Annals of Physical and Rehabilitation Medicine 62 (2019) 397–402

Available online at ScienceDirect www.sciencedirect.com

Elsevier Masson France EM|consulte www.em-consulte.com

Original article

Association of spasticity and motor dysfunction in chronic stroke

Svetlana Pundik [a,b,*], Jessica McCabe [a], Margaret Skelly [a], Curtis Tatsuoka [c], Janis J. Daly [d,e]

[a] Louis Stokes Cleveland VA Medical Center, 10701, East Boulevard, Cleveland, OH 44106, USA
[b] Department of Neurology, Case Western Reserve University School of Medicine, 11100, Euclid avenue, Cleveland, OH 44106, USA
[c] Department of Biostatistics, Case Western Reserve University, 11000, Euclid Avenue, Cleveland, OH 44106, USA
[d] Department of Neurology, College of Medicine, University of Florida; McKnight Brain Institute, University of Florida, 1149 Newell Drive, Gainesville, FL 32611, USA
[e] Department of VA National Brain Rehabilitation Research Center of Excellence, Research Service, 151-A, 1611 S.W. Archer Road, Gainesville, FL 32608, USA

ARTICLE INFO

Article history:
Received 31 October 2017
Accepted 7 July 2018

Keywords:
Chronic stroke
Muscle tone
Spasticity
Modified Ashworth Scale
Fugl-Meyer
Weakness
Prevalence

ABSTRACT

Background: The prevalence of increased muscle tone after stroke is frequently reported as 30% to 40%, and the condition is often concurrent with motor control deficits, manifesting as an inability to isolate paretic-limb joint movements.
Objective: The objectives of this retrospective analysis were to 1) report the prevalence of increased muscle tone in a convenience sample of 128 chronic stroke survivors with moderate/severe motor deficits and 2) quantify the relation between tone and motor impairment in chronic stroke survivors.
Methods: Analyses included descriptive statistics and multiple regression modeling, with the modified Ashworth Scale score (MAS; tone) as a predictor of isolated joint movement control (Fugl-Meyer score [FM]; motor impairment).
Results: Increased muscle tone was present in 97% of subjects. Increased muscle tone was associated with impaired motor control (FM; upper extremity, $P = 0.008$; lower extremity, $P = 0.03$) after adjusting for age, time since stroke and sex. We found a significant difference between flexor and extensor strength for finger, elbow, hip and knee joints ($P < 0.002$). Participants were classified in high and low MAS score groups. With high MAS score and for muscles of finger flexion and forearm pronation, we found a trend toward impaired strength of antagonist muscles (finger extensors and forearm supinators, respectively) as compared with low MAS score for these same muscle pairings.
Conclusions: The prevalence of increased tone was higher in this study than in previous reports. Increased muscle tone in chronic stroke survivors with persistent motor dysfunction could be associated with impaired motor control and differential muscle strength of antagonistic muscles.
Published by Elsevier Masson SAS.

1. Introduction

Abnormal muscle tone and spasticity are consequences of injury to the central nervous system. High muscle tone has serious consequences in terms of financial cost and quality of life[1]. It is a commonly encountered impairment that coexists with motor deficits poststroke [2,3]. However, its prevalence in the chronic phase (> 3 months) shows inconsistencies. Studies assessing muscle tone, typically measured with the modified Ashworth Scale (MAS), describe a varied prevalence among stroke survivors, with values from 17% [4] to 79% [5,6] and the most frequently reported prevalence 19% to 43% [3,7–12]. Similarly, according to other clinical measures (Tone Assessment Scale), the prevalence of abnormal tone was 36% to 38% [10,13]. Some studies have indicated that higher rates of spasticity are more likely when patients exhibit paresis [14]. However even then, rates were below 45% [14]. To date, the prevalence of increased tone among chronic stroke survivors with moderate/severe motor deficits remains undetermined.

The presence of increased muscle tone can have a negative impact on motor control [6,9,10] and motor learning [15] after stroke. Impaired muscle tone is often associated with abnormal posture and abnormal co-activation of agonist and antagonist muscles [16]. Increased tone, especially within zones of spasticity, has a heightened negative impact on motor performance in both single [17] and multi-joint movements [18]. Zones of spasticity have been defined as the angular range within which this spasticity is detected, and when present, can have an adverse effect on voluntary motor performance [15]. Importantly, movement within

* Corresponding author. Cleveland VA Medical Center, 10701 E. Boulevard, Cleveland, OH 44106, USA.
E-mail addresses: sxp19@case.edu (S. Pundik), jmccabe@fescenter.org (J. McCabe), peggy.skelly@gmail.com (M. Skelly), curtis.tatsuoka@case.edu (C. Tatsuoka), Janis.daly@neurology.ufl.edu (J.J. Daly).

https://doi.org/10.1016/j.rehab.2018.07.006
1877-0657/Published by Elsevier Masson SAS.

these zones results in diminished motor learning capability [15] and precludes practice of more coordinated movements.

Here we conducted a retrospective analysis to reexamine the prevalence of increased tone in a convenience sample of chronic stroke survivors with moderate/severe motor deficits. Because of the paucity of information regarding the specific impact of tone on muscle strength and motor control, we also characterize the relation between increased muscle tone and motor dysfunction.

2. Methods

2.1. Selection of participants

This study was a retrospective secondary analysis of a 128-participant cohort of chronic stroke survivors with persistent moderate/severe motor deficits of the upper or lower limb. Inclusion criteria were $>$ 6 months after ischemic or hemorrhagic stroke, cortical or subcortical lesion, hemiparesis, and $\geq$ trace muscle contraction in major muscle groups. Exclusion criteria were inability to follow commands or provide informed consent, medically unstable, and $>$ 1 stroke. Research oversight was provided by the Medical Center Institutional Research Board and informed consent was obtained from participants.

2.2. Outcome measures

The MAS was used to quantify muscle tone [19]. The MAS is the most commonly used measure of muscle tone [14]. For participants in the upper-limb cohort (n = 82), 9 muscles were individually scored, then scores were summed for an overall MAS score. For participants in the lower-limb group (n = 46), 10 muscles were individually scored, then scores were summed. Strength was assessed by a 12-point ordinal scale ranging from 0 to 5 [20]; grades within this scale that were "+" or "−" were converted to numerical values for analysis (e.g., 2+ = 2.33; 3− = 2.66). During testing, the limb was positioned to isolate muscle movement as much as possible. To quantify motor control, we used the Fugl-Meyer Upper Extremity scale (FMUE; 33 items; 0–66 points) and Fugl-Meyer Lower Extremity scale (FMLE; 17 items; 0–34 points) [21].

2.3. Statistical analysis

Analyses involved using SPSS v21 (IBM, Armonk, NY). Data were tested for normality by the Lilliefors method, and most data were non-normally distributed. Therefore, nonparametric statistics were used. To identify the prevalence of increased tone as well as agonist and antagonist muscle strength in the entire cohort (n = 128), we generated descriptive statistics. For the remainder of the analyses, the upper-limb (n = 82) and lower-limb (n = 46) groups were analyzed separately. Multiple linear regression analysis tested the association of the explanatory variable sum MAS for the UE or LE and the dependent variable FMUE or FMLE, respectively, adjusting for age, time since stroke, and sex. Linear regression assumptions relating to normality and homoskedasticity were supported by evaluating residuals using Q-Q plots and scatter plots, confirming the acceptability of using this model for this particular analysis. Collinearity was evaluated by the variance inflation factor (VIF), where VIF $>$ 10 denotes unacceptable collinearity between predictors. To study the relation between tone and strength, participants were first stratified into groups of "high" and "low" MAS scores for each individual muscle (high MAS score $\geq$ 2; low MAS score $<$ 2). The two groups were compared by Mann-Whitney U test by muscle strength of the antagonist muscle. In addition, we compared the strength of agonist versus antagonist muscles within each muscle pair at a given joint by the Wilcoxon Signed Rank test. Results were considered statistically significant at $P < 0.05$ corrected for multiple comparisons by the Holms procedure [22]. Because of the exploratory nature of this secondary analysis, we discuss results with and without correction for multiple comparisons.

3. Results

Table 1 provides participant characteristics.

3.1. Prevalence of increased tone

For the total cohort (UE and LE groups combined), 97% of the participants had increased muscle tone (MAS score $\geq$ 1 in at least one muscle). For the UE group, all participants had increased tone (MAS score $\geq$ 1 in at least one muscle); and for the LE group, 91% had increased tone (MAS score $\geq$ 1 in at least one muscle). In the UE group, 67.9% had at least one muscle with a MAS score $\geq$ 2 and in the LE group, 39.1% had at least one muscle with a MAS score $\geq$ 2. For UE muscles, there was an average across subjects of 5/9 total UE muscles with a mean MAS score $\geq$ 1, and for LE muscles, there was an average of 1/10 LE muscles with a mean MAS score $\geq$ 1. For UE muscles, the highest mean MAS score was in finger flexors, followed by wrist flexors, elbow flexors, shoulder internal rotators, and forearm pronators (Table 2). For LE muscles, the highest mean MAS score was in ankle plantarflexors, followed by hip adductors, knee extensors, knee flexors and hip internal rotators (Table 2).

3.2. Characteristics of muscle weakness and muscle tone across a joint

The weakest UE muscles (mean strength grade $<$ 2) for the UE group were finger extensors, followed in order by wrist flexors, wrist extensors and forearm supinators, (Table 2). The weakest LE muscles (mean strength grade $<$ 2) for the LE group were ankle plantarflexors and knee flexors (Table 2). For each UE pair of muscles at a given joint, comparison of agonist versus antagonist strength showed equal weakness of agonist versus antagonist for all pairs except finger flexors/extensors and elbow flexors/extensors, ($P \leq 0.002$, adjusted for multiple comparison; Table 2). Similarly, we found equal weakness for all LE pairs

Table 1
Participant characteristics and Fugl-Meyer scores.

Characteristics	Total	Upper extremity (n = 82)	Lower extremity (n = 46)
Age, median (IQR)	59 (15)	58 (13)	60 (17)
Sex (number of participants/% male)	90/70.31	56/68.29	34/73.91
Side of hemiparesis (number of participants/% left affected)	52/40.62	34/41.46	18/39.13
Time since stroke, months, median (IQR)	22.11 (25.67)	23.6 (24.1)	19.12 (33.25)
Stroke etiology, ischemic or hemorrhagic (number of participants/% ischemic)	98/78.56	59/71.95	39/84.78
Fugl-Meyer score (FMUE or FMLE), median (IQR); range	NA	22 (10); 8–49	20 (5); 6–28

IQ: interquartile range; FMUE: Fugl-Meyer upper extremity; FMLE: Fugl-Meyer lower extremity; NA: not assessed.

Table 2
Modified Ashworth scale (MAS) score and strength for pairs (double line) of agonist and antagonist muscles.

Muscles (pairs of agonists and antagonists)		MAS score, median (IQR)	*P*-value*	Strength grade, median (IQR)	*P*-value**
Upper extremity	Finger flexors	2 (1)	<0.0001	2.67 (2)	<0.00001
	Finger extensors	0 (0)		0 (1.67)	
n = 82	Wrist flexors	1.5 (1)	<0.0001	1.67 (0.66)	ns
	Wrist extensors	0 (0)		1.67 (0.66)	
	Elbow flexors	1.5 (1)	<0.0001	2.67 (1.34)	0.002
	Elbow extensors	1 (1.5)		2.33 (1.66)	
	Forearm pronators	1.5 (0.5)	<0.0001	2.17 (1.66)	ns
	Forearm supinators	0 (0)		2 (1.67)	
	Internal rotators	1 (0.5)	NA†	2.33 (1.33)	NA†
Lower extremity	Ankle dorsiflexors	0 (0)	<0.0001	2 (0.12)	ns
	Ankle plantarflexors	1.5 (1)		1.67 (2)	
n = 46	Knee extensors	0 (1.5)	ns	3.68 (1.66)	<0.00001
	Knee flexors	0 (1.5)		1.67 (1)	
	Hip abductors	0 (0)	<0.0001	2.33 (0.67)	ns
	Hip adductors	1 (1)		2.33 (0.67)	
	Hip extensors	0 (1)	0.01	2.33 (1.08)	0.01
	Hip flexors	0 (0)		2.85 (1)	
	Hip external rotators	0 (1)	ns	2.15 (1)	ns
	Hip internal rotators	0 (1.5)		2 (0.66)	

UE: upper extremity; LE: lower extremity; ns: not significant; NA†: not assessed. Comparison with an antagonist not assessed as an external rotator spasticity was not collected.
* Comparing agonist-antagonist MAS score by Mann-Whitney U test.
** Comparing agonist-antagonist strength by Mann-Whitney U test.

except knee flexors/extensors and hip flexors/extensors ($P \leq 0.01$, adjusted for multiple comparison; Table 2).

We found a difference in MAS score for 4 of 4 pairs of UE muscles: finger, wrist, elbow flexor/extensor and forearm pronator/supinator ($P < 0.0001$, adjusted for multiple comparison; Table 2). For LE muscles, we found a difference in MAS score for 3 of 5 muscle pairs: ankle plantar/dorsiflexors, hip adductor/abductors and hip flexor/extensor ($P < 0.01$, adjusted for multiple comparisons; Table 2).

3.3. Association between tone and motor deficits

3.3.1. Impaired strength and abnormal muscle tone

For each muscle in the UE, we stratified the MAS score as high muscle tone (MAS score ≥ 2) and low muscle tone (MAS score < 2; [11]), then compared the low to high MAS group for a given muscle by the strength of the antagonist muscle. Muscle strength tended to be lower in muscle groups with antagonists characterized by high tone, measured at rest (Table 3). Specifically, results showed a trend toward more impaired strength in finger extensors and forearm supinators when they opposed high muscle tone (MAS score ≥ 2, high MAS group) versus when they opposed low muscle tone (MAS score < 2, low MAS group). For example, we found a trend toward impaired strength in finger extensors in individuals with high muscle tone of finger flexors (unadjusted $P = 0.036$; Table 3). For the LE, there were too few participants with high muscle tone (MAS score ≥ 2).

3.3.2. Motor impairment (Fugl-Meyer) and muscle tone

Sum MAS score for the whole limb was a significant predictor of FM after adjusting for age, time since stroke, and sex for both the UE group ($P = 0.008$; overall adjusted $R^2 = 0.10$) and LE group ($P = 0.03$; overall adjusted $R^2 = 0.17$; Table 4).

4. Discussion

The results of this study extend the literature by showing 1) a higher than previously reported prevalence of increased

Table 3
Comparison of low versus high MAS score groups by strength of the antagonist muscle opposing agonist tone.

Muscle	Low muscle tone group (MAS score < 2 in agonist) antagonist muscle strength grade, mean (SD)	High muscle tone group (MAS score ≥ 2 in agonist) antagonist muscle strength grade, mean (SD)	*P*-value*
Finger extensors	1.00 (1.67) (*n* = 38)	0.00 (1.50) (*n* = 43)	0.14 (0.036)
Finger flexors	2.67 (1.83) (*n* = 80)	2.33 (0.00) (*n* = 1)	NA
Wrist extensors	1.67 (0.66) (*n* = 48)	1.67 (0.83) (*n* = 33)	0.6 (0.2)
Wrist flexors	1.67 (0.66) (*n* = 78)	2.67 (1.50) (*n* = 3)	NA
Forearm supinators	2.33 (1) (*n* = 68)	1.00 (2.33) (*n* = 13)	0.07 (0.014)
Forearm pronators	2.165 (1.66) (*n* = 80)	1.67 (0) (*n* = 1)	NA
Elbow extensors	2.33 (1.66) (*n* = 59)	2.33 (1.33) (*n* = 22)	0.79 (0.79)
Elbow flexors	2.67 (1.34) (*n* = 70)	2.67 (0.75) (*n* = 11)	0.6 (0.27)
Ankle dorsiflexors	2.33 (2.83) (*n* = 32)	1.5 (2.67) (*n* = 14)	0.42 (0.21)
Ankle plantarflexors	1.67 (1.67) (*n* = 46)	NA (*n* = 0)	NA
Knee extensors	4.00 (1.66) (*n* = 39)	2.67 (2.00) (*n* = 7)	NA
Knee flexion	1.67 (1.16) (*n* = 40)	1.67 (0.66) (*n* = 6)	NA
Hip abductors	2.33 (0.69) (*n* = 43)	1.67 (1.25) (*n* = 3)	NA
Hip adductors	2.33 (0.67) (*n* = 46)	NA (*n* = 0)	NA
Hip extensors	2.33 (1.08) (*n* = 45)	1.67 (0) (*n* = 1)	NA
Hip flexors	3.00 (1.00) (*n* = 43)	2.33 (1.25) (*n* = 3)	NA

NA; not applicable because of disproportion group sizes.
* *P*-value by Mann Whitney U test adjusted for multiple comparisons using the Holms procedure followed by unadjusted *P*-value in parentheses.

Table 4
Regression analysis predicting motor impairment (Fugl-Meyer score) by MAS score, adjusting for age, time since stroke and sex.

	Upper extremity ($n = 77$) parameter estimate (P-value)	Lower extremity ($n = 44$) parameter estimate (P-value)
Sum MAS score (9 UE muscles or 10 LE muscles)	−0.304 ($P = 0.008$)	−0.33 ($P = 0.03$)
Age	−0.027 (ns)	−0.04 (ns)
Time since stroke	0.015 (ns)	0.27 (ns)
Sex	−0.030 (ns)	0.46 (ns)

ns; non-significant.

muscle tone (MAS) in chronic stroke survivors with persistent moderate/severe motor impairment; 2) that increased tone (MAS) significantly predicted overall motor impairment, with the model accounting for 10% to 17% of the variance; and 3) a significant difference between strength of flexor and extensor muscles of the finger, elbow, hip and knee.

5. High prevalence of increased muscle tone in chronic stroke with persistent motor impairment

Our cohort showed a higher prevalence of increased muscle tone by the MAS score than recent reports [5,19] for individuals with chronic stroke (> 3 months) with persistent motor impairment. In contrast, other studies generally reported a much lower prevalence, with the most frequently reported prevalence 19% to 43% [3,7–12]. In these studies, participants were recruited acutely and followed longitudinally, and they presumably included many individuals with improved motor control to potentially normal over time along with normalization of muscle tone. One study identified abnormally increased tone in 79% of individuals with residual weakness at 1 year after stroke [5]. However, 28% of participants regained upper-limb dexterity, which potentially underestimates the true occurrence rates in patients with greater residual motor impairment. Additionally, when alternative measures of muscle tone were used, such as muscle activity recorded by electromyography (EMG), rates were variable [23–25]. In one study of EMG, 42% of individuals had increased spasticity – a component of increased tone – of the elbow flexors [23]. In another EMG study, 87% of individuals at 6 weeks poststroke had detectable spasticity, whereas in the same cohort, only 44% had abnormal tone according to the MAS score [24]. Importantly, in a cohort of patients with no functional movement of the upper limb at 36 weeks post-stroke, 92% had increased spasticity as measured by EMG [25]. Although the sample size was small ($n = 30$) and measurement of spasticity was limited to the wrist, the study demonstrated greater occurrence of increased spasticity in functionally impaired individuals in the chronic phase. In our cohort, we analyzed most muscle groups within the affected limb and found that 97% demonstrated clinically detectable increased muscle tone by the MAS score. The discrepancies in the literature in terms of prevalence may be due to evaluating individuals with mild motor impairment, using tools with low sensitivity, and analyzing only select muscles. Our study extends the literature by demonstrating higher than previously reported prevalence of increased tone across multiple muscles with an easily used clinical measure.

5.1. Tone and motor control

The impact a given impairment may have on motor control must be determined to inform treatment. Furthermore, the relations between the array of impairments that lead to dysfunctional motor control must be analyzed. Clinical experience supports our study findings that tone and motor function are significantly related. The limb posture assumed by many stroke patients includes digit/wrist flexion, forearm pronation, elbow flexion and shoulder internal rotation [26]. When movement is attempted, patients often demonstrate an abnormal flexor synergy in which flexion of the elbow, wrist and fingers is obligatorily linked [26]. In our study, within these same muscles, we found higher tone. This flexor synergy movement pattern precludes volitional, selective joint control and prevents hand opening for grasp, wrist stabilization for any functional task, and placement of the limb in a useful workspace. The abnormal restriction imposed by this habitual dyscoordinated flexor position worsens disuse of the upper limb, muscle atrophy, weakness, and further motor impairment.

5.2. Relation between increased muscle tone and motor impairment

Motor deficits and increased tone can and do coexist because central pathways that control both volitional movement (pyramidal tracts) and muscle tone (non-pyramidal tracts) originate in the primary motor cortex and descend side by side [27,28]. Notably, both pyramidal and non-pyramidal tracts ultimately synapse on the lower motor neuron in the spinal cord. A vascular stroke lesion is likely to damage both tracts simultaneously. In fact, pure pyramidal tract strokes are rare [29]. Furthermore, based on structural proximity, it is reasonable to consider that stroke lesions that impair a greater number of pyramidal motor tracts and cause greater motor deficits can also damage more non-pyramidal pathways that control muscle tone and thus result in greater spasticity. Therefore, these structural correlates are a likely explanation for our results. Our findings are consistent with others [6] in supporting clinical observations that increased muscle tone occurs in individuals with severe motor deficits.

In addition, our results support a proportional relation between high muscle tone and low FM score. This association accounted for 10% to 17% of the variance in FM score. The presence of such a relation is important to recognize. Others have reported these types of associations [6,9,30–32]. However, the findings, outcome measures and cohorts were somewhat different from our study. In a study by Kong et al. [6], only individuals with severe tone (MAS score ≥3) were more likely to have diminished dexterous function of the upper limb. Dexterous function was measured with the Motor Assessment Scale, which assesses overall motor performance and not just upper-limb function (e.g., includes items of gross mobility such as walking). This measurement may explain why dexterity was found only in individuals with the highest tone. In contrast, we found motor deficits across the full range of abnormal tone, with an inverse linear association between abnormal tone and diminished motor performance. Eder et al. reported a significant correlation between the MAS score and a novel drawing test measuring limb dexterity [31] but did not correlate findings to other measures of motor performance (e.g., FM). Although Lin and Sabbahi found a significant relation between increased tone and FM [32], the study included only 10 participants and only wrist flexor tone was assessed by the MAS. Similarly, in the study by Sorinola et al. [30], tone and function were significantly correlated, but only wrist flexors were tested. Finally, Welmer et al. [9] reported a similar relation as in our study

between increased tone and measures of global function and movement abilities, although in their cohort, only 34% of individuals had increased tone. In our study, we demonstrated a relation between a highly established clinical measure of motor performance (FM) and the clinically utilized MAS, thus accounting for both proximal and distal upper-limb tone in our model. Our findings support and add to the existing literature by demonstrating a relation between upper-limb tone and motor function.

For the lower limb, our results support the literature documenting a relation between LE tone and motor function [33–36]. For example, others have reported increased tone of LE muscles related to fall risk [33], reduced postural stability [34] and walking ability [35,36]. In contrast, one report found no association between increased tone and motor function for LE but the presence of such an association for the upper extremity [9]. Our study provides further evidence of a relation between lower-limb tone and motor function with the finding of the association between summed MAS and FMLE.

5.3. Tone and weakness in agonist/antagonist muscle pairs

In some agonist/antagonist pairs, muscle tone and strength were greater in one muscle of the pair versus the other. For the upper limb, finger and elbow flexors were stronger than extensors for the finger and elbow. In contrast, for the lower limb, extensors had significantly greater strength than flexors for the hip and knee. For these muscle pairs, high strength was observed in the muscles with higher tone of a given pairing. Of note, the differential in strength parallels flexor synergy in the arm and extensor synergy in the leg, which was observed in another study [7] in the first few weeks to 3 months after stroke but not at 18 months. In our study, strength differences between agonists and antagonists were not observed for wrist flexor/extensor or pronator/supinator pairings despite a significant difference in the amount of tone observed for these pairings. For the leg, we found no differences in strength grade for hip abductor/adductor, internal/external rotator and ankle dorsi/plantar flexor pairings despite significant differences in tone observed within these pairings. Our results demonstrate that a muscle can have both increased tone and strength compared to its antagonist (e.g., increased tone and greater strength grade of the finger flexors as compared to the finger extensors), but the reasons for this are not completely clear.

One possible explanation is the occurrence of co-contraction of the muscle pairs, which could preclude normal activation in one or both of the muscles. This relation has been reported for elbow flexors and extensors [17] and may explain our observations, although we are not able to confirm this explanation for other muscle groups, given that we did not study EMG signals in the current study. Another reason may lie in variation in the non-pyramidal motor tracts that innervate different motor neurons. After stroke and other types of brain injury, patients may demonstrate abnormal posturing with flexor synergy of the upper limbs and extensor synergy of the lower limbs [37]. Disinhibition of the red nucleus along with facilitation of the rubrospinal tract leads to abnormal activation of the upper-limb flexor muscles manifested as the upper-limb flexor synergy [37]. Furthermore, after injury, for the lower limbs, there is a bias of extensor-tract influence on lower-limb muscle activation leading to extensor synergy of the lower limbs [37,38]. These differences in brain control of muscles that contribute to reflexive posturing in the brainstem centers such as red nucleus and the non-pyramidal tracts may further explain our findings of strength differences between flexor and extensor muscle pairings.

We also demonstrated a trend for 2 upper-limb muscle pairs, with greater weakness in an agonist muscle and high muscle tone in the concomitant antagonist. First, digit extensors were weaker when greater tone was present in digit flexors. Similarly, forearm supinators were somewhat weaker when greater tone was present in forearm pronators (Table 3). In contrast, others have reported that antagonist muscle tone was not related to agonist muscle weakness [39]; however, in that study, only more proximal upper-limb muscles (elbow flexors and shoulder internal rotators) were examined. For the distal arm muscle pairs, increased tone of the antagonist may diminish the muscle strength of its agonist for select upper-limb muscles. A potential explanation may be that this high tone within the antagonist prevents sufficient practice of activation/movement in the agonist muscles, leading to further disparity in motor function. Another factor to consider is that in central nervous system disorders, standard clinical strength testing alone is not able to differentiate between abnormal resistance from the antagonist versus impaired force production in the agonist. This limitation may be a confounding factor of the strength testing results in both our study and that of others. Use of other measurement methods for muscle strength may yield a different result.

5.4. Study limitations

One study limitation is that participants were self-referred and thus may not represent the general population. A second limitation is that this cohort did not include those with MAS score = 4. A third limitation is the measures available to assess strength and tone. Strength testing was conducted according to standard manual muscle testing methods. As described above, these methods may contain confounding due to the inability to differentiate between abnormal tone of the antagonist and impaired force production of the agonist. Therefore, results should be considered within that limitation. While MAS is currently the measure most utilized to measure tone, it relies on the accuracy of the clinician to perform it properly and consistently. Furthermore, MAS quantifies only one aspect of muscle over-activity. Better detection of spasticity may be obtained by using instrumented measures such as EMG during movement at different velocities [40]. This type of spasticity measure may lead to improved detection, quantification and understanding of its impact on motor performance.

6. Conclusions

Our study found a higher than previously reported prevalence of increased muscle tone in chronic stroke survivors with moderate to severe motor impairment. Increased tone was associated with greater motor impairment. Reasonably, spastic muscles may not allow for adequate practice to strengthen the antagonist muscles or practice of more coordinated movements. Thus, over time, this limitation in ability to move against opposing elevated muscle tone may have caused relative weakness and greater motor dysfunction. It is important to clinically assess muscle tone given the demonstrated relation between increased tone and weakness in muscle pairs as well as the impact of tone on motor performance. Because tone may adversely affect motor control, future research is needed to explore the multifactorial relation between muscle tone and motor dysfunction poststroke and to identify interventions to ameliorate tone. In terms of clinical practice, our results support the importance of managing increased tone in conjunction with motor training to provide for optimal practice of more coordinated movements and greater benefits from strength training.

Funding

Department of Veterans Affairs Research and Development, Office of Rehabilitation Research and Development (grant nos. B6636 W, B3709R, B9024S, A3102R, B2226R, B9024S).

Authors' contributions

SP developed the design for the current study, developed the statistical analysis plan, analyzed data, and worked on all manuscript versions. JM acquired/analyzed and interpreted data, and worked on the final manuscript. MS assisted with data analysis, generated statistics and worked on the final manuscript. CT supervised and conducted statistical analyses. JD acquired funding; developed/designed the studies; acquired/analyzed and interpreted data; and edited final versions of the manuscript.

Disclosure of interest

The authors declare that they have no competing interest.

References

[1] Zorowitz RD, Gillard PJ, Brainin M. Poststroke spasticity: sequelae and burden on stroke survivors and caregivers. Neurology 2013;80:S45–55. http://dx.doi.org/10.1212/WNL.0b013e3182764c86.

[2] Lundstrom E, Smits A, Terent A, Borg J. Time-course and determinants of spasticity during the first six months following first-ever stroke. J Rehabil Med 2010;42:296–301. http://dx.doi.org/10.2340/16501977-0509.

[3] Wissel J, Schelosky LD, Scott J, Christe W, Faiss JH, Mueller J. Early development of spasticity following stroke: a prospective, observational trial. J Neurol 2010;257:1067–72. http://dx.doi.org/10.1007/s00415-010-5463-1.

[4] Lundstrom E, Terent A, Borg J. Prevalence of disabling spasticity 1 year after first-ever stroke. Eur J Neurol 2008;15:533–9. http://dx.doi.org/10.1111/j.1468-1331.2008.02114.x.

[5] Kong KH, Chua KS, Lee J. Symptomatic upper limb spasticity in patients with chronic stroke attending a rehabilitation clinic: frequency, clinical correlates and predictors. J Rehabil Med 2010;42:453–7. http://dx.doi.org/10.2340/16501977-0545.

[6] Kong KH, Chua KS, Lee J. Recovery of upper limb dexterity in patients more than 1 year after stroke: Frequency, clinical correlates and predictors. Neuro Rehabil 2011;28:105–11. http://dx.doi.org/10.3233/NRE-2011-0639.

[7] Welmer AK, Widen HL, Sommerfeld DK. Location and severity of spasticity in the first 1-2 weeks and at 3 and 18 months after stroke. Eur J Neurol 2010;17:720–5. http://dx.doi.org/10.1111/j.1468-1331.2009.02915.x.

[8] Sommerfeld DK, Eek EU, Svensson AK, Holmqvist LW, von Arbin MH. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. Stroke 2004;35:134–9. http://dx.doi.org/10.1161/01.STR. 0000105386.05173.5E.

[9] Welmer AK, von AM, Widen HL, Sommerfeld DK. Spasticity and its association with functioning and health-related quality of life 18 months after stroke. Cerebrovasc Dis 2006;21:247–53. http://dx.doi.org/10.1159/000091222.

[10] Watkins CL, Leathley MJ, Gregson JM, Moore AP, Smith TL, Sharma AK. Prevalence of spasticity post stroke. Clin Rehabil 2002;16:515–22.

[11] Kong KH, Lee J, Chua KS. Occurrence and temporal evolution of upper limb spasticity in stroke patients admitted to a rehabilitation unit. Arch Phys Med Rehabil 2012;93:143–8. http://dx.doi.org/10.1016/j.apmr.2011.06.027.

[12] Urban PP, Wolf T, Uebele M, Marx JJ, Vogt T, Stoeter P, et al. Occurence and clinical predictors of spasticity after ischemic stroke. Stroke 2010;41:2016–20. http://dx.doi.org/10.1161/STROKEAHA.110.581991.

[13] Leathley MJ, Gregson JM, Moore AP, Smith TL, Sharma AK, Watkins CL. Predicting spasticity after stroke in those surviving to 12 months. Clin Rehabil 2004;18:438–43. http://dx.doi.org/10.1191/0269215504cr727oa.

[14] Sommerfeld DK, Gripenstedt U, Welmer A-K. Sapasticity after stroke: an overview of prevalence, test instruments, and treatments. Am J Phys Med Rehabil 2012;91:814–20. http://dx.doi.org/10.1097/PHM.0b013e3182513a3.

[15] Subramanian SK, Feldman AG, Levin MF. Spasticity may obscure motor learning ability after stroke. J Neurophysiol 2018;119:5–20. http://dx.doi.org/10.1152/jn.00362.2017.

[16] Dewald JP, Pope PS, Given JD, Buchanan TS, Rymer WZ. Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. Brain 1995;118:495–510.

[17] Levin MF, Selles RW, Verheul MH, Meijer OG. Deficits in the coordination of agonist and antagonist muscles in stroke patients: implications for normal motor control. Brain Res 2000;853:352–69.

[18] Musampa NK, Mathieu PA, Levin MF. Relationship between stretch reflex thresholds and voluntary arm muscle activation in patients with spasticity. Exp Brain Res 2007;181:579–93. http://dx.doi.org/10.1007/s00221-007-0956-6.

[19] Thibaut A, Chatelle C, Ziegler E, Bruno MA, Laureys S, Gosseries O. Spasticity after stroke: physiology, assessment and treatment. Brain Inj 2013;27:1093–105. http://dx.doi.org/10.3109/02699052.2013.804202.

[20] Cuthbert SC, Goodheart GJ. On the reliability and validity of manual muscle testing: a literature review. Chiropr Osteopat 2007;15:4. http://dx.doi.org/10.1186/1746-1340-15-4.

[21] Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S. 1.The post-stroke hemiplegic patient. 1. a method for evaluation of physical performance. ScandJ Rehabil Med 1975;7:13–31.

[22] Holm S. A simple sequentially rejective multiple test procedure. Scand J Stat 1979;6:65–70.

[23] Ada L, O'Dwyer N, O'Neill E. Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. Disabil Rehabil 2006;28:891–7. http://dx.doi.org/10.1080/09638280500535165.

[24] Malhotra S, Cousins E, Ward A, Day C, Jones P, Roffe C, et al. An investigation into the agreement between clinical, biomechanical and neurophysiological measures of spasticity. ClinRehabil 2008;22:1105–15. http://dx.doi.org/10.1177/0269215508095089.

[25] Malhotra S, Pandyan A, Rosewilliam S, Roffe C, Hermens H. Spasticity and contractures at the wrist after stroke: time course of development and their association with functional recovery of the upper limb. Clin Rehabil 2011;25:184–91. http://dx.doi.org/10.1177/0269215510381620.

[26] Carr J, Shepherd R. Neurological Rehabilitation: Optimizing Motor Performance. Churchill Livingston/Elselvier; 2010.

[27] Denny-Brown D. The Nature Of Dystonia. Bull NYA Acad Med 1965;41:858–69.

[28] Keizer K, Kuypers HG. Distribution of corticospinal neurons with collaterals to the lower brain stem reticular formation in monkey (Macaca fascicularis). Exp Brain Res 1989;74:311–8.

[29] Lawrence DG, Kuypers HG. The functional organization of the motor system in the monkey I. The effects of bilateral pyramidal lesions. Brain 1968;91:1–14.

[30] Sorinola IO, White CM, Rushton DN, Newham DJ. Electromyographic response to manual passive stretch of the hemiplegic wrist: accuracy, reliability, and correlation with clinical spasticity assessment and function. Neurorehabil Neural Repair 2009;23:287–94. http://dx.doi.org/10.1177/1545968308321778.

[31] Eder CF, Popovic MB, Popovic DB, Stefanovic A, Schwirtlich L, Jovic S. The drawing test: assessment of coordination abilities and correlation with clinical measurement of spasticity. Arch Phys Med Rehabil 2005;86:289–95. http://dx.doi.org/10.1016/j.apmr.2004.04.045.

[32] Lin FM, Sabbahi M. Correlation of spasticity with hyperactive stretch reflexes and motor dysfunction in hemiplegia. Arch Phys Med Rehabil 1999;80:526–30.

[33] Wei T-S, Liu P-T, Chang L-W, Liu S-Y. Gait asymmetry, ankle spasticity, and depression as independent predictors of falls in ambulatory stroke patients. PloS One 2017;12:e0177136. http://dx.doi.org/10.1371/journal.-pone.0177136.

[34] Singer JC, Mansfield A, Danells CJ, McIlroy WE, Mochizuki G. The effect of post-stroke lower-limb spasticity on the control of standing balance: Inter-limb spatial and temporal synchronisation of centres of pressure. Clin Biomech Bristol Avon 2013;28:921–6. http://dx.doi.org/10.1016/j.clinbiomech.2013.07.010.

[35] Pradon D, Roche N, Enette L, Zory R. Relationship between lower limb muscle strength and 6-minute walk test performance in stroke patients. J Rehabil Med 2013;45:105–8. http://dx.doi.org/10.2340/16501977-1059.

[36] Ng SS, Hui-Chan CW. The timed up & go test: its reliability and association with lower-limb impairments and locomotor capacities in people with chronic stroke. Arch Phys Med Rehabil 2005;86:1641–7. http://dx.doi.org/10.1016/j.apmr.2005.01.011.

[37] Schenkman ML. Clinical neuroscience for rehabilitation. Upper Saddle River, N.J: Pearson Prentice Hall; 2013.

[38] Haines DE, Mihailoff GA. Fundamental neuroscience for basic and clinical applications. E-Book. Philadelphia: Elsevier Health Sciences; 2017.

[39] Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 1987;67:206–7.

[40] Calota A, Levin MF. Tonic stretch reflex threshold as a measure of spasticity: implications for clinical practice. Top Stroke Rehabil 2009;16:177–88. http://dx.doi.org/10.1310/tsr 1603-177.