in the medial gastrocnemius muscle according to the Modified Modified Ashworth Scale (MMAS≥1) [14]; (3) cognitive (MMSE≥24) [15]; and (4) the ability to walk alone for 10 m with or without assistive devices. Subjects were excluded if they: (1) had pain in the lower limbs; (2) were participating in a special training program; (3) used anti-spastic drugs or botox injections; or (4) had other central nervous system lesions. All subjects were outpatients, were living at home, and were undergoing physical therapy at the time of the study. Subjects signed an informed consent form to participate in the study, and all procedures were confirmed by the local Ethics Committee of Shahid Beheshti University of Medical Sciences, Tehran, Iran (reference number: IR.SBMU.REC.1394.133).

Clinical Evaluation

In the neurological assessment, muscle tone, hyperreflexia, and ankle clonus were evaluated in the affected lower limb. The MMAS was used for the clinical evaluation of muscle tone in the ankle joint [14]. In the MMAS, an ordinal scale (0-4) is applied to rate spasticity: 0 equals no increase in muscle tone; 1, a minor increase in muscle tone indicated by a catch-and-release movement or minimum resistance at the end of range of motion (ROM), while flexing or extending the affected part; 2, major increase in muscle tone as indicated by a catch movement in the middle of ROM and resistance in the remainder of ROM (the affected part can move easily); 3, major increase in muscle tone associated with difficulty in passive movement; and 4, rigid flexion or extension of the affected part. During the evaluation, the patient was in a supine position with head held in the midline, arms along the trunk, and lower limbs in the extended position [14]. The examiner put a hand under the ball of the foot and used the other hand to stabilize the limb near the ankle joint; the ankle was moved from maximum plantar flexion to maximum dorsiflexion [14]. A 4-point scale was used to evaluate the Achilles deep tendon reflex (DTR) when stimulated with a clinical hammer. The ankle clonus was evaluated with "Yes" or "No" options. A physiotherapist measured the passive ROM of the ankle joint using a handheld goniometer. In this evaluation, the patient remained in the supine position with the knee fully extended. The axis of the goniometer was fixed on the lateral malleolus. The angle between the fibular shaft and the fifth metatarsal bone was also calculated (6).

Evaluation of Neural Parameters

The H-reflex and the M-wave were acquired with an EMG machine (Cadwell Co., Kennewick, Washington, USA). The band pass filter was set at 20 Hz-2KHz. The amplified signals were digitized and stored in the computer's hard disk for subsequent calculation of the Hmax/Mmax ratio and the H-reflex latency. An experienced neurologist conducted electrophysiological examinations. The examiner was blind to the group assignment of the subjects. For this examination, patients were requested to lie in a prone position with feet hanging from the bed [16]. The skin was prepared, and then the cathode was placed in the popliteal fossa on the tibial nerve. The active electrode was placed on the gastro-soleus muscle (halfway between the medial malleolus and medial tibia condyle). The reference electrode was placed on the insertion of the Achilles tendon. The ground electrode was tied between the active and the reference electrodes (Figure 1).

Rectangular pulse electrical stimulation (duration, 1 ms; stimulus frequency, 1 pulse every 5 seconds) was applied to stimulate the tibial nerve [16]. First, by moving the electrode so as to detect noticeable contractions of the gastrocnemius muscle, we characterized the most appropriate site for the stimulation of the tibial nerve passing through the popliteal fossa. Following that, the amplitude of the current was slowly increased to record H-reflexes. H-max was described as the response with the greatest amplitude. The intensity of the stimulus was increased until optimal M-wave was attained. Latency of the H-reflex was calculated from onset of stimulation until the first deflection of H-reflex.

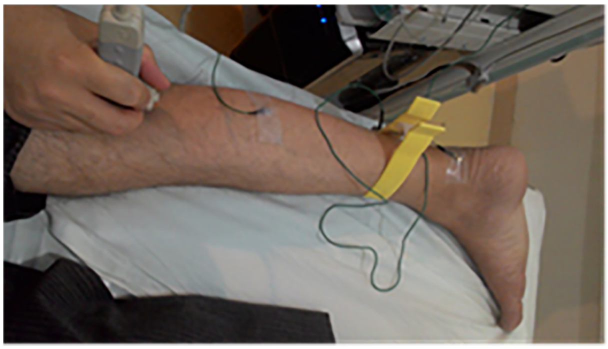

Figure 1: Picture of electrodes and stimulator placement for obtaining and recording the H-reflex and M-response.

Evaluation of Mechanical Parameters

Mechanical parameters included muscle thickness and muscle pennation angle of medial gastrocnemius muscle using a real-time, B-mode ultrasonography device (Accuvix V10 system, Medison Co., Seoul, Korea). A linear probe (7.5 MHz, 50mm) was used to obtain images of the medial gastrocnemius muscle on the affected side. An ample amount of gel was used to prevent pressure-induced changes in muscle tissues. Patients were requested to lie in a prone position with their feet hanging from the bed. To prevent the possible effect of tonicity changes (due to stimulation of a reflex response in spastic muscles), the examiner did not correct the position of the knee or ankle joint. For ultrasonography, the probe was placed perpendicular to the muscle belly of the spastic gastrocnemius, with a 1/3 proximal distance between the lateral tibia condyle and lateral malleolus of ankle marked with a horizontal line (line A) (Figure 2).

The probe was placed horizontally on line A, and then the distance between the lateral and the medial gastrocnemius muscle was marked with a vertical line (line B) (Figure 3). The midpoints between line B and the innermost edge as well as line B and the outermost edge of the leg were specified with two vertical lines (C and D lines, respectively). Then the probe was longitudinally positioned as its center met the intersection of line A with line C or line D.

The distance between superficial and deep aponeurosis in the middle of the image was considered as muscle thickness of the medial gastrocnemius. To measure the pennation angle, the angle between deep aponeurosis and visible fascicle closest to the center of the image was measured (Figure 4).

Functional Outcome Measurements

Two scales were used to evaluate lower limb function. The first scale was the 10-meter Walking Timed Test (10-m WTT), for which the speed of walking was calculated over 10 meters using a stopwatch. Calculations were carried out over the middle 10 meters of a 14-m distance to prevent the effects of deceleration and acceleration. The second functional test was the Timed Up and Go (TUG) test, a reliable and valid instrument for measuring functional mobility (ICC, 0.99) [17]. Patients were asked to stand up, walk 3 meters, turn around, walk back, and then sit down. A stopwatch was used to measure the time required for the test. An independent examiner, blind to subject assignment, performed the clinical assessments.

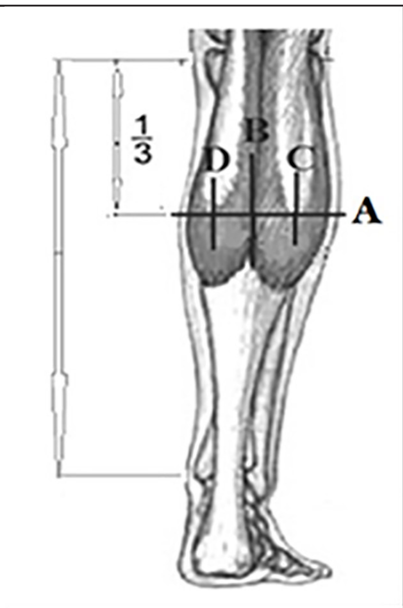

Figure 2: Sonographic scanning sites (A=The horizontal line on the 1/3 proximal way distance between the tibia lateral condyle and the end of the lateral malleolus; B=The vertical line on the place between the bulks of the medial and the lateral gastrocnemius muscles; C=The vertical line on the midway between line B and the innermost edge of the leg; D=The vertical line on the midway between line B and the outermost edge of the leg.)

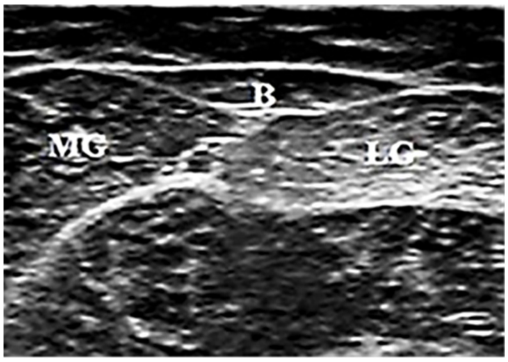

Figure 3: Ultrasonography image on line A (Figure 2) for detecting the area between the medial and lateral gastrocnemius bulks (MG=Medial Gastrocnemius, LG=Lateral Gastrocnemius, B=The place between the bulks of the medial and the lateral gastrocnemius muscles)

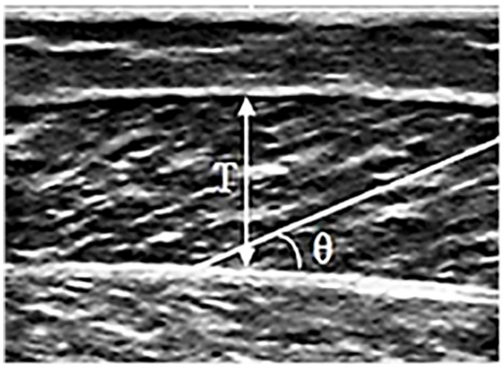

Figure 4: Ultrasonography image by placing the ultrasound probe on line C (Figure 2) to detect the thickness and pennation angle of the medial gastrocnemius (T=Thickness, θ=Pennation angle)

Statistical Analysis

SPSS version 22.0 (SPSS Inc., Chicago, USA) was used to analyze the data. The Shapiro–Wilk test was used to determine data normality. Because MMAS and DTR are ordinal variables, Spearman's rank correlation test was used to quantify associations among variables. The significance level was set at 0.05.

Table 1: Characteristics of chronic stroke patients (n=45)

Age (years)	Weight (Kg)	Height (Cm)	Post-Stroke Onset (Month)	Stroke Side	Gender
55.62±12.54 (30–76)	68.07±12.01 (40–100)	162.33±8.91 (150–180)	22.42±18.04 (3–60)	R=22 L=23	M=21 F=24

R, right; L, left; M, male; F, female

Table 2: Correlation matrix of variables (Spearman's *p*)

Outcome measures	MMAS	ADTR	ROM	HR	R	PA	MT	TUG
ADTR	0.406**							
ROM	-0.465**	-0.270						
HR	-0.320*	-0.322*	0.534**					
R	-0.199	0.014	0.060	0.002				
PA	0.296*	-0.005	-0.251	-0.310*	-0.058			
MT	0.389**	-0.003	-0.027	-0.150	-0.071	0.545**		
TUG	0.071	0.166	-0.179	-0.147	-0.039	-0.190	-0.319*	
WTT	0.025	0.163	-0.108	-0.118	-0.270	-0.237	-0.345*	0.953**

MMAS, Modified Modified Ashworth Scale; ADTR, Achilles Deep Tendon Reflex; ROM, Range of Motion; HR, H-Reflex; R, Ratio; PA, Pennation Angle; MT, Muscle Thickness; TUG, Timed up and GO; WTT, Walking Timed Test. *Significant correlation (P<0.05). **Significant correlation (P<0.01)

Results

General Features of the subjects: The mean duration of post-stroke onset±S.D was 22.42±18.04 (range, 3-60 months) (Table 1). All stroke patients showed clinically enhanced Achilles' tendon reflex (DTR≥2), and all stroke patients had increased medial gastrocnemius muscle tone to passive manual dorsiflexion with knee in extension (MMAS≥1). Fifteen patients showed clinical clonus in the ankle joint.

Correlation among variables: As shown in Table 2, MMAS had a poor negative association with H-reflex latency (r=-0.320, P=0.032) as well as a poor direct association with DTR (r=0.406, P=0.006) and a poor negative association with ROM (r=-0.465, P=0.001). The manually measured dorsiflexion ROM had a moderate significant correlation with H-reflex latency (r=0.534, P<0.001). In addition, DTR was inversely associated with H-reflex latency (r=-0.322, P=0.031).

As shown in Table 2, MMAS had a poor and significant relationship with pennation angle (r=0.296, P=0.049) and muscle thickness (r=0.389, P=0.008). The pennation angle had a moderate positive correlation with muscle thickness (r=0.545, P<0.001). As shown in Table 2, there was no significant correlation between the clinical and functional outcomes. In functional outcomes, a strong significant correlation was found between WTT and TUG results (r=0.953, P<0.00).

Discussion

This study revealed a poor and significant correlation between MMAS and neural and mechanical properties.

The Modified Ashworth Scale (MAS) has been widely used by practitioners because of its straightforward and simple application [18]. Nevertheless, it cannot detect the relative contribution of neural and mechanical components in spastic muscle stiffness [5]. Moreover, there is no consensus regarding the inter- and intra-rater reliability of this scale, as the scores are subjective and largely dependent on the examiner's experience. Therefore, some researchers are uncertain about the inter-and intra-rater reliability of this clinical scale [12].

In the present study, a scale similar to MAS, referred to as MMAS, was used. This scale has an extremely good inter-and intra-rater reliability regarding plantar flexors of the ankle joint [14, 19]; however, it, too, is subjective but cannot separate the neural and mechanical components of spastic muscles.

Correlation between neural properties and clinical measures: Neurophysiological assessment is the analysis of electrical activity (e.g. EMG signals) to determine spasticity [18]. In some studies, EMG has been applied to examine the response stimulated by either the muscle (M-reflex) or electrical stimulation of the peripheral nerve supplying the muscle (H-reflex) in spastic patients with exaggerated responses; it is also related to the extent of spasticity [18]. H-reflex latency and H_{max}/M_{max} ratio can be used as reliable indices to measure α motor neuron excitability [20].

No significant correlation was observed between H_{max}/M_{max} ratio and clinical assessments. Although a significant correlation between MAS and H_{max}/M_{max} ratio has been suggested in the literature [21], in some studies, including the present research, MAS had no significant association with the H_{max}/M_{max} ratio [22, 23]. This discrepancy probably arises from differences in study populations and the parameters used to indicate motor neural excitability [21]. Furthermore, the weak association between the extent of spasticity and neurophysiological test results could be due to the shortcomings of MMAS. In this scale, resistance to passive muscle stretching is measured based on the examiner's subjective analysis. Spasticity, thixotropy, and fixed muscle contractures are normally indicated in this type of resistance [23].

Correlation between mechanical properties and clinical measures: According to Dietz and Sinkjaer, changes in mechanical muscle properties are the main reason for muscle hypertonia [4]. Some studies have used ultrasonography to examine disruption in the normative architecture of the gastrocnemius muscle in chronic stroke patients [9, 10, 24]. The present study found a poor positive correlation between MMAS and mechanical properties, which was in agreement with previous studies [25].

Cheng-Ya Huang et al. reported a significant relationship between MAS and the mechanical properties of spasticity (represented by MMG M_{max} amplitude) [21]. Picelli et al. revealed a low significant association between MAS and echo intensity of the gastrocnemius muscle in chronic stroke patients [24] .They reported that a lower MAS score correlates with a greater pennation angle and greater muscle thickness [24]. In contrast, Yang et al., in agreement with the current study, showed a positive correlation between MAS and pennation angle and muscle thickness [26]. They suggest that increased muscle tone makes muscle fibers produce concentric contractions and increases stretch torque. Thus, the component of torque perpendicular to the muscle fascicles membrane and angle to axial torque increased [26]. According to the present results, although MMAS was not as efficient as quantitative analysis, it could be partly indicative of changes in the passive properties of spastic hypertonic ankle.

Correlation between functional outcomes and clinical measures: Inconsistent with some previous studies [27], the present research indicated that ankle spasticity had no significant correlation with TUG or WTT scores. The negative results could be related to the subjects' compensatory movements such as increasing hip and knee flexion to release the affected foot in the swing phase [28]. Moreover, patients' continuous disability following stroke (due to stroke-related impairments or negative adaptations of the body associated with maladaptive behaviors) may be another contributing factor [29]. In addition, the correlation between spasticity and gait velocity has not been confirmed, as static spasticity has been measured in place of dynamic spasticity [27, 30]. In this regard, Lamontagne et al. first measured the spasticity of plantar flexors among subjects with hemiparesis during walking. This method of measuring dynamic spasticity has confirmed efficiency for gait velocity [30]. One of the most important limitations of the present study was that muscle spasticity was assessed statically. Another limitation of our study was the lack of access to objective tools for measuring spasticity. Therefore, it is suggested that future studies employ objective tools to measure spasticity dynamically. It is further recommended that similar studies be conducted on other upper motor neuron lesions, e.g. spinal cord injury or multiple sclerosis.

Conclusion

As the findings of the present study suggest, MMAS as a clinical scale can indicate changes in neural and mechanical properties of spastic muscles; however, its performance is not as accurate as quantitative analysis. The findings further suggest that MMAS scoring may be dependent on both neural and mechanical components.

Acknowledgment

The authors of this study express their gratitude to all participants in this project, especially the personnel at Hazrat Abolfazl Rehabilitation Center and at Isfahan Neuroscience Research Center.

Funding

The current study is the results of a thesis for the PhD degree in physical therapy registered at the Shahid Beheshti University of Medical Sciences (Research project number: IR.SBMU.REC.1394.133).

Conflict of Interest: None declared.

References

1. Carr JH, Shepherd RB. Neurological rehabilitation: optimizing motor performance: Elsevier Health Sciences; 2010.
2. Wissel J, Manack A, Brainin M. Toward an epidemiology of poststroke spasticity. Neurol. 2013;80(3 Supplement 2):S13-S9.
3. Lance JW. The control of muscle tone, reflexes, and movement Robert Wartenbeg Lecture. Neurol. 1980;30(12):1303-.
4. Dietz V, Sinkjaer T. Spastic movement disorder: impaired reflex function and altered muscle mechanics. Lancet Neurol. 2007;6(8):725-33.
5. Galiana L, Fung J, Kearney R. Identification of intrinsic and reflex ankle stiffness components in stroke patients. Exp brain res. 2005;165(4):422-34.
6. Ghasemi E, Khademi-Kalantari K, Khalkhali-Zavieh M, Rezasoltani A, Ghasemi M, Akbarzadeh Baghban A, et al. The Effect of Functional Stretching Exercises on Neural and Mechanical Properties of the Spastic Medial Gastrocnemius Muscle in Patients with Chronic Stroke: A Randomized Controlled Trial. J Stroke Cerebrovasc Dis : the official journal of National Stroke Association. 2018;27(7):1733-42.
7. Hu X, Suresh NL, Chardon MK, Rymer WZ. Contributions of motoneuron hyperexcitability to clinical spasticity in hemispheric stroke survivors. Clin Neurophysiol. 2015;126(8):1599-606.
8. Berenpas F, Martens AM, Weerdesteyn V, Geurts AC, van Alfen N. Bilateral changes in muscle architecture of physically active people with chronic stroke: A quantitative muscle ultrasound study. Clinical neurophysiology : Clin Neurophysiol. 2017;128(1):115-22.
9. Gao F, Grant TH, Roth EJ, Zhang L-Q. Changes in passive mechanical properties of the gastrocnemius muscle at the muscle fascicle and joint levels in stroke survivors. Arch Phys Med Rehabil. 2009;90(5):819-26.
10. Dias CP, Lanferdini FJ, Onzi EdS, Goulart NBA, Becker J, Gomes I, et al. The influence of hemiparesis on triceps surae morphological and mechanical properties in stroke survivors. Isokinet Exerc Sci. 2016;24(2):157-64.
11. Li L, Tong KY, Hu X. The effect of poststroke impairments on brachialis muscle architecture as measured by ultrasound. Arch Phys Med Rehabil. 2007;88(2):243-50.
12. Blackburn M, van Vliet P, Mockett SP. Reliability of measurements obtained with the modified Ashworth scale in the lower extremities of people with stroke. Phys Ther. 2002;82(1):25-34.
13. Pradines M, Ghedira M, Portero R, Masson I, Marciniak C, Hicklin D, et al. Ultrasound structural changes in triceps surae after a 1-year daily self-stretch program: a prospective randomized controlled trial in chronic hemiparesis. Neurorehabil Neural Repair. 2019;33(4):245-59.
14. Ghotbi N, Nakhostin Ansari N, Naghdi S, Hasson S. Measurement of lower-limb muscle spasticity: intrarater reliability of Modified Modified Ashworth Scale. J Rehabil Res Dev. 2011;48(1):83.
15. Ansari NN, Naghdi S, Hasson S, Valizadeh L, Jalaie S. Validation of a Mini-Mental State Examination (MMSE) for the Persian population: a pilot study. Applied neuropsychology. 2010;17(3):190-5.
16. Bakheit A, Maynard V, Shaw S. The effects of isotonic and isokinetic muscle stretch on the excitability of the spinal alpha motor neurones in patients with muscle spasticity. Eur J Neurol. 2005;12(9):719-24.
17. Podsiadlo D, Richardson S. The timed "Up & Go": a test of basic functional mobility for frail elderly persons. J Am Geriatr Soc. 1991;39(2):142-8.
18. Kim KS, Seo JH, Song CG. Portable measurement system for the objective evaluation of the spasticity of hemiplegic patients based on the tonic stretch reflex threshold. Med Eng Phys. 2011;33(1):62-9.
19. Ghotbi N, Ansari NN, Naghdi S, Hasson S, Jamshidpour B, Amiri S. Inter-rater reliability of the Modified Modified Ashworth Scale in assessing lower limb muscle spasticity. Brain injury.

2009;23(10):815-9.
20. Angel R, Hofmann W. The H reflex in normal, spastic, and rigid subjects: studies. Arch Neurol. 1963;8(6):591-6.
21. Huang C-Y, Wang C-H, Hwang S. Characterization of the mechanical and neural components of spastic hypertonia with modified H reflex. J Electromyogr Kinesiol. 2006;16(4):384-91.
22. Tsai KH, Yeh C-Y, Chang HY, Chen JJ. Effects of a single session of prolonged muscle stretch on spastic muscle of stroke patients. Proceedings-National Science Council Republic of China Part B Life Sciences. 2001;25(2):76-81.
23. Bakheit A, Maynard V, Curnow J, Hudson N, Kodapala S. The relation between Ashworth scale scores and the excitability of the α motor neurones in patients with post-stroke muscle spasticity. J Neurol Neurosurg Psychiatry. 2003;74(5):646-8.
24. Picelli A, Tamburin S, Cavazza S, Scampoli C, Manca M, Cosma M, et al. Relationship between ultrasonographic, electromyographic, and clinical parameters in adult stroke patients with spastic equinus: an observational study. Arch Phys Med Rehabil. 2014;95(8):1564-70.
25. Chung SG, van Rey E, Bai Z, Roth EJ, Zhang L-Q. Biomechanic changes in passive properties of hemiplegic ankles with spastic hypertonia. Arch Phys Med Rehabil. 2004;85(10):1638-46.
26. Yang Y-B, Zhang J, Leng Z-P, Chen X, Song W-Q. Evaluation of spasticity after stroke by using ultrasound to measure the muscle architecture parameters: a clinical study. Int J Clin Exp Med. 2014;7(9):2712.
27. Hsu A-L, Tang P-F, Jan M-H. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. Arch Phys Med Rehabil. 2003;84(8):1185-93.
28. Freire B, Abou L, Dias CP. Equinovarus foot in stroke survivors with spasticity: a narrative review of muscle–tendon morphology and force production adaptation. Int J Ther Rehabil. 2020;27(1):1-8.
29. Dean CM, Richards CL, Malouin F. Task-related circuit training improves performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. Arch Phys Med Rehabil. 2000;81(4):409-17.
30. Lamontagne A, Malouin F, Richards CL. Locomotor-specific measure of spasticity of plantarflexor muscles after stroke. Arch Phys Med Rehabil. 2001;82(12):1696-704.