A NEW TOOLKIT FOR MEASURING SPASTICITY: A PILOT STUDY INVESTIGATING THE VALIDITY AND RELIABILITY OF THE BIOTONE SYSTEM FOR PATIENTS POST-STROKE

by:

Saleh M. Aloraini

Submitted in partial fulfillment of the requirements
for the degree of Master of Science

at

Dalhousie University
Halifax, Nova Scotia
July 2014

TABLE OF CONTENTS

LIST OF TABLES v
LIST OF FIGURES vi
ABSTRACT vii
LIST OF ABBREVIATIONS AND SYMBOLS USED viii
ACKNOWLEDGEMENTS x
CHAPTER 1 INTRODUCTION 1
1.1 RATIONAL FOR THE THESIS 2
1.2 BACKGROUND 2
1.2.1 Overview of Stroke 2
1.2.2 Physical effects of Stroke 3
1.3 SPASTICITY LITRATURE REVIEW 4
1.3.1 Overview of Spasticity 4
1.3.2 Differences between Spasticity and Hypertonia 4
1.3.3 Prevalence of Spasticity 5
1.3.4 Pattern of Increased Tone After Stroke 6
1.3.5 Measurement of Spasticity After Stroke 6
1.4 THE BIOTONE STUDY 15
1.4.1 Context of Thesis 15
1.4.2 Contribution of Thesis to Larger BioTone Study 15
1.4.3 Sample Size of BioTone Study 15
1.4.4 The BioTone System 16
1.4.5 Assessment Procedure 19
1.4.5 Data Processing 20
1.5 STATEMENT OF PURPOSE 25
CHAPTER 2 MEASUREMENT OF SPASTICITY AFTER STROKE USING CLINICAL MEASURES: A SYSTEMATIC REVIEW 26
2.1 INTRODUCTION 28
2.2 METHODS 29
2.2.1 Literature Search 29
2.2.2 Study Selection 29
2.2.3 Data Extraction and Quality Assessment 30

2.3 RESULTS 30
2.3.1 Description of the Measurement Tools 34
2.3.2 Validity of the Measurement Tools 40
2.3.3 Reliability of the Measurement Tools 44
2.3.4 Responsiveness of the Measurement Tools 46
2.4 DISCUSSION 46
2.5 LIMITATION OF THIS REVIEW 49
2.6 RECOMMENDATIONS AND CONCLUSION 49
2.7 DECLARATION OF CONFLICT OF INTEREST 50
CHAPTER 3 METHODS 51
3.1 STUDY DESIGN 52
3.2 OBJECTIVES 52
3.2.1 Primary Aims 52
3.2.2 Secondary Aims 52
3.3 HYPOTHESES 52
3.3.1 Primary Hypotheses 52
3.3.2 Secondary Hypotheses 53
3.4 PARTICIPANTS 53
3.4.1 Inclusion Criteria 53
3.4.2 Exclusion Criteria 53
3.4.3 Recruitment and Setting 54
3.4.4 Screening 54
3.4.5 Sample Size 55
3.5 PROCEDURE 55
3.6 DATA PROCESSING 56
3.6.1 Elbow Musculature Data 56
3.6.2 Pendulum Test Data 58
3.7 DATA ANALYSIS 59
CHAPTER 4 RESULTS 61
4.1 PARTICIPANTS 62
4.2 SPASTICITY MEASURES 62
4.2.1 Clinical Spasticity Measures 62
4.2.2 BioTone Spasticity Measures 64

4.3 COMPARING KINEMATIC AND EMG DATA SETS 68
4.3.1 Difference between Spastic Onset Times 68
4.3.2 Correlation between Kinematic and EMG Data Sets 68
4.4 CONCURRENT VALIDITY OF BIOTONE MEASURES 69
4.5 CONSTRUCT VALIDITY OF BIOTONE MEASURES 71
4.6 TEST-RETEST RELIABILITY OF SPASTICITY MEASURES 72
4.6.1 Reliability of Spasticity Measures for Elbow Muscles 72
4.6.2 Reliability of Spasticity Measures for Knee Extensors 72
4.7 SECONDARY RESULTS 73
4.7.1 Spasticity Measures and Active Range of Motion 73
4.7.2 Spasticity Measures and Strength of Elbow Musculature 74
CHAPTER 5 DISCUSSION 76
5.1 DISCUSSION 77
5.2 LIMITATIONS 81
5.3 CONCLUSIONS 81
REFERENCES 82
APPENDIX A: Copyright Permission Letters 95
APPENDIX B: Modified Ashworth Scale 99
APPENDIX C: Tardieu Scale 101

LIST OF TABLES

Table 1 Quality of studies rated with critical appraisal scale 32
Table 2 Summary of studies 37
Table 3 Reliability of spasticity measures 44
Table 4 Characteristics of participants 62
Table 5 Participant's scores on clinical spasticity measures 63
Table 6 Spastic onset times 65
Table 7 Indicators of elbow flexors spasticity 66
Table 8 Indicators of elbow extensors spasticity 67
Table 9 Indicators of knee extensors spasticity 68
Table 10 Correlation between kinematic variables and EMG metrics 69
Table 11 Correlation between BioTone variables and spasticity clinical measures for elbow muscles 70
Table 12 Correlation between BioTone variables and spasticity clinical measures for knee extensors 70
Table 13 Correlation between variables obtained from the theoretical model and metrics obtained from the motion curve of the non-paretic elbow 71
Table 14 Intra-class correlation coefficients of measures for elbow muscles 72
Table 15 Correlation between elbow active range of motion deficit and indicators of elbow muscles spasticity 73
Table 16 Correlation between elbow strength deficits and indicators of spasticity 75

LIST OF FIGURES

Figure 1 The BioTone system 16
Figure 2 The ShapSensor™ fiber-optic goniometer 17
Figure 3 Isometric strength dynamometer 18
Figure 4 Pendulum test wheelchair 19
Figure 5 EMG electrodes and electrogoniometer placement for upper extremity 20
Figure 6 Derivation of, and deviation from, theoretical curve 22
Figure 7 Determining spastic reaction from kinematic data 23
Figure 8 Determining spastic reaction from EMG data 24
Figure 9 Flowchart of search and selection strategy 31
Figure 10 Variables used from the non-paretic elbow motion curve 57
Figure 11 The pendulum test 59
Figure 12 Interval plots of clinical spasticity measures for elbow flexors and elbow extensors 64

ABSTRACT

Purpose: This pilot study assessed the concurrent and construct validity and test-retest reliability of the BioTone system as a tool to quantify spasticity in patients following stroke.

Methods: 15 adults post-stroke (65±11 years, 11 males) with spasticity in upper and/or lower limb muscles participated. The BioTone system was used to measure spasticity elicited during fast (120-140 deg/sec) passive stretching of bilateral elbow flexors, elbow extensors and knee extensors. Spastic reaction onset time, angular velocity at onset (ΔV) and acceleration at onset (ΔA) were determined by analyzing, using MATLAB, departures of electrogoniometric data from a theoretical kinematic model based on a constant jerk profile. In addition, the root mean square departure for angular velocity (εV) and acceleration (εA) were calculated. EMG recordings were also analyzed to identify spastic reaction onset time, discrete change in EMG intensity and EMG amplitude density of the stretched muscle (ΔStr, εStr) and its antagonist (ΔNStr, εNStr). Other variables from the theoretical curve, which were the maximum velocity and absolute maximum acceleration of the theoretical model (MAX-V and MAX-A) and the root mean square of the theoretical model angular velocity and acceleration (V and A), were derived to determine the construct validity by comparing them to the corresponding variables obtained from the movement curve of the non-hemiparetic side. For knee extensor muscles, the relaxation index (RI) was calculated using the pendulum test. Relationships between the biometric results and the Modified Ashworth Scale (MAS) and Tardieu scale (TS) were explored. Test-retest reliability of all measurements was conducted with six participants, using an inter-test interval of <1 week.

Results: Most participants displayed mild spasticity. Significant correlations were found in MAX-A and V of elbow flexors calculated using the theoretical profile and the non-hemiparetic side as references (rho=0.66, p=0.003; rho=0.56, p=0.015). No significant differences were revealed between spastic onset time predicted from kinematic data and EMG data. Significant correlations were found between Elbow flexor MAS and ΔV and εV (rho=0.49, p=0.03; rho=0.47, p=0.04), and between TS of elbow extensors and ΔNStr (rho=0.46, p=0.04). For the knee extensor muscles, the RI index was significantly correlated with the MAS (rho=–0.54, p=0.023) and with TS (rho=–0.65, p=0.006). Significant correlations of certain variables were demonstrated on repeat testing — ΔA and εA during stretch of elbow flexors (p=0.012, p=0.017) and εStr during stretch of elbow extensors (p=0.018) and RI during pendulum test (p=0.002).

Conclusion: These findings provide preliminary information of aspects of validity and reliability of the BioTone system. The results showed that the BioTone measures have low to moderate concurrent validity, and low construct validity, whereas test-retest reliability was moderate for some of the variables. Further investigation of this device as a clinical tool to objectively measure spasticity in patients post-stroke is warranted.

Impact of the study: To reduce functional, emotional, and financial burdens of a common aftermath of stroke — spasticity— a valid, reliable and user-friendly tool of objectively measuring its clinical presentation is needed. This study provides preliminary evidence to support further development of the BioTone system as a potential device to fill this void.

LIST OF ABBREVIATIONS AND SYMBOLS USED

A	Root mean square of the acceleration of the theoretical model
A_N	Area under the curve for acceleration of the motion curve of the non-paretic limb
ADLs	Activities of daily living
APTS	Ankle Planter flexors Tone Scale
AROM	Active range of motion
AS	Ashworth Scale
BI	Barthel Index
BTX-A	Botulinum Toxin Type A
Cm	Centimeters
CSI	Composite Spasticity Index
CV	Coefficient of variation
EMG	Electromyography
H-reflex	Hoffman's Reflex
HRQL	Health-related quality of life measure
ICC	Intra-class correlation coefficient
K	Kappa Statistic
Kg	Kilograms
Kw	Weighted Kappa Statistic
MAS	Modified Ashworth Scale
MAX-A	Peak magnitude for acceleration of the theoretical model
MAX-A_N	Peak magnitude for acceleration of the motion curve of the non-paretic limb
MAX-V	Peak magnitude for velocity of the theoretical model
MAX-V_N	Peak magnitude for velocity of the motion curve of the non-paretic limb
MMAS	Modified-Modified Ashworth Scale
MTS	Modified Tardieu Scale
MVC	Maximal Voluntary Contraction
NSRC	Nova Scotia Rehabilitation Centre
P	P-value
PROM	Passive Range of Motion
R	Pearson's Product-Moment Correlation Coefficient
RHO	Spearman's Rank Correlation Coefficient
RI	Relaxation Index
ROM	Range of motion
RPM	Resistance to passive movement
SEM	Standard error of measurement
SR	Stretch reflex
SRM	Standardized response mean
TAS	Tone Assessment Scale
Tau-b	Kendall tau rank correlation coefficient
Te	Spastic onset time derived from EMG data
Tk	Spastic onset time derived from kinematic data
TS	Tardieu Scale

TSRT	Tonic Stretch Reflex Threshold
T-reflex	Tendon jerk reflex measured using EMG
UE	Upper extremity
UMNL	Upper motor neuron lesion
V	Root mean square of the angular velocity for theoretical model
V_N	Area under the curve for velocity of the motion curve of the non-paretic limb
VAS	Visual analogue Scale
WHO	World Health Organization
ΔA	Peak magnitude of departure in acceleration from reference profile
ΔAROM	Difference in elbow flexion active range of motion between the non-paretic side and paretic side
Δ Flexors	Difference in elbow flexion muscles strength between the non-paretic side and paretic side
Δ Extensors	Difference in elbow extension muscles strength between the non-paretic side and paretic side
ΔStr	EMG intensity at onset time for stretched muscle
ΔNStr	EMG intensity at onset time for non-stretched muscle
ΔV	Peak magnitude of departure in velocity from reference profile
εA	Root mean square departure from reference profile for acceleration
εStr	Area under the curve for stretched muscle
εNStr	Area under the curve for non-stretched muscle
εV	Root mean square departure from reference profile for velocity

ACKNOWLEDGEMENTS

Taking on an endeavor like this was very rewarding for me, but I could not have done this without support and guidance of many people. I would particularly like to thank and acknowledge Dr. Marilyn MacKay-Lyons, who has shown the attitude and the substance of a genius; she continually and persuasively conveyed a spirit of adventure in regard to research and scholarship. Without her supervision and constant help this thesis would not have been possible and I will always be grateful to her. I would also like to thank my committee members, Dr. Shaun Boe and Dr. Gordon Gubitz, who have been very supportive and encouraging from the outset.

This project would not have happened without the generous assistance of the patients at the Nova Scotia Rehabilitation Centre; a very special thanks to them. I would also like to acknowledge the staff at the Nova Scotia Rehabilitation Centre who have been encouraging and accommodating. Many thanks to Alison McDonald and Kim Parker for their help with recruitment and assessments.

Further acknowledgement is due to the researchers from the Institute of Biomedical Engineering at the University of New Brunswick. Many thanks to Dr. Chris McGibbon and Andrew Sexton for their support and help.

A special thanks to my family. Words cannot express how grateful I am to my mother and father for all of the sacrifices that they have made on my behalf. Their prayer for me was what sustained me thus far. I would also like to thank all of my friends who supported me in writing, and incented me to strive towards my goal. At the end I wish to thank my wife Muneerah and children Mohammed and Nawal for their patience, assistance, support and faith in me.

CHAPTER 1

INTRODUCTION

1.1 RATIONAL FOR THE THESIS

Following an upper motor neuron lesion (UMNL), such as stroke, patients can present with a variety of sensorimotor disorders.[1, 2] These disorders can be broadly classified as 'positive features' (e.g., abnormal reflex responses, clonus, spasticity, etc.) and 'negative features' (e.g., muscle weakness, loss of dexterity, etc.).[1] Although both positive and negative features contribute to the resulting functional loss in patients with an UMNL,[1, 2] clinicians pay substantial attention to one particular positive feature – spasticity – because it often interferes with functional recovery and may lead to secondary complications such as contractures, weakness and pain.[1-3]

Spasticity is one of the common clinical manifestations following stroke.[4, 5] It is characterized by an involuntary, velocity-dependent increase of the stretch reflex with exaggerated tendon jerks, which causes the stretched muscle to activate inappropriately during passive and active movements.[6, 7] Various approaches are available for managing patients who have spasticity such as bracing, botulinum toxin type A (BTX-A) and selective dorsal rhizotomy.[1, 3] However, determining the efficacy of these management techniques depend on measurement tools that can objectively quantify spasticity.[1, 3, 8]

The most commonly used clinical tools for measuring spasticity are the Ashworth scale (AS) and the modified Ashworth scale (MAS).[9] However, various studies have questioned the validity of these scales.[10-12] On the other hand, there are technologies that have been shown to accurately measure spasticity but they are often limited to laboratory-based research and are not suitable for everyday clinical use.[13-16] Clearly, there is a need for a clinically usable tool that can produce accurate and objective measures of spasticity, which are clinically relevant and meaningful for clinicians. Therefore, the purpose of this study is to investigate the clinical utility of a new device, the BioTone system, in measuring spasticity among patients post-stroke.

1.2 BACKGROUND

1.2.1 OVERVIEW OF STROKE

Stroke is an acute neurological deficit caused by cerebrovascular etiology that leads to rapid loss of brain function.[17] Ischemia and hemorrhage are the two major

mechanisms through which brain damage occurs in stroke.[18] Ischemic stroke represents about 80% of all strokes and it is characterized by decreased or lack of blood flow to the brain depriving neurons of necessary substrates.[18] Hemorrhagic stroke represents about 20% of all strokes originating from deep penetrating vessels and causing localized pressure injury through disruption of connecting pathways of the vessels.[18]

Stroke is the leading cause of neurological disability in Canada. It is estimated that it affects 50,000 Canadians each year with an estimated cost of $3.6 billion in physician services, hospital costs, lost wages, and decreased productivity.[19] Over 300,000 adults suffer from the consequences of stroke, with approximately two-thirds suffering from neurological deficits that hinder their participation in activities of daily living (ADLs).[20] Further, stroke is a global epidemic, which is a major contributor to morbidity and mortality.[21, 22] According to a report published by the World Health Organization (WHO),[21] the global annual incidence of stroke is estimated to be 9 million with a prevalence of 30.7 million stroke survivors. Furthermore, the WHO estimates that over 70% of the people who survived a stroke worldwide suffer from post-stroke impairments that affect their quality of life and ADLs.[21, 22] These figures indicate that there is a great need for post-stroke rehabilitation, which means developing better assessment tools and more effective therapeutic interventions.

1.2.2 PHYSICAL EFFECTS OF STROKE

Effects of stroke vary from one individual to another depending on the type (ischemic or hemorrhagic), severity and location in the brain.[17] These effects are categorized depending on the function they impair - physical, cognitive and emotional functions.[17] The physical impairments can be classified as 'negative features' and 'positive features',[1] which, collectively, lead to functional impairments and disability.[1, 2] Negative features include those that have been lost such as muscle weakness, loss of dexterity and fatigability.[2] Positive features, which are features that are not normally present, include abnormal reflex responses, spasms, clonus, dyssynergic movement patterns and spasticity.[2] Spasticity, a major contributor to physical impairment and disability,[1, 3] is the focus of my thesis.

1.3 SPASTICITY LITERATURE REVIEW

1.3.1 OVERVIEW OF SPASTICITY

Spasticity is defined as a motor disorder, where there is a velocity-dependent increase in muscle tone and exaggerated tendon jerks that result from over excitability of the stretch reflex.[6] The pathophysiological basis of spasticity is not completely understood; however, spasticity pathophysiology may be attributed to neuro-physiological changes that occur following stroke.[7] The neural changes that cause increased muscle tone may be attributed to the imbalance of inputs from central motor pathways, such as the dorsal reticulospinal tract and other descending pathways, to the interneuronal circuits of the spinal cord.[7] The main tract that inhibits spinal reflex activity is the dorsal reticulospinal tract, which arises from the ventromedial reticular formation.[7] It runs very close to the lateral corticospinal (pyramidal) tract. The dorsal reticulospinal tract is under facilitatory control of cortical motor areas, thereby augmenting the inhibitory drive.[7] The main excitatory pathway, which also arises from the brainstem, is the medial reticulospinal tract. Damage to these tracts causes loss of inhibitory control, leading to increased alpha motor neuron excitability at the segmental cord level and subsequent increase in muscle tone.[7]

1.3.2 DIFFERENCES BETWEEN SPASTICITY AND HYPERTONIA

Hypertonia and spasticity are terms that have confounded the discussion of muscle tone after stroke. Spasticity is a specific form of hypertonia due to a velocity-dependent increase in tonic stretch reflex.[7] This increase in the tonic stretch reflex causes an increased resistance to passive movement, which results from abnormal spinal processing of proprioceptive input (i.e., neural component).[7] Hypertonia, which also causes an increased resistance to passive movement, is the broader term used to describe a muscle with an abnormally increased tone due to conditions such as spasticity, rigidity, dystonia or non-neural changes that occur following an UMNL.[23] The non-neural factors that cause an increased resistance to passive movement are changes that occur in the mechanical visco-elastic properties of the muscle fiber and other soft tissues.[24, 25] Early after an UMNL, histological changes in the muscles, such as muscle fiber atrophy and loss of sarcomeres, have been shown to contribute to muscle stiffness.[24, 25] These changes

lead to increased tension development and altered reflex sensitivity.[24, 25] In addition, the accumulation of intramuscular connective tissue, increased fat content and degenerative changes at the musculo-tendinous junction cause reduced muscle compliance.[24, 25] In summary, spasticity refers to the increased resistance to passive movement, which is velocity-dependent, due to the neural changes after an UMNL. Hypertonia is the abnormally increased tone that could be attributed to changes in the neural and non-neural components.

1.3.3 PREVALENCE OF SPASTICITY

Spasticity develops in 19%[4]-43%[5] of stroke survivors, and occurs less commonly in older survivors than younger individuals due to the decrease in tendon and tonic reflexes in the aging population.[26] In a cohort study, Sommerfeld et al.,[27] assessed 95 patients within 1 week and 3 months post-stroke using the MAS, self-reported muscle stiffness, tendon reflexes, several motor impairment measures, and the Barthel index (BI). Of the 95 patients studied, 64 were hemiparetic, 18 were spastic, 6 reported muscle stiffness, and 18 had increased tendon reflexes 3 months after stroke. Patients without spasticity (n=77) had statistically significantly better motor and activity scores than patients with spasticity.[27] In an 18-month follow-up study with the same cohort of patients, the authors evaluated the frequency of spasticity and its association with functioning and health-related quality of life measure (HRQL).[28] Of the 66 patients studied, 38 were hemiparetic; of these, 13 displayed spasticity, 12 had increased tendon reflexes, and 7 reported muscle stiffness 18 months after stroke. Although there was a weak correlation between spasticity and HRQL, the hemiparetic patients without spasticity had significantly higher BI and HRQL scores.[28] These studies suggest that spasticity may have a negative impact on long-term functional improvement in patients post-stroke.

Post-stroke spasticity has put an enormous burden to the society as evidenced by a study conducted by Esquenazi,[29] to investigate the human and economic burden of spasticity and muscle over-activity. He found out that in the United States, where more than 800,000 people are affected by stroke each year, over $73.8 billion is spent to care

for patients with post-stroke complications and 80% of this expenditure is allocated to stroke survivors with spasticity.[29]

1.3.4 Pattern of Increased Tone After Stroke

Spasticity after stroke is more common, and more severe, in the upper limbs than lower limbs.[4] Schinwelski and Slawek[26] postulated that this pattern is associated with greater impairment in voluntary movements in the upper extremity (UE). Disparity in neural control might explain this difference - UE function is mainly influenced by supraspinal control while the lower limb is highly influenced by spinal locomotor centers.[26] Moreover, spasticity following stroke differs between flexor and extensor muscles depending on the injury type and location.[1] In general, spasticity follows UE flexor and lower extremity extensor patterns.[1] In a study by Hefter et al.,[30] the authors found that the flexor pattern was present in more than 85% of the 665 subjects with UE spasticity.

1.3.5 Measurement of Spasticity After Stroke

Various measures (or scales) are available for the assessment of spasticity. These scales may be used for diagnostic purposes, or to help determine and evaluate spasticity management methods.[8] These measures can be divided into two main types: (i) non-instrumented measures, and (ii) instrumented measures.

Non-Instrumented Measures

Non-instrumented measures are clinical tools that can be used to measure spasticity and do not require the use of instruments. These tools include the AS and its modified forms (MAS and modified-modified Ashworth scale [MMAS]), the Tardieu scale (TS) and its modified form (modified Tardieu scale [MTS]) and the Composite Spasticity Index (CSI).

Ashworth Scales

The AS, first suggested by Ashworth in 1964,[31] involves assessment of stretch resistance by passively moving the spastic limb in one direction of movement in order to assess the antagonist muscles for that movement (i.e., moving in flexion to test extensor muscles' spasticity). The patient is asked to relax while the joint is moved rapidly (over

one second) through the range of motion (ROM) and resistance is graded using a 5-point ordinal scale from (0-4).[31] The MAS is an upgraded scale of the original AS developed by Bohannon and Smith,[32] in response to the observation that many patients demonstrated a degree of spasticity between the level of 1 and 2 on the AS. Therefore, a grade (1+) was added between 1 and 2 of the AS, resulting in a 6-point ordinal scale. The procedure for performing the MAS is similar to the AS.[32] Later, Ansari et al.[33] developed the MMAS to address the issue of accurately grading levels of spasticity in light of documented disagreements between grades 1 and 2 on the AS and between 1 and 1+, and 1+ and 2 on the MAS.[33] The investigators reasoned that the poor reliability of the AS and MAS may be due to the lack of clarity of grade definitions. To address this issue, the investigators suggested using the same procedure of the AS and MAS when using the MMAS, with a 5-point ordinal scale (0-4) but with different definitions than the AS or MAS for each grade of the MMAS scale.[33]

Numerous studies have questioned the validity of the various Ashworth scales. Fleuren et al.[10] found that the AS was not significantly associated with electromyography (EMG) data but was significantly correlated (Spearman's rank correlation coefficient: rho ≥ 0.54, $p<0.05$) with measured torque from the resistance to passive movement (RPM). Similarly, Patrick and Ada,[34] found that the AS was not significantly correlated with fast stretch-induced EMG-activity but significantly correlated with RPM (rho ≥ 0.52, $p<0.01$).[35] Evidence of validity is somewhat more compelling for the MAS than the AS. Several studies have shown that there was no significant correlation between the MAS and electrophysiological measures of spasticity (e.g., Hoffman reflex [H-reflex]).[15, 36-38] However, a significant correlation has been reported between MAS and the magnitude of EMG response to stretch test (rho=0. 21, p=0.02).[39] Moreover, the MAS was found to have a significant negative correlation with stretch reflex latency (rho=–0.37, $p<0.01$) and a positive correlation with stretch reflex area (rho=0.36, $p<0.01$), both measured by EMG.[38] Similarly, RPM (measured using a torque device) was shown by two studies to have a significant correlation with MAS (rho ≥ 0.51, $p<0.01$).[38, 40] In regard to the MMAS, validity studies have shown conflicting results - a significant correlation between the MMAS and the H-reflex parameters was not found in one study,[41] whereas in another study a significant correlation with the H-reflex (rho=0.39, p=0.04) was reported.[42]

Brashear et al.[43] reported weighted Kappa statistic (Kw) of 0.67-0.74 for intra-rater reliability of the AS when used to assess muscles crossing the elbow and wrist joints of patients post stroke.[43] In terms of inter-rater reliability of the AS, another study reported that the intra-class correlation coefficient (ICC) was 0.58 for elbow flexors and 0.63 for knee extensors.[10] Gregson et al.[44] found great variability in reliability of the MAS of the ankle plantar flexors, knee flexors, elbow flexors and wrist flexors among patients post stroke - Kw=0.59-0.94 for intra-rater reliability and Kw=0.45-0.96 for inter-rater reliability.[44] Ansari et al.[45] reported that the Kappa statistics (K) for intra-rater and inter-rater reliability for the MMAS of the knee extensors among patients post stroke were K=0.82 and K=0.72, respectively.[45] Finally, another study reported that the inter-rater agreement of the MMAS of elbow flexors was Kw=0.81.[46]

Tardieu Scales

Tardieu et al.[47] developed the TS as a method for measuring spasticity. In developing the TS, EMG was used to determine the reflex activity when the elbow was passively extended from 60-110° at a variety of speeds. The investigators concluded that the threshold to elicit a stretch reflex occurred at a specific speed for each patient and postulated that this threshold speed would vary among subjects. They also compared the angular displacement of slow and fast passive range of motion (PROM) and suggested that differences between the two angles could determine the presence of soft tissue changes.[47] Held and Pierrot-Deseilligny,[48] further developed Tardieu's work, publishing the TS scale in 1969. They identified three factors for rating the intensity of spasticity: (i) strength and duration of the stretch reflex; (ii) angle at which the stretch reflex is activated; and (iii) speed necessary to trigger the stretch reflex.[48] The TS procedure involves the passive mobilization of the limb, starting with the limb placed where the muscle to be tested is in its least stretched position. The limb is moved at three speeds: V1-as slow as possible, V2-speed of the limb segment falling under gravity, and V3-as fast as possible (faster than the rate of the natural drop of the limb segment under gravity). In each of the previous speeds, the assessor takes note of two aspects. First is the quality of muscle reaction to stretch, which is graded using a 6-point ordinal scale. The second aspect is the angle of muscle reaction (spastic onset).[49]

The MTS was developed Boyd and Graham,[50] for the purpose of further standardizing the procedure of spasticity assessment. The MTS is very similar to the TS and uses the same 6-point ordinal scale of the TS but has two main differences. First, only two speeds are utilized for the test (V1 and V3). Second, the difference between the angle of 'catch' (R1) and the full range of motion (R2) is calculated. The authors suggested that a large difference between R1 and R2 is indicative of spasticity whereas a small difference is suggestive of passive restraint (i.e., muscle stiffness or contracture).[50]

Patrick and Ada,[34] found that the TS of hemiparetic elbow and ankle joint muscles was significantly correlated with fast stretch-induced EMG activity (Pearson's product-moment correlation coefficient [r]=0.62-0.86, $p<0.01$). Conversely, Naghdi et al.[51] did not find a significant correlation between the MTS of wrist flexors and H-reflex parameters. Paulis et al.[52] reported an ICC of 0.66 for intra- and inter-rater reliability of the TS of elbow flexors among patients post stroke. Singh and colleagues,[53] found that the intra-rater agreement for the MTS when used to assess elbow flexors was (ICC=0.85) and when used to assess ankle planter flexors was (ICC=0.86). Similarly, Ansari et al.[54] reported the ICC for intra- and inter-rater reliability of the MTS of the ankle plantarflexors in the stroke population to be 0.68 and 0.71, respectively.[54]

Composite Spasticity Index

The CSI estimates both phasic (tendon jerk, clonus) and tonic (resistance to stretch) aspects of the stretch reflex (SR) response and combines the scores to derive a composite score ranging from 1-16.[55] The first sub-test is the tendon jerk, which is given a score from 0-4. The second sub-test is resistance to full range passive joint displacement, which is performed at moderate speed (>100 degrees/second). The amount of resistance is then given a grade of 0, 2, 4 or 8. The final part of the test is clonus test, which is graded from 1-4. The overall score is interpreted as mild spasticity (0-9), moderate spasticity (10-12), or severe spasticity (13-16).[55]

Levin and Feldman,[56] found a significant negative relationship ($r=-0.65$, $p<0.05$) between the CSI of the plantarflexors and the stretch reflex thresholds measured by EMG. In addition, Ng and Hui-Chan[57] reported excellent 1-week test-retest reliability of

the CSI of the plantarflexors of affected and unaffected limbs among 10 patients with chronic stroke (ICC = 0.97 and 0.80, respectively).

In conclusion, non-instrumented measures of spasticity are practical tools for clinicians for assessing patients who have spasticity. They involve grading the resistance to passive motion using ordinal scales. They are easy to use and to do not require extensive training. Moreover, the reliability of these scales is moderate to good, especially when an experienced clinician conducts the assessment. However, the validity of theses scales is still somewhat questionable.

Instrumented Measures

Instrumented spasticity tools utilize a variety of instruments to obtain a measure of spasticity. These measures include the pendulum test, myotonometery, force/torque measurement and electrophysiological measures.

Pendulum Test

In 1951 Wartenberg,[58] reported his results "following personal experience with this test over many years." The test to which Wartenberg referred to involved positioning patients in sitting with their legs over the edge of a table, passively straightening both knees, then dropping the limbs and observing the pendulous motion that followed. Wartenberg provided detailed accounts of the qualitative behavior of pendulous limbs in patients with stroke, Parkinsonism, lower motor neuron involvement and other diverse conditions.[58] Several subsequent studies have given due credit to this pioneering work by referring to “Wartenberg's pendulum test”.[59-64] The current version of the test, developed by Bajd, Bowman, and Vodovnik,[59-61] is used to measure spasticity in the knee extensors. The test involves applying an electrogoniometer to the knee, and passively extending and then dropping the lower extremity with the patient in a sitting position. The drop usually elicits a stretch reflex among patients with knee extensor spasticity.[59-61] A relaxation index (RI), calculated using the electrogoniometric data, is the ratio of the difference between the angle at end of initial movement into flexion and angle at the start of test (i.e., magnitude of the first drop) and the difference between the angle at end of test and angle at start of test (i.e., magnitude of the initial angle).[59-61] The RI is considered to be a

measurement of spasticity—the lower the number the higher the amount of spasticity in the limb.[59-61]

Bohannon et al.[62] assessed the validity and reliability of the pendulum test on both affected and unaffected knee extensors of eight patients with chronic stroke. After demonstrating that the pendulum test was significantly correlated with the AS (rho>0.57) and had a test-retest reliability of (ICC>0.84), the investigators concluded that pendulum test is a valid and reliable test to assess knee extensor spasticity in the stroke population.[62] Le-Cavorzin et al.[65] studied the pendulum test in 15 people post-stroke with spasticity and 10 non-disabled controls by measuring the RI, reflex-mediated torque evoked in quadriceps femoris, viscosity, and elasticity. The reflex-mediated torque evoked in quadriceps femoris, as well as muscle biomechanical parameters (viscosity and elasticity), were calculated using mathematical modeling. The authors sought to discriminate between neural and non-neural factors that contributed to muscle spasticity to determine if the pendulum test's RI is a true representation of knee extensor spasticity. Both viscosity and reflex-mediated torque were found to be significantly higher in the spastic group ($p<0.05$). A significant non-linear (logarithmic) correlation ($r^2=0.85$) was found between the AS and the computed reflex-mediated torque, emphasizing the non-linear behavior of this scale. The RI calculated from the pendulum test exhibited an unsuitable U-shaped pattern of variation with increasing reflex-mediated torque. In contrast, the area under the goniometric curve revealed a linear relationship, which is more convenient for routine estimation of spasticity. The investigators concluded that the pendulum test is a reliable and simple technique for measuring spasticity in patients following stroke.[65] However, they agreed that the RI had limitations, and suggested that it be replaced by more valid measures, such as the area under the goniometric curve, especially for the assessment of therapeutic procedures.[65]

Myotonometery

The Myotonometer is a device that measures muscle compliance following a short mechanical pulse exerted by the testing end of the device (an acceleration probe) on the tested muscle.[66] This device allows assessment of muscle tone at rest. The acceleration transducer at the testing end of the device records the damped oscillations of the muscle

response. Three parameters of the tested muscle are obtained: (i) natural oscillation frequency, which characterizes muscle tone, (ii) logarithmic decrement of damping, which characterizes muscle elasticity, and (iii) muscle stiffness.[66] Unfortunately, no studies have investigated the relation of these parameters in relation to any other instrumented or non-instrumented spasticity measures. However, a number of studies reported on the reliability of the Myotonometer among the stroke population.[66-68] Chuang et al.[68] found the test-retest reliability of the biceps brachii tone parameter of the device to be ICC=0.96. Similarly, two other studies reported high test-retest reliability (ICC=0.93-0.96) for flexor carpi radialis and ulnaris muscles.[66, 67]

Force/Torque Measurement

Several studies have used torque measurement as a method for assessing spasticity.[69-74] This approach involves passively moving the tested limb, either manually or using a motor system. Some investigators measured the RPM and derived an index for spasticity by calculating the difference between slope of the force-angle curve from slow and fast stretches obtained using linear regression techniques.[69-72] In other cases, a dynamic equation of movement was calculated using the RPM to decompose the measured resistance into the resistance from the visco-elastic properties of the muscle and resistance due to spasticity.[73, 74]

Studies that have investigated the validity of torque measurement reported conflicting results. Two studies concluded that RPM was not correlated with EMG data or the MAS.[69, 71] However, Lee et al.,[73] found that the Spasticity Index, obtained using a dynamic equation of movement to analyze the RPM, was significantly correlated with the MAS (rho= 0.86, $p<0.001$). Voerman et al.[72] also found that RPM did have a significant correlation with the MAS but only at low velocity passive movement (rho=0.73, $p<0.05$). Additionally, Voerman et al.[72] reported the range in ICCs for intra- and inter-reliability of the RPM of the wrist flexors among patients post stroke to be 0.67-0.89 and 0.96-0.99, respectively.

Electrophysiological Measures

Electrophysiological measures for quantifying spasticity involve the use of EMG to measure the responses evoked by one or more of the following: (i) electrical

stimulation of the peripheral nerve supplying the muscle (H-reflex); (ii) passive stretch of the muscle (i.e., the SR); or (iii) tendon tap (T-reflex).[75] These tests can be used to evaluate whether the responses are exaggerated in people with spasticity and the extent to which the findings relate to the degree of spasticity measured clinically.[75] When measuring spasticity using the H-reflex, the EMG following an electrical stimulation records the magnitude of the first response (i.e., M-wave), and then records the magnitude of the second response, (i.e., H-reflex). It has been suggested that the ratio of the H-reflex to the M-wave (H/M ratio) can be used as a measure for spasticity.[76] The H/M ratio is used as an indicator for muscle spasticity because it was found to be increased in spastic muscles, reflecting the enhanced excitability in the monosynaptic stretch reflex arc in spastic muscles.[77] For the SR, EMG data collected while the SR is elicited either manually or using a motor system are analyzed to determine a measure of spasticity.[78] Finally, the T-reflex measures the intensity of muscle reaction following a tendon tap. It has been suggested the T-reflex may be used to measure spasticity.[79]

Levin and Hui-Chan,[77] recruited 10 patients post-stroke and seven age-matched non-disabled controls to examine the reliability of the H-reflex and SR and the correlation between altered reflex functions and the clinical measurement of spasticity using the CSI. Their results showed that H-reflex and SR latencies were shorter ($P < 0.05$), and reflex amplitudes were significantly greater (H/M ratios, $P<0.05$; duration and magnitude of SR, $P<0.005$) in subjects with spasticity. Further, the results of the tests were highly reproducible; however, only the SR was found to be significantly related to CSI data.[77] The same team of researchers later compared stretch reflex threshold regulation in 11 subjects with spastic hemiparesis and 6 non-disabled controls.[56] With the subject seated with both arms and forearms in a hand mold attached to a manipulandum controlled by a torque motor, the elbow was passively and repeatedly extended from 30 degrees flexion through an arc of 100 degrees at 7 different velocities (8-160 degrees/second for subjects with hemiparesis, and 32-300 degrees/second for controls). Displacement and velocity of the forearm and EMG signals from two elbow flexors and two elbow extensors were recorded. Phase diagrams (velocity versus angle) were then plotted and the threshold angles for muscle activation at each velocity of stretch were used to determine the SR threshold and the slope of the relationship between the SR

thresholds and velocity. The results showed that SR thresholds were decreased in the patients post-stroke compared to non-disabled controls and that the thresholds were velocity-dependent. Further, the threshold value correlated with the severity of clinically measured spasticity using the CSI ($r = -0.65$, $p < 0.05$).[56]

Min et al.,[79] examined the relationship between the MAS and EMG-derived amplitude and latency of T-reflex of the biceps brachii muscle in 21 patients with spastic hemiplegia after ischemic stroke. Two investigators conducted the initial assessment and one repeated the assessment protocol one week later to estimate inter-rater and intra-rater reliability. The results showed that amplitude of the biceps' T-reflex increased with increasing level of the MAS for both raters ($rho > 0.46$, $p < 0.01$). However, there was no significant correlation between latency and MAS. Further, the ICCs to assess inter-rater reliability of latency and amplitude of biceps T-reflex were 0.91 and 0.82, respectively. The authors also reported the intra-rater reliability for latency ($rho = 0.94$, $p < 0.01$) and for amplitude ($rho = 0.64$, $p < 0.01$). The investigators concluded that the biceps' T-reflex was a good quantitative measurement of biceps spasticity with acceptable inter- and intra-rater reliability, and moderate correlation with the MAS.[79] However, Fellows and colleagues,[80] noted that while SR response measured by EMG reached its peak at 3 months post stroke and was consistent up to 1-year post stroke, a change in the T-reflex response was observed at the 1-year assessment. The authors concluded that the T-reflex does not provide a complete picture of the pathological changes in the reflex responses in spasticity.[80]

In summary, the validity and reliability of instrumented measures of spasticity show better results when compared to non-instrumented measures. However, most instrumented measures are not developed for clinical use.[8] Further, administering of these instruments and interpretation of results require training, which is another reason why these devices are scarcely used in the clinical setting.

1.4 THE BIOTONE STUDY

1.4.1 CONTEXT OF THESIS

This study was part of a larger study aimed at investigating the BioTone system as a clinical tool to measure spasticity in people with the diagnosis of spinal cord injury (SCI), traumatic brain injury (TBI), multiple sclerosis (MS) or stroke. The aim of the larger BioTone study is to examine the relationship between an assumed "gold standard" of spasticity measurement (i.e., criterion), which is the MAS, and a number of indicators of spasticity obtained using the BioTone, through application of a multiple regression type model. (More details of the BioTone system are provided below).

1.4.2 CONTRIBUTION OF THESIS TO LARGER BIOTONE STUDY

The overall aim of this study was to carefully examine the concurrent and construct validity and test-retest reliability of the BioTone system in a small cohort of people post-stroke. The specific contribution of this thesis to the larger BioTone study was to: (i) examine the concurrent validity of the BioTone spasticity measures by comparing them to the TS in addition to the MAS, (ii) examine the construct validity of the BioTone measures by comparing the variables obtained using a theoretical model of elbow motion to variables obtained from the elbow motion curve of the non-paretic side, and (iii) examine test-retest reliability of the BioTone and clinical measures of spasticity.

1.4.3 SAMPLE SIZE OF BIOTONE STUDY

The BioTone study aims to recruit a total sample of convenience of 160 patients who will be recruited from 4 different sites/hospitals. For the larger study, the software program G*Power 3 was used to determine the appropriate sample size required to detect a significant relationship. Given that (i) five predictors will be used, (ii) the effect size may only be of medium size (due to variance expected in patients) or f2=0.2 (Cohen's effect size index), (iii) the significance level is α=0.01 (to protect against type I error) and (iv) power to detect a significant relationship is β=0.95, the required sample size will be 137 subjects. Assuming that ~ 15% of cases are not suitable for analysis (due to technical difficulties, incomplete examinations, or withdrawal of consent), the total sample to be

recruited is 160 participants from all diagnoses (i.e., 40 participants from each group, SCI, TBI, MS and stroke).

1.4.4 THE BIOTONE SYSTEM

Overview of the BioTone System

The BioTone system is a portable toolkit for conducting comprehensive, quantitative examination of muscle impairment and function in patients with mobility disorders. Developed by the Institute of Biomedical Engineering (IBME) at the University of New Brunswick, the BioTone system consists of multiple instruments that can conduct neurologic and musculoskeletal assessment of upper and lower extremity mobility and impairment. The instruments that compose the BioTone System are the following: 2-channel EMG, electrogoniometer, isometric dynamometer, pendulum test wheelchair, analog to digital converter and a laptop computer that includes a software designed for the BioTone system (Figure 1).

Figure 1. The BioTone system.

Two Channel EMG

Part of the BioTone system is an EMG device that was also developed by IBME. This EMG uses snap connection leads to attach to off-the-shelf, gelled Ag/AgCl, bipolar electrodes that mount directly to the skin with an adhesive pad. The electrode interface provides signal conditioning (BandPass filtered 16-350Hz) and amplification to provide a 0–5V analog voltage signal. The sensor consists of 2 EMG channels with 5 snap leads and a small, flat electronic circuit board (0.5 x 1.5 x 5cm) in-line with the leads and cable.

Electrogoniometer

The ShapeSensor™ is an electrogoniometer that has a single degree of freedom (flexion/extension) sensor that utilizes embedded fiber-optic and electronics technology to provide an analog voltage in the range of 0–5V, proportional to joint angle over approximately +/- 180 degrees of range. Physically, the device consists of 2 small plastic boxes (1.5 x 3.5 x 5.5cm) connected by a 20cm x 0.5cm piece of coated ribbon steel that is allowed to flex and bend to produce the signal (Figure 2).

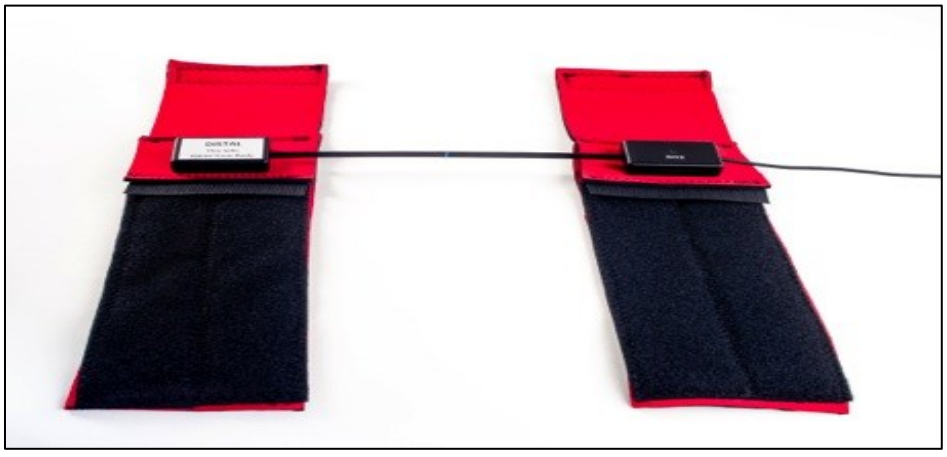

Figure 2. The ShapeSensor™ fibre-optic goniometer.

Isometric Dynamometer

An isometric dynamometer is used to assess the force exerted by the elbow muscles during flexion and extension movements. The device is adjustable to accommodate the direction of movement (i.e., into flexion or extension). The force device is a brace worn between the forearm and the upper arm with a hinge at the elbow. A strain gauge type force transducer is mounted on the device to measure both extension (tension load) and flexion (compression load) and it can measure up to 1300 Newton of force (Figure 3).

Figure 3. Isometric strength dynamometer.

Pendulum Test Wheelchair

The pendulum test wheelchair is a wheelchair modified to perform the pendulum test. This wheelchair has a custom-made seat cushion that does not obstruct the movement of the knee when performing the pendulum test. Additionally, this wheelchair allows adjusting the seat tilt to ensure that the participant's femur is in a horizontal position while performing the test (Figure 4).

Figure 4. Pendulum test wheelchair.

Analog to Digital Converter

The BioSI is a 10-bit analog to digital converter that allows capturing data (sampling) from the multiple sensors and streaming data live to the laptop. The wearable sensors and other devices that make up the toolkit, such as the electrogoniometer and EMG electrodes, are all attached to the BioSI. The BioSI is then attached the laptop computer to display and save data captured using this device.

Laptop Computer with BioTone Software

A laptop computer is used to display and save the data transmitted from the BioSI device through a USB port. Using a software application designed for the BioTone system, the data collected from each of the examination protocols is displayed and saved.

1.4.5 ASSESSMENT PROCEDURE

The BioTone assessment is conducted on the muscles of the elbow on the paretic side of the participant. First, spasticity assessment is conducted using the MAS (Appendix B)[32] to assess the muscles of the affected side elbow muscles. Then, the two-channel 5 electrodes EMG are placed in accordance with SENIAM guidelines on the biceps brachii and on the triceps brachii with the reference electrode on the back of the palm (Figures 5-A, 5-B).[81, 82] The electrogoniometer (ShapeSensor™) is placed inside the pockets of 2 mounting straps, with one strap attached to the upper arm and the other on the forearm. The electrogoniometer is placed on the lateral side of the upper extremity

with the midpoint marker placed adjacent to the elbow joint to accurately measure movement (Figure 5-C). Then, with the participant in the supine position and completely relaxed, the stretch reflex test was conducted for elbow flexion and extension. The movement is performed passively within one second (similar to the approach of the MAS) while EMG and kinematic data were recorded. Three trials are performed for each movement of the elbow. Following the stretch reflex test, active and passive ROM are assessed for the elbow joint for both flexion and extension with the participant in sitting position. Finally, three trials of maximal-effort isometric muscle strength testing of the elbow flexors and extensors are done with the participant in a seated position.

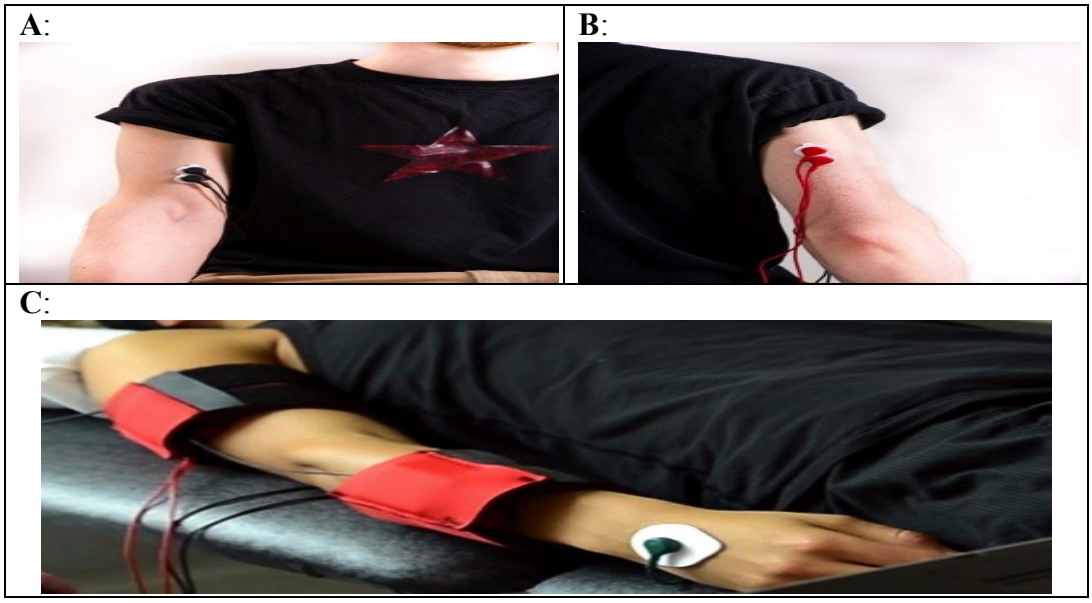

Figure 5. EMG electrodes and electrogoniometer placement for upper extremity. A: electrode placement for biceps brachii. B: electrode placement for triceps brachii. C: Upper extremity after placement of electrodes and electrogoniometer.

1.4.6 DATA PROCESSING

In order to obtain measures of spasticity for the elbow musculature data using the BioTone system, the kinematic and EMG data had to be processed. The procedure for processing the kinematic and EMG data for the elbow muscles, as described by the developers of the Biotone system,[83] is provided in the following sections.

The kinematic data obtained via electrogoniometry reflect both the examiner's passive movement of the participant's UE and any involuntary muscle response (i.e., spastic reaction). Therefore, a motion free from a spastic reaction would result in a motion profile identical to the examiner's intended motion (constant jerk) and there would not be a kinematic departure from the intended motion.[84] Departure from the intended motion (theoretical profile) should correlate with the corresponding EMG intensity. Thus, in the following sections a description is provided as to how a theoretical model was developed, which represents the motion of the participant's UE as if no spastic reaction were present during the stretch test. In addition, how the onset point and angle of the spastic reaction were determined using the kinematic and EMG data is explained; and the process for identifying the variables to be used in the statistical analysis is described.

Theoretical Model of Elbow Motion

The theoretical kinematic model was based on a constant jerk movement induced by the examiner during passive motion of the participant's UE.[84] A custem interactive program was written in MATLAB (MathWorks Inc. Natick, MA) to do the processing of deriving the theoretical model. The electrogoniometric data collected via the BioTone were first low-pass filtered at 6Hz (zero-lag, 4th order Butterworth). The start time (**T1**) and end time (**T2**) of testing were determined to establish maximum and minimum limits of elbow motion. **The maximal slope of motion** was determined from the initial movement prior to the spastic onset. The minimum angle (**Min-Angle**),is the point of the beginning of motion or change in angle. A temporal variable (**tm**) is defined as the time between T1 and the intersection of the slope with a projected line from Min-Angle. The maximum angle (**Max-Angle**) is the point at which change of angle stops. Finally, **Tr** is defined by the intersection of the slope with a line projected from Max-Angle plus tm. Tr represents the (theoretical) time at which end of motion occurs if the actual motion was not disturbed by the spastic reaction (Figure 6).

A theoretical acceleration profile was then developed between T1 and Tr to represent a constant jerk motion. The peak of acceleration is quite sensitive to range of movement and duration; therefore, the acceleration curve was given peaks of +/- 1000

(degrees/second)^2, then integrated to estimate the velocity profile, and then integrated once more to estimate angle profile. The upper and lower limits of motion are known from the actual profile, which were used to scale the theoretical angle profile. Numerical differentiation (5-point Lagrangian method) was used to compute velocity and acceleration profiles for the theoretical and actual profiles.[83]

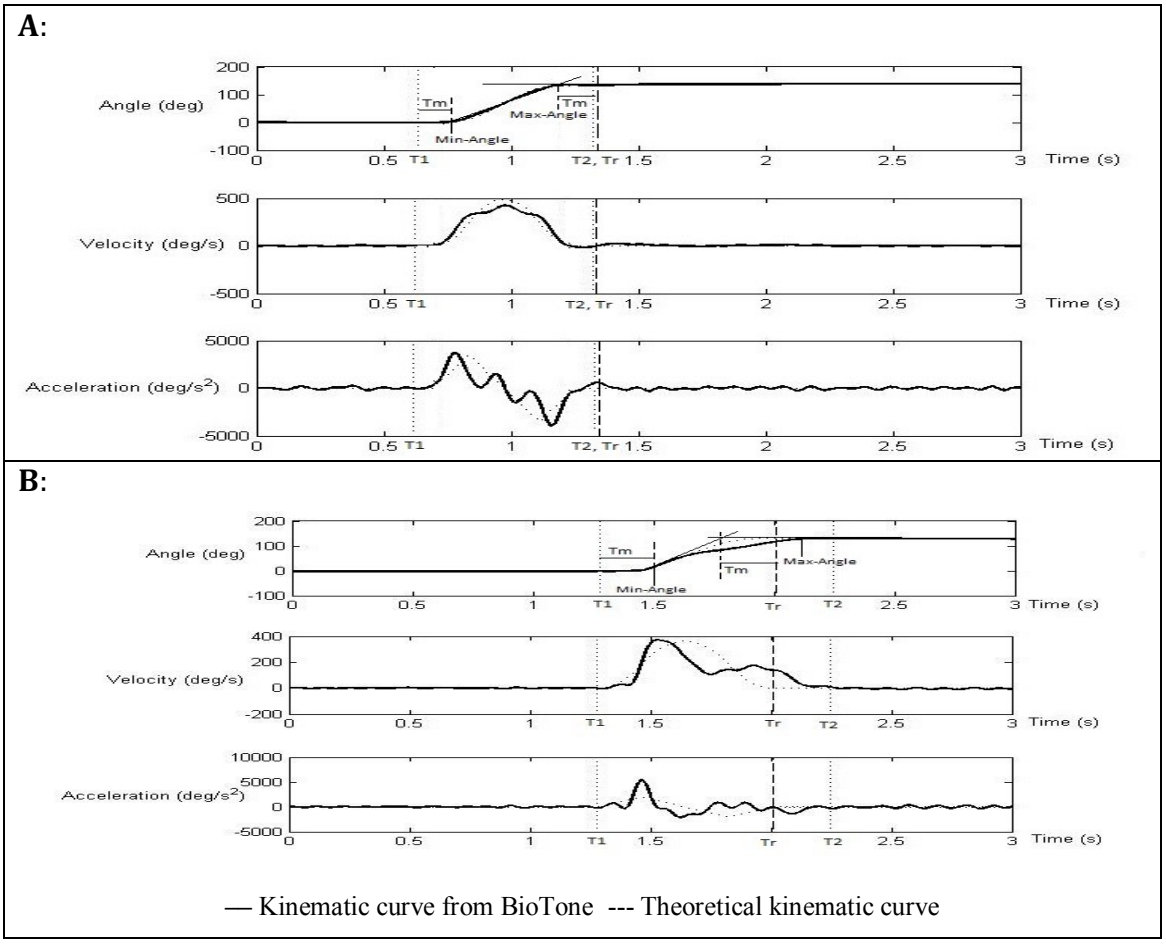

Figure 6: Derivation of, and deviation from, theoretical curve. **A**: Elbow flexion trial (assessing elbow extensors) for the participant 5 on the non-paretic side (MAS=0). **B**: Elbow flexion trial (assessing elbow extensors on the paretic side) for the same participant (MAS=1+). The solid line represents the actual motion, whereas the dashed line represents the theoretical model.

Determining Spastic Onset and Angle in Kinematic Model

The spastic reaction that occurs during the stretch reflex test causes a sudden change in acceleration. Thus, the spastic onset during the stretch reflex test should correspond to the time of maximum departure in acceleration from the theoretical profile. The time of maximum departure in velocity from theoretical profile was determined, and then the time of maximum departure in acceleration from theoretical profile was determined. The time of maximum departure in acceleration from theoretical profile was used to establish spastic onset time (**Tk**) as determined from kinematic data. Also, the angle corresponding to Tk was determined to establish the spastic onset angle (Figure 7).

Determining Tk allowed dividing the kinematic profile into three segments. First was *pre-onset*, which was defined by the time between start of testing and spastic onset (T1-Tk). Second was *post-onset*, which was the time between spastic onset and end of theoretical profile (Tk-Tr). Third was *post-theoretical*, which was the time between end of theoretical profile and the end of testing (Tr-T2) (Figure 7).

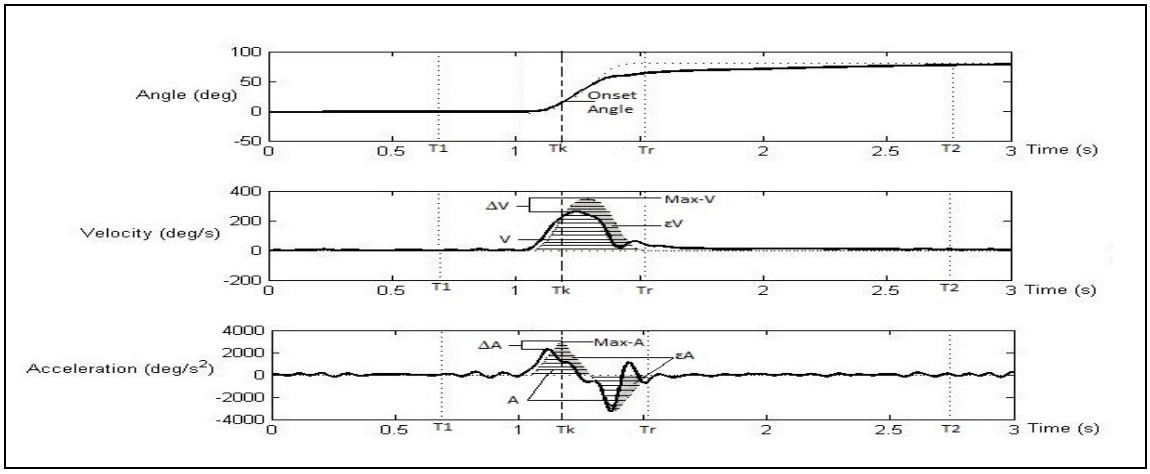

Figure 7: Determining spastic reaction from kinematic data. Elbow flexion trial for participant 10 with MAS=2.

EMG Signal Processing

EMG signal data were processed by band-pass filtering (20-400 Hz), rectifying and then low-pass filtering at 10 Hz. A zero-lag, 4th order Butterworth filter was used for

processing. Spastic onset time was determined from the EMG data through the following procedure. First, the stretched muscle EMG signal was scaled to 0-1 by dividing the signal values by the maximum EMG value recorded during the trial. Then if the averaged scaled-EMG signal increased by more than 20%, the time of first peak EMG signal was selected as EMG onset time (**Te**) (Figure 8). If the change in signal did not exceed 20%, the trial was deemed "no-onset". Further, the difference in onset time (**δT**) between EMG onset and kinematic onset was calculated (Te-Tk).

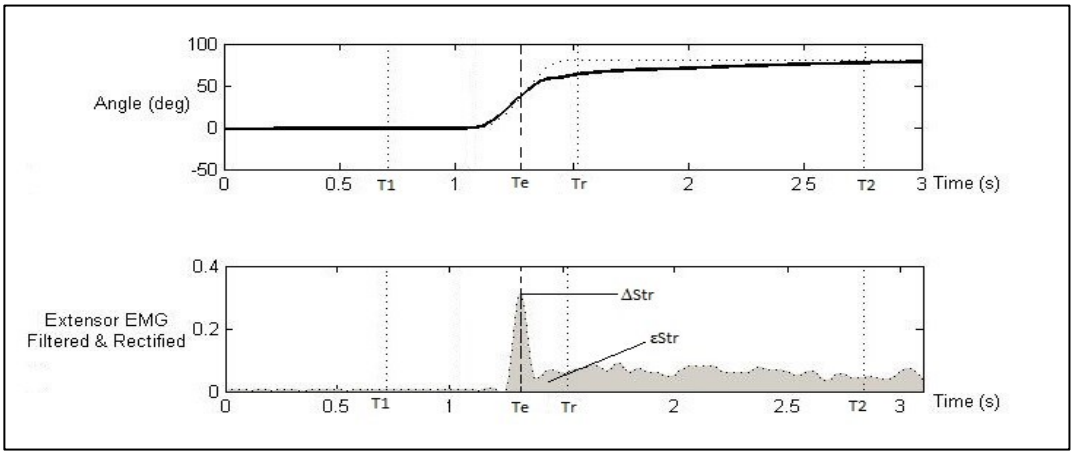

Figure 8: Determining spastic reaction from EMG data

Variables Used To Quantify Spasticity

Spastic muscle reaction during stretch reflex testing can either be sudden or prolonged muscle reaction. Therefore, two types of variables from the previous data may be used to represent the spastic muscle reaction: (i) Muscle reflex intensity, and (ii) Muscle reflex density.

1- Muscle reflex intensity: Two kinematic variables were selected that represent a discrete event during stretch reflex test. These were the peak magnitudes of departures from theoretical profile for velocity (Δ**V**) and acceleration (Δ**A**) (see Figure 7). These variables represent the difference between the theoretical model and the actual kinematic trial. Moreover, two discrete EMG variables were selected which were peak, non-scaled EMG at time Te for stretched muscle (Δ**Str**) and for

the opposite or non-stretched muscle (Δ**NStr**). Also, pre-onset mean EMG signal was subtracted from these values to give measure of change in EMG signal intensity (see Figure 8).

2- Muscle reflex Density: Spastic reaction to stretch reflex test may be a prolonged reaction rather than a sudden one. Therefore, measures that represent resistance to PROM were based on gross deviations from the theoretical profile rather than sudden deviations. These measures represent density departures from theoretical curve. For kinematic data, two variables were determined by quantifying the root mean square departure for angular velocity (**εV**) and acceleration (**εA**) between T1 and T2 (shaded regions in Figure 7). EMG density measures were the area under the curve between T1 and T2 for stretched (**εStr**) and non-stretched (**εNStr**) muscles (shaded region in Figure8).

1.5 STATEMENT OF PURPOSE

The literature review shows that there are a wide set of assessment tools that may be used to measure spasticity. However, various issues are associated with these tools such as inconsistent psychometric properties and feasibility of using the measure in the clinical setting. Therefore, further development of measurement tools that are valid, reliable, and clinically applicable is warranted. One such tool is the BioTone system, which is a portable toolkit for conducting comprehensive, quantitative examination of muscle impairment and function in patients with mobility disorders. The primary purpose of my thesis was to assess, in a preliminary way, the concurrent and construct validity and test-retest reliability of the BioTone technology as a clinical measurement of spasticity among patients following stroke.

In addition, a systematic review was conducted of the evidence for studies that have used clinical measures of spasticity among the patient after stroke. This review is included as chapter in this thesis and has been submitted for publication. I made a substantial contribution to the research and writing of this manuscript

CHAPTER 2

MEASUREMENT OF SPASTICITY AFTER STROKE USING CLINICAL MEASURES: A SYSTEMATIC REVIEW

ALORAINI, S M

GAVERTH, J

YEUNG, E

MACKAY-LYONS, M

SUBMITTED TO *DISABILITY AND REHABILITATION JOURNAL* JULY 2014

ABSTRACT

OBJECTIVE: To identify and appraise the literature on clinical measures of spasticity that have been investigated in people after stroke.

DATA SOURCES: The literature search was done in 4 databases (PubMed, CINAHL, Embase and The Cochrane Library) up to February 2014.

STUDY SELECTION: Studies involving adult patients post stroke that aimed to measure spasticity using a clinical assessment tool were included.

DATA EXTRACTION: Two independent raters reviewed the included articles using a critical appraisal scale and a structured data extraction form.

DATA SYNTHESIS: A total of 40 studies examining 15 spasticity assessment tools in patients post stroke were reviewed. None of the reviewed measurement tools demonstrated satisfactory results for all psychometric properties evaluated, and the majority lacked evidence concerning validity, absolute reliability and responsiveness.

CONCLUSIONS: This systematic review found limited evidence to support the use of most of clinical measures of spasticity for people post stroke. Future research examining the application and psychometric properties of these measures is warranted.

KEYWORDS: systematic review; muscle spasticity; assessment tools; stroke; rehabilitation

2.1 INTRODUCTION

Following stroke, patients present with a variety of sensorimotor disorders such as spasticity, muscle weakness and impaired sensation.[2, 85] Spasticity, a sequela present in 19%[4] - 43%[5] of stroke survivors, is clinically manifested as a resistance to passive stretch.[7, 86] This resistance may be attributed to neural and non-neural components.[1, 7, 8] The neural component — a velocity dependent increase in the tonic stretch reflex[1, 6-8] — is most often referred to as spasticity.[1, 7] Non-neural factors such as changes in the mechanical properties of collagen, tendons and muscle fibers (e.g., loss of sarcomeres, subclinical contractures) also contribute to the resistance.[87] The co-occurrence of neural and non-neural causes of increased passive movement resistance makes measuring spasticity difficult.

The most commonly used clinical tools for measuring spasticity, the Ashworth and the modified Ashworth scales (AS and MAS),[9] are ordinal scales that quantify resistance to passive movement.[31, 32] However, the validity of these scales has been questioned because they do not distinguish between neural (spasticity) and non-neural factors contributing to the resistance.[10-12] Further, these ordinal-based scales may not be considered objective especially that they are subject to the rater's own interpretation of the definition of each level of the spasticity measure.[12, 33, 88] On the other hand, instrumented approaches for measuring spasticity, by using different combinations of torque and electromyography (EMG) measurements, may more accurately quantify spasticity.[75, 89] However, expensive equipment requiring specific training to operate and lack of standardized protocols make this type of assessment difficult to implement in a clinical setting.[8] Nevertheless, there is a need for objective clinical tools that are quick and easy to use, and that generate information that can easily be interpreted by the clinician.[8] Over the past few decades' attempts have been made to address this gap but clinical implementation has lagged.[73, 74, 90] Lack of uptake may be because the methods have not been fully developed for clinical use and the psychometric properties of these methods for application to specific diagnostic groups are not well known. Whether the assessment tool used to measures spasticity is instrumented or not, knowledge of the performance characteristics and limitations of spasticity measures for particular patient

populations is necessary for accurate interpretation of assessment findings and subsequent clinical decision-making.[91, 92]

Given that stroke has an impact on long-term disability,[93] spasticity may interfere with stroke recovery,[27] and clinical presentation of spasticity following stroke differs from other conditions,[94] critical appraisal of clinical tools available to measure spasticity after stroke is warranted. To date, reviews on this topic have not focused on clinical devices and have not been specific to the stroke population.[75, 89, 95-97] Therefore, the aim of our systematic review was to identify and appraise the literature on clinically usable measures of spasticity that have been investigated in people after stroke.

2.2 METHODS

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.[98]

2.2.1 LITERATURE SEARCH

A comprehensive literature search was conducted in (i) Pubmed (1950-), (ii) CINAHL (1982-), (iii) Embase (1966-), and (iv) The Cochrane library (1993-). All searches included available records until February 2014 using the following keywords and Medical Subject Headings (MeSH terms): (1) Stroke OR Cerebrovascular Accident OR Hemipleg*, (2) Measur* OR Assess*, and (3) Spas* OR Hyperton*. The searches in all databases were combination as follows: 1 AND 2 AND 3. No limits were applied to the search.

2.2.2 STUDY SELECTION

Two independent reviewers (SA and EY) reviewed the title of each article for initial selection. Abstracts of articles that appeared to be related to the measurement of spasticity were then read and the full articles of relevant abstracts were retained for further examination. The same reviewers read the full text of the papers remaining from the previous stage to confirm that the studies met the inclusion criteria: (i) human adults (≥18 years of age); (ii) participants diagnosed with stroke; (iii) stated objective of the study was to measure spasticity, which had to be in a passive condition by eliciting the stretch reflex (Lance, 1980)[6] ; (iv) spasticity measurement under study was a clinical

scale or a device for use in clinical practice as indicated by the authors; (v) original research articles written in English and published in peer-review journals. Reviews, reports, case studies and studies that involved <10 patients were not included. In the event that the two reviewers had different results, the discrepancy was resolved through consensus.

2.2.3 DATA EXTRACTION AND QUALITY ASSESSMENT

Data regarding patients' characteristics, assessment tools used, tested segment(s), objective of the study and psychometric properties studied were extracted using a structured form. All measurement tools used in the studies were examined in terms of general qualities as measurement tools, practical quality, and quality specific to the measurement of spasticity. The studies included in the review were also assessed using a 12-item critical appraisal scale that rates the quality of different aspects of the included studies.[99] This scale has been used in previous systematic reviews examining rehabilitation assessment tools.[96, 100]

2.3 RESULTS

Forty articles describing fifteen different instruments or measures that met the selection criteria (Figure 9). The critical appraisal of the quality of each study is presented in Table 1, which shows a wide range of study quality, with the highest score on the 12 items ranging from 2 to 10. The frequency of high scores was greatest for 'Data' (n=34 of 40 papers) and 'Statistics' (n=31) whereas none of the studies included sample size justification and only seven (n=7) studies presented hypotheses. Table 2 provides a summary of each of the 40 studies, which are divided into six categories: Ashworth scales (n=18 studies), Tardieu scales (n=5), multi-item scales (n=4), electrophysiological (n=3 studies), pendulum test (n=1), and force/torque measurements (n=9). Below is a brief description of each of the assessment measures.

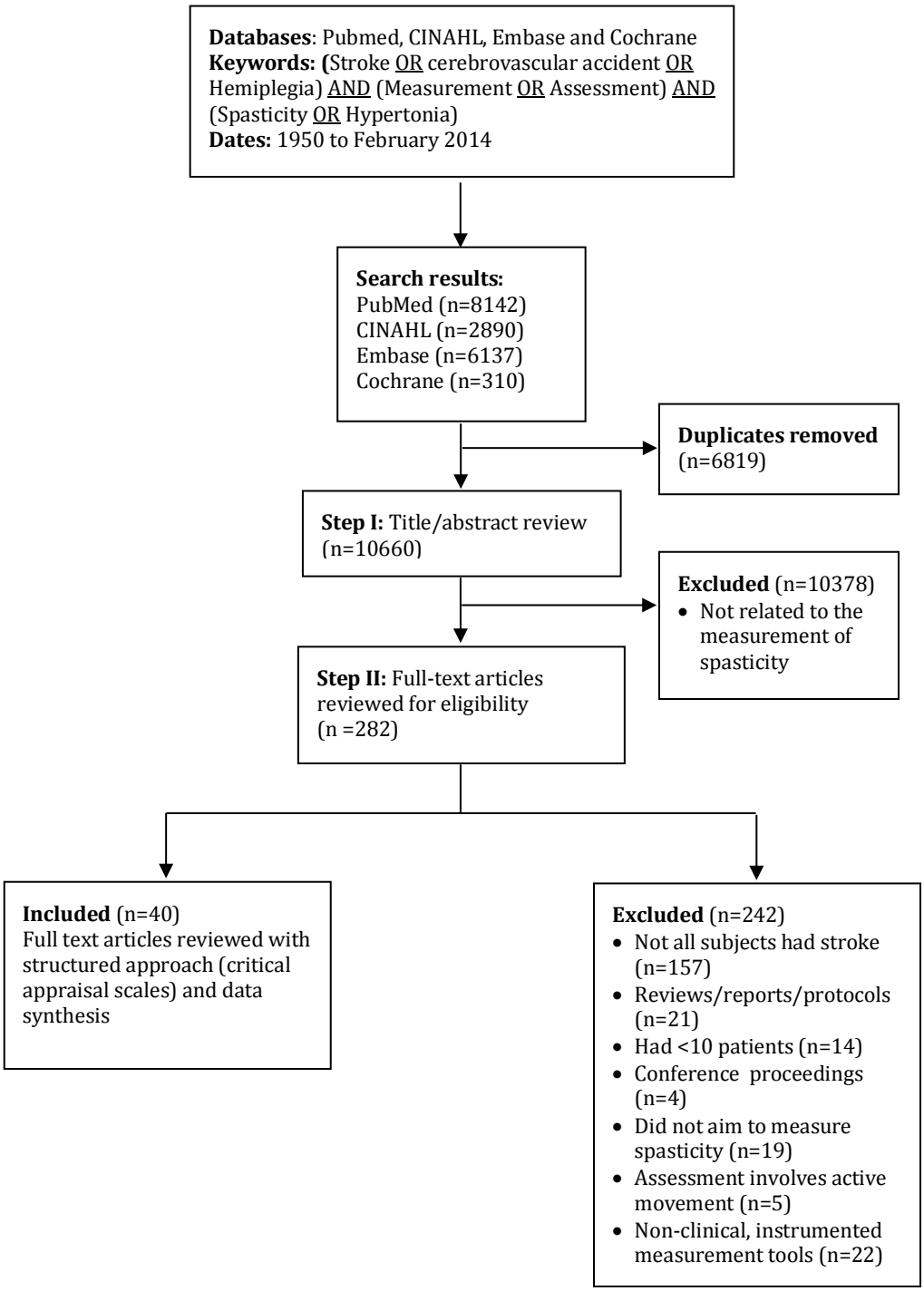

Figure 9. Flowchart of search and selection strategy

Table 1. Quality of studies rated with critical appraisal scale*

Studies	Background	Criteria	Hypothesis	Scope	Sample	Retention	Measure	Methods	Data	Statistics	Secondary	Conclusions
Calota et al.[90]	2	2	2	2	0	2	2	2	2	2	0	2
Ansari et al.[45]	2	1	2	2	0	2	2	2	2	1	1	2
Voerman et al.[72]	2	2	0	2	0	2	2	2	2	2	2	1
Blackburn et al.[101]	2	2	0	2	0	2	2	2	2	2	0	2
Gaverth et al.[102]	2	2	0	2	0	2	2	2	2	2	0	2
Kim et al.[103]	2	1	2	1	0	NA	2	2	2	2	2	2
Naghdi et al.[51]	2	2	0	2	0	NA	2	2	2	2	2	2
Ansari et al.[54]	2	2	1	2	0	1	2	2	2	1	0	2
Lindberg et al.[74]	1	2	0	2	0	NA	2	2	2	2	2	2
Naghdi et al.[37]	2	2	0	2	0	NA	2	2	2	2	1	2
Patrick and Ada,[34]	1	2	2	2	0	NA	1	2	2	1	2	2
Paulis et al.[52]	1	2	2	2	0	2	1	2	2	2	0	1
Kaya, et al.[104]	2	2	0	2	0	NA	2	2	2	2	0	2
Malhotra et al.[69]	2	2	0	2	0	NA	2	2	2	2	0	2
Naghdi et al.[42]	1	2	0	2	0	NA	2	2	2	2	1	2
Singh et al.[53]	2	2	0	2	0	2	1	2	2	1	0	2
Sorinola et al.[105]	1	2	0	1	0	1	2	1	2	2	2	2
Naghdi et al.[41]	2	1	0	2	0	NA	2	2	2	2	1	1
Gregson et al.[106]	1	1	0	1	0	2	2	2	2	2	0	1
Kumar et al.[107]	1	1	0	2	0	NA	1	1	2	2	2	2
Takeuchi et al.[108]	2	2	0	2	0	2	1	1	2	1	0	1
Vattanasilp and Ada,[35]	1	1	0	2	0	NA	2	2	2	2	0	2
Wu et al.[109]	1	1	0	1	0	2	1	2	2	2	0	2
Ansari et al.[46]	1	1	0	2	0	NA	1	2	2	2	0	2
Cooper et al.[39]	1	2	0	2	0	NA	1	1	2	2	0	2
Gregson et al.[44]	1	1	0	2	0	2	0	2	2	2	0	1
Min et al.[79]	1	2	0	2	0	1	2	0	2	2	0	1
Pandyan et al.[70]	2	1	0	2	0	NA	2	1	2	2	0	1
Bakheit et al.[110]	1	2	0	2	0	NA	2	2	1	1	0	1
Barnes et al.[111]	1	0	0	2	0	NA	2	2	1	2	0	2

Studies	Background	Criteria	Hypothesis	Scope	Sample	Retention	Measure	Methods	Data	Statistics	Secondary	Conclusions
Chen et al.[112]	1	2	0	1	0	2	1	1	1	2	0	1
Pandyan et al.[71]	1	1	0	1	0	NA	1	1	2	2	0	2
Lee et al.[73]	1	0	0	1	0	NA	2	0	2	2	0	2
Milanov, I.[15]	2	0	0	2	0	NA	0	1	2	2	0	1
Pandyan et al.[40]	0	2	0	2	0	NA	0	0	1	2	2	1
Pisano et al.[38]	2	1	0	2	0	NA	0	0	2	2	0	1
Pomeroy et al.[113]	1	1	1	1	0	NA	1	2	1	1	0	1
Worley et al.[114]	1	1	0	1	0	NA	2	1	2	1	0	1
Ghotbi et al.[36]	0	0	0	1	0	NA	0	1	2	2	0	2
Lin et al.[115]	0	1	0	0	0	NA	2	2	1	1	0	1

Background: Relevant background on spasticity measurement and research question, **Criteria**: Inclusion/exclusion criteria, **Hypothesis**: Specific hypothesis, **Scope**: Appropriate scope of psychometric properties, **Sample**: Appropriate sample size calculation, **Retention**: appropriate retention/follow-up, **Measure**: Specific descriptions of the measures (administration, scoring, interpretation procedures), **Methods**: Standardization of methods, **Data**: Data presented for each hypothesis or purpose, **Statistics**: Appropriate statistical tests, **Secondary**: Appropriate secondary analyses, **Conclusions**: Conclusions/clinical recommendations supported by analyses and results, **NA:** Not applicable

*maximal score=2 , minimal=0

2.3.1 DESCRIPTION OF THE MEASUREMENT TOOLS

Ashworth Scales

(i) Ashworth Scale (AS): The AS, suggested by Ashworth in 1964,[31] is a 5-point ordinal scale that qualifies the resistance (tone increase) of muscles to passive movement.[31]

(ii) Modified Ashworth Scale (MAS): The MAS is a 6-point ordinal scale, which is a modification of the original AS. Bohannon and Smith,[32] found that many patients demonstrated levels of spasticity between the level of 1 and 2 on the AS. In order to make the scale more discrete, they added a grade (1+) between 1 and 2 of the AS.[32]

(iii) Modified-Modified Ashworth Scale (MMAS): Ansari et al.[33] further modified the MAS to address the issue of accurately grading levels of spasticity in light of the documented AS disagreement between grades 1 and 2 and the MAS disagreement between 1 and 1+, and 1+ and 2.[33] The investigators reasoned that the poor reliability of the AS and MAS may be due to the lack of clarity of grade definitions. To address this issue, the investigators suggested using a 5-point ordinal scale (0-4) but with different definitions than the AS or MAS.[33]

Tardieu Scales

(i) Tardieu Scale (TS): Tardieu et al.[47] used EMG to determine the reflex activity when the elbow was passively extended at a variety of speeds. They concluded that a stretch reflex was elicited at a specific speed (or faster) for the respective patient and postulated that this speed would vary with different subjects. Held and Pierrot-Deseilligny further developed Tardieu's work, publishing a scale in 1969.[48] The TS rates the resistance of spastic muscles to passive stretching at three different velocities on a 6-point ordinal rating scale and measure the angle of catch resulting from passive stretch using a goniometer.[49]

(ii) Modified Tardieu Scale (MTS): Boyd and Graham,[50] modified the TS for the purpose of standardizing the procedure of spasticity assessment. The MTS uses the same 6-point ordinal scale of the TS but has two main differences. Only two speeds are utilized for passive stretch, slow to assess passive range of motion and fast to assess the spastic

reaction. The MTS quantifies the difference between the angle of 'catch' (R1) and the full range of motion (R2). The authors suggest that a large difference between R1 and R2 is indicative of spasticity whereas a small difference is suggestive of passive restraint (i.e., muscle stiffness or contracture).[50]

Multi-item Scales

(i) Tone Assessment Scale (TAS): The TAS consists of 12 questions, subdivided into three sections.[111] The first section, which includes questions 1-3, evaluates the resting posture of the patient in different segments of the body (e.g., shoulder symmetry). The second section, which includes questions 4-9, grades the response to passive movement in different joint of the body both in lower and upper extremities using the ordinal scale of the MAS. The final section, which comprises questions 10-12, assesses movement in response to active efforts to check for abnormal movements (e.g., associated reactions).[111]

(ii) Categorization of Tone and Visual Analogue Scale (VAS): One study in our review described categorizing tone and the VAS as two approaches to assess spasticity.[113] For tone categorization, a clinician assessed the limb and then classified it as spastic, flaccid or normal. For the VAS, the raters evaluated muscle tone using a 100-mm vertical VAS with anchor points at the top and bottom for highest and lowest muscle tone respectively.[113]

(iii) Ankle Plantarflexors Tone Scale (APTS): The APTS is divided into 3 subtests.[108] The first subtest is the stretch reflex test, which evaluates the planter flexors reaction to fast passive stretch into dorsiflexion, and grades the reaction into 5 grades. The second subtest is middle range resistance, which evaluates the resistance felt by the rater during slow passive range of movement of the ankle in the middle range and graded from 0-4. The final subtest is final range resistance, which is similar to the middle range subtest and uses similar grades and performed on the final range of ankle dorsiflexion.

(iv) 3-item Hypertonus Measure: Worley et al.[114] suggested using a 3-item measurement tool for evaluating spasticity. The first item of this tool is the measurement of the resting angle, where the resting angle of the affected limb is measured while it is relaxed at the patient's side. The next item is the measurement of the resistance angle.

Similar to the Tardieu scale, the angle of catch is noted and measured. The final item is the quality of resistance to passive movement. First the limb is moved slowly across the possible range of movement to note the available range. Then, a fast, 1-second, stretch test is done and the resistance is rated using a 5-point ordinal scale.

Electrophysiological Measures

(i) The tonic stretch reflex threshold (TSRT) is obtained by manually stretching the spastic muscle at various fast speeds.[90, 103] The joint angle is recorded with an electrogoniometer and the myoelectric response is recorded using surface EMG. The TSRT is estimated using linear regression (stretch reflex threshold angle and velocity). By extrapolating the regression line to intercept with the velocity axis at 0°/second, a TSTR angle is determined.[90, 103]

(ii) With the patient attached to a wrist rig, manually imposed sinusoidal muscle stretches of the spastic muscle are performed at various speeds guided by a target trace on a monitor. Surface EMG amplitude is normalized to that of the maximal voluntary contraction (MVC) and used as a measure of spasticity.[105]

Pendulum Test

The knee pendulum test has been applied to the elbow joint using electrogoniometry and EMG. The test measures the tone of the elbow flexors muscles, where the elbow is moved in a simulated pendulum motion with the aid of an accessory apparatus. Four parameters are obtained: (i) stiffness, (ii) threshold angle, (iii) viscosity, and, (iv) damping ratio, with the latter defined as an index of spasticity.[115]

Force/Torque Measurements

(i) A computer-controlled step motor system, the NeuroFlexor™, measures resistance induced by passive wrist extension. Through biomechanical modeling, the reactive force recorded during slow and fast velocities is used to estimate elastic, viscous, and neural components of the resistance.[74, 102]

(ii) Manual slow and fast ramp stretches are imposed, and the reactive force and joint angle are recorded using a force transducer and an electrogoniometer. The resistance to passive movement (RPM), which is difference between slope of the force-angle curve

from slow and fast stretches obtained using linear regression techniques, is used as an index of spasticity. In three studies surface EMG was used and the difference in mean squared amplitude between slow and fast stretches is used as an index of spasticity.[69-72]

(iii) Manually imposed sinusoidal muscle stretches are performed at various speeds and the reactive force is recorded. Using a dynamic equation of movement the reactive resistance is divided into inertial, elastic and viscous components.[73, 109, 112] The viscous component is estimated based on the relationship between externally imposed joint displacements and the corresponding joint resistance and used as the measurement index of spasticity.

Table 2. Summary of studies

Study	Measure(s) used	Population	*n*	Segment tested	Objective of study
Ashworth Scales					
Ansari et al.[46]	MMAS	Mean age=60y, 5 F/16 M; median time since stroke= 11m	21	Elbow	Inter-rater reliability of the MMAS.
Ansari et al.[45]	MMAS	Mean age =67y, 8 F/7 M; mean time since stroke= 14.1m	15	Knee	Inter-rater and Intra-rater reliability of the MMAS
Bakheit et al.[110]	MAS and H-reflex	Mean age= 60.8y, 8 F/17 M; mean time since stroke= 13.7m	25	Ankle	Investigate correlation between MAS and neuro-physiological tests
Blackburn et al.[101]	MAS	Mean age= 76.1y, 19 F/17 M; time since stroke= 2w-12w.	36	Knee and Ankle	Inter-rater and intra-rater reliability of MAS
Cooper et al.[39]	MAS and EMG	Mean age= 65y; mean time since stroke= 29m	31	Knee and ankle	Validity of the MAS by comparing to surface EMG
Ghotbi et al.[36]	MAS and H-reflex	Mean age= 61.8y, 3 F/17 M; mean time since stroke= 20.7m	20	Ankle	Validity of the MAS by comparing to H-reflex
Gregson et al.[106]	MAS and TAS	Median age= 74y, 14 F/18 M; median time since stroke= 48d	32	Elbow	Inter- and intra-rater reliability of the TAS and MAS.
Gregson et al.[44]	MAS	Median age 73y, 15 F/20M; median time since stroke=40d	35	Elbow, wrist, knee and ankle	Inter-rater and intra-rater reliability of the MAS
Kaya, et al.[104]	MAS and MMAS	Mean age= 60.5y, 23 F, 41 M; mean time since stroke= 15.7w	64	Elbow	Inter-rater reliability of MAS and MMAS.
Kumar et al.[107]	MAS and torque measurement	Median age= 72y, 45 F/66 M; median time since stroke= 11m	111	Elbow	Validity of the MAS by comparing it to resistance to passive movement
Milanov[15]	MAS, T-reflex, H-reflex and EMG	Mean age= 57.9y, 87 F/ 33 M; mean time since stroke= 36.3m	120	Not specified	Correlation between neuro-physiological and clinical methods for evaluating spasticity
Min et al.[79]	MAS, T-reflex and EMG	Mean age= 58y, 8 F/14 M; mean time since stroke= 32.2d	21	Elbow	Correlation between MAS and amplitude and latency of T-reflex.

Study	Measure(s) used	Population	*n*	Segment tested	Objective of study
Naghdi et al.[41]	MMAS and H-relex	Mean age= 58.9y, 7 F/5 M; mean time since stroke= 27.2m	12	Wrist	Validity of the MMAS by comparing to H-reflex
Naghdi et al.[42]	MMAS and H-reflex	Mean age= 57.9y, 14 F/13 M; mean time since stroke= 20.8m	27	Wrist	Validity of the MMAS by comparing to H-reflex
Naghdi et al.[37]	MAS and H-reflex	Mean age= 57.9y, 14 F/13 M; mean time since stroke= 20.8m	27	Wrist	Validity of the MAS by comparing to H-reflex
Pandyan et al.[40]	MAS and torque measurement	Patients post stroke. Characteristics not reported.	63	Elbow	Validity of the MAS by comparing it resistance to passive movement
Pisano et al.[38]	MAS, H-reflex, EMG and torque measurement	Mean age= 45.3y, 14 F/39M; mean time since stroke= 30.7m	53	Wrist	Correlating MAS with EMG and biomechanical measures
Vattanasilp and Ada[35]	AS, tendon jerk, EMG and torque measurement	Mean age= 66.5y; time since stroke= 2w-5y.	44	Ankle	Examine relationship between different measures of spasticity
Tardieu Scales					
Ansari et al.[54]	MTS	Mean age= 57.5y, 13F/17 M; mean time since stroke= 28.5m	30	Ankle	Inter-rater and Intra-rater reliability of the MTS
Naghdi et al.[51]	MTS, H-reflex and EMG	Mean age= 52.7y, 9 F/11 M; mean time since stroke= 33m	20	Wrist	Validity of the MTS by comparing to H-reflex
Patrick and Ada[34]	AS, TS, EMG and torque measurement	Mean age= 63y, 5 F/11 M; mean time since= 3y	16	Elbow and Ankle	Validity of the TS by comparing it to AS and to EMG and resistance to passive movement
Paulis et al.[52]	TS	Mean age= 70.2y, 6 F/7 M.	13	Elbow	Test–retest and inter-rater reliability of TS.
Singh et al.[53]	MTS	Mean age= 64y, 37 F/54 M; mean time since stroke= 57.5d	91	Elbow and ankle	Intra-rater reliability of the MTS
Multi-item Scales					
Barnes et al.[111]	TAS	Median age= 72y, 9 F/6 M; median time since stroke= 5m	15	General	To develop a reliable, multi-item, ordinal scale and assess inter-rater reliability
Pomeroy et al.[113]	Categorization of tone and VAS	Patients post stroke. Characteristics not reported.	39	Elbow and knee	Inter-rater reliability of clinical categorization of tone (spastic / normal / flaccid) and VAS
Takeuchi et al.[108]	MAS, TS, APTS and torque measurement	Mean age= 77.5y, 43 F/31 M; mean time since stroke= 1084.2d	74	Ankle	To develop and evaluate the reliability and validity of the APTS
Worley et al.[114]	3-item, hypertonus measure	Mean age= 66.6y, 28 F/17 M; mean time since stroke= 41d	45	Elbow	Determine reliability of a potential measure of hypertonus
Electrophysiological Measures					
Calota et al.[90]	TSRT	Mean age (62.9y), 4 females and 16 males. Mean time since stroke (77.9m)	20	Elbow	Intra-rater and inter-rater reliability of TSRT and correlation between it and MAS
Kim et al.[103]	TSRT	Mean age (63.5y), 8 females and 7 males. Mean time since stroke (6.7m)	15	Elbow	Develop a portable system for quantifying spasticity, using TSRT and its relationship with MAS

Study	Measure(s) used	Population	*n*	Segment tested	Objective of study
Sorinola et al.[105]	Normalized EMG by MVC	Mean age (53.7y), 4 females and 6 males. Mean time since stroke (30.9m)	10	Wrist flexors	Reliability of a rig to measure spasticity and relationship with EMG and MAS
Pendulum Test					
Lin et al.[115]	Damping ratio	Mean age (57.7y), all males.	11	Elbow	Modify Pendulum test to use on elbow joint
Force/Torque Measurement					
Lindberg et al.[74]	NeuroFlexor	Mean age (61.3y), 14 females/ 17 males. Mean time since stroke (5.3y)	31	Wrist	Development and validation of the NeuroFlexor
Gaverth et al.[102]	NeuroFlexor	Mean age (53.8y), 7 females/ and 27 males. Mean since stroke (5 y)	34	Wrist	Test-retest and inter-rater reliability the NeuroFlexor
Pandyan et al.[70]	RPM	Mean age (67.3 y), 6 females/10 males.	16	Elbow	Develop a biomechanical measure of spasticity
Pandyan et al.[71]	RPM and EMG	Median age (61y), 6 females/ 8 males. Median time since stroke (48m)	14	Elbow flexors	Validity of a biomechanical device for measuring spasticity
Voerman et al.[72]	RPM and EMG	Mean age (56.6y), 2 females/10 males. Mean time since stroke (3.9y)	12	Wrist	Sensitivity, inter-rater/ test-retest reliability and validity of the device
Malhotra et al.[69]	RPM and EMG	Median age (74y), 46 females/ 54 males. Median time since stroke (3w)	100	Wrist flexors	To quantify agreement between MAS, EMG and a clinically usable device for measuring spasticity
Lee et al.[73]	Viscosity Component	Mean age (57y). Time since stroke (2w-5y)	15	Elbow	Develop a portable device for quantifying spasticity
Wu et al.[109]	Viscosity Component	Mean age (57.5y), 8 females and 5 males. Mean time since stroke (31m)	13	Elbow	Utilizing a device to measure spasticity before and after Botox
Chen et al.[112]	Viscosity Component	Mean age (57.3y), 6 females and 4 males. Mean time since stroke (37.7m)	10	Elbow	Responsiveness of device before and after Botox

n: Number of participants; **MMAS**: Modified-Modified Ashworth Scale; **F**: female; **M**; male; **MAS**: Modified Ashworth Scale; **H-reflex**: Hoffman's Reflex; **EMG**: Electromyography; **TAS**: Tone Assessment Scale; **T-reflex**: Tendon Jerk Reflex Measured using EMG; **AS**: Ashworth Scale; **MTS**: Modified Tardieu Scale; **TS**: Tardieu Scale; **VAS**: Visual Analogue Scale; **APTS**: Ankle Planter flexors Tone Scale; **TSRT**: Tonic Stretch Reflex Threshold; **MAS**: Modified Ashworth Scale; **MVC**: Maximal voluntary contraction; **RPM**: Resistance to passive movement; **EMG**: Electromyography; **y**: years; **m**: months; **w**: Weeks; **d**:days.

2.3.2 VALIDITY OF THE MEASUREMENT TOOLS

Several methods have been used to explore the validity of the different scales and instruments in the studies included in our review. These include correlations with clinical scales and with different neurophysiological measures as well as the ability of the tool to discriminate between measured responses in affected and non-affected muscles. The aforementioned modified Ashworth scale was often used as a bench mark for other measures based on its merit as the most commonly used clinical scale. Different applications of the surface electromyography (EMG) were also used including the H-reflex. The EMG is a direct way of measuring the reflex response to stimuli and an expression of spasticity as defined by Lance.[6] Examples of H-reflex parameters used in the included studies are the amplitude, latency, ratio between the amplitudes of the H-reflex and the muscle response to an electrical impulse to a peripheral nerve, usually the tibial or median nerve (H_{max}:M_{max} ratio) as well as the ratio between the slope of the H-reflex and M-response recruitment curves (H_{slope}:M_{slope} ratio). Both ratios have been used to quantify the excitability of the motor neurons.[75]

Ashworth Scales

In the study by Patrick and Ada,[34] they defined the presence of spasticity as a score of 1 or greater on the AS. Their results showed that the AS had a 63% agreement (Kappa=0.24, p=0.02) with the presence of spasticity as determined by EMG.[34] Further, the authors found that there was no significant correlation between the AS and fast stretch-induced EMG-activity.[34] Conversely, the AS was found to have a significant correlation with RTPM measured using a torque system (rho=0.52-0.64, $p<0.01$).[35]

Studies investigating the validity of the MAS have reported conflicting results. Bakheit et al.[110] used the Hoffman's reflex (H-reflex) by electrically stimulating the tibial nerve and then using EMG placed over the ankle planterflexors to record the magnitude of the first response (i.e., M-wave), and then the magnitude of the second response, (i.e., H-reflex) to estimate ratio of the H-reflex to the M-wave (Hmax:Mmax ratio). The authors found that there was not significant differences in the H-reflex parameters (H-reflex latency and Hmax:Mmax ratio) between two groups who have different MAS scores of the ankle planterflexors (MAS 1 and 2).[110] Also, 2 studies have shown that

there were no significant differences in RTPM when comparing different groups who have different MAS scores.[40, 107] However, Pandyan et al.[40] reported that there is a significant difference ($p < 0.01$) in RTPM between patients who score '0' on the MAS and patients who had a score grater than '0'. Cooper et al.[39] who categorized EMG responses to stretch test as (i) no response, (ii) short, (iii) sustained or (iv) undetermined, showed that the MAS had a significant correlation with type of response (Cramer's V= 0.27, p= 0.023). The MAS has not been shown to significantly correlate with the parameters of the H-reflex (latency, amplitude, Hmax:Mmax ratio and Hslope:Mslope ratio).[15, 36-38] As well, Milanov[15] reported that there were no significant correlations between the MAS and any of the EMG-measured manual tendon reflex parameters (amplitude and latency). However, Min et al.[79] showed that there was a low correlation between the MAS and the amplitude of the tendon reflex in the biceps brachii muscle (rho=0.46-0.57, $p<0.01$) but no significant correlation between MAS and the tendon reflex latency measured using EMG.[79]

The MAS has been shown to have significant correlations with EMG parameters obtained during stretch tests. Cooper et al.[39] demonstrated that the MAS correlated with magnitude of EMG response to stretch test (rho=0. 21, p=0.022) and Pisano et al.[38] reported that MAS correlated with stretch reflex latency (rho=-0.37, $p<0.01$) and stretch reflex area (rho=0.36, $p<0.01$). Similarly, RTPM was shown by two studies to have a significant correlation with MAS (rho=0.51, rho=0.55, $p<0.01$).[38, 40] Finally, the MAS was found to have a significant negative correlation with the speed of the stretch reflex test (rho=-0.46, $p<0.001$).[38]

In regard to the MMAS, only two studies investigated its validity.[41, 42] The first study showed that there was no significant correlation between the MMAS and the H-reflex parameters.[41] However, the second study found that the MMAS did have a significant correlation with both the H-reflex Hmax:Mmax and Hslope:Mslope ratios (rho=0.39, rho=0.39, p=0.04).[42] This study also found that the MMAS was correlated with the H-reflex Hslope (rho=0.45, p=0.02).[42]

Tardieu Scales

In the aforementioned study by Patrick and Ada,[34] they defined the presence of spasticity as a score of 2 or higher on the TS. Their results showed that there was a 100% agreement (Kappa=1.0) with the presence of spasticity as determined by EMG.[34] Further, the authors found that there was a significant correlation between the TS and fast stretch-induced EMG activity (Pearson's r=0.62-0.86, $p<0.01$).[34] Regarding the MTS, Naghdi and colleagues,[51] found that there were significant differences ($p<0.001$) between two groups who have different MTS scores (MTS 0 and 2) in terms of R1, R2 and R2-R1 of the MTS. However, the authors noted that there was no significant correlation between the MTS and the H-reflex parameters.[51]

Multi-item Scales

Of the four scales in this category, only one, the APTS, was examined in relation to other spasticity assessment tools. None of the studies included in this review investigated the validity of the TAS, categorization of tone and VAS and the 3-item hypertonus measure. Takeuchi et al.[108] found that the APTS stretch reflex subtest had significant correlations with the TS (rho=0.85-0.94, $p<0.01$), and with the MAS (rho= 0.26-0.41, $p<0.05$) but was not correlated with RTPM. Further, the middle range subtest showed significant correlations with the TS (rho= 0.31-0.42, $p<0.01$), with the MAS (rho= 0.59-0.62, $p<0.01$) and with RTPM (rho= 0.44-0.50, $p<0.01$). Finally, the authors found that the final range subtest showed significant correlations with the TS (rho= 0.30-0.31, $p<0.01$), with the MAS (rho= 0.67-0.68, $p<0.01$) and with RTPM (rho= 0.55, $p<0.01$).

Electrophysiological Measures

Evidence of validity of the TSRT has been presented showing the ability to discriminate between groups of patients with different severity of spasticity.[103] However, there are conflicting results in the association between TSRT and MAS scores — Kim et al.,[103] showed a strong association (rho= -0.74, $p<0.05$) between TSRT and MAS whereas Calota et al.,[90] did not (rho = -0.26, $p>0.44$). With the electrophysiological approach published by Sorinola et al.,[105] a strong association (rho=0.72, $p<0.05$) was

found between MAS scores and the normalized EMG amplitude at low speed of stretch test (50-74 degrees/second) although this association was not present at higher speeds.

Pendulum Test

Lin et al.,[115] showed that the damping ratio derived from the elbow pendulum test was significantly different between the two sides of patients post stroke, and also different when compared to non-disabled individuals .

Force/Torque Measurements

Lindberg et al.,[74] reported that the neural component of the passive movement resistance measured using the NeuroFlexor method was both velocity dependent and associated with integrated EMG during the movement ($rho>0.58$, $p < .001$). They also demonstrated that both the EMG response and the neural component were significantly reduced during an ischemic nerve block test. In addition, a moderate correlation between the neural component and MAS scores was found ($rho>0.6$, $p<0.001$).

Evidence of validity of the second approach using force/torque measurements (manual slow and fast ramp stretches) was found to be contradictory. Voerman et al.,[72] reported that the RPM parameters were not significantly different between patients with spasticity and non-disabled individuals. In contrast, Pandyan et al.,[70] found a significant difference when comparing the non-affected side to the affected side among patients post stroke suffering from spasticity. Voerman et al.,[72] also reported that MAS was only correlated with RPM at low speed testing ($rho=0.73$, $p<0.05$) whereas two other studies did not find any associations between any of the EMG and RPM parameters or MAS score.[69, 71] However, Pandyan et al.,[71] found that the EMG parameters were significantly different during slow and fast speed stretching ($p>0.1$). Similarly, Voerman et al.,[72] showed that the EMG responses were significantly different between patients post stroke and non-disabled subjects.

The third approach (manual sinusoidal motion) showed consistent evidence of validity. The studies showed that the viscosity component derived in this method was highly correlated with the MAS ($rho= 0.86$, $rho= 0.99$, $p<0.001$)[73, 109] and could distinguish between subjects who suffer from spasticity and non-disabled individuals,[73]

as well as between measurements pre and post treatment with botulinum toxin type A (BTX-A).[112]

2.3.3 RELIABILITY OF THE MEASUREMENT TOOLS

The level of reliability of the spasticity measures presented in this review is found in Table 3. In total, 6 studies reported on the reliability of the Ashworth scales (MAS and MMAS but no report on AS),[44-46, 101, 104, 106] 3 studies reported on the Tardieu scales,[52-54] 5 studies reported on the multi-item scales,[106, 108, 111, 113, 114] 2 studies reported on electrophysiological measures,[90, 105] and 2 studies reported on force/torque measurements (NeuroFlexor and ramp stretches but no report on sinusoidal method).[72, 102] There were no reports on the reliability of the elbow pendulum test in any of the included studies. Further, only one study reported on absolute reliability, which refers to the stability or precision of the measure.[116] Gäverth et al.[102] reported the absolute reliability for the spasticity measure (neural component) of the NeuroFlexor using the coefficient of variation (CV%). The CV% assesses measurement stability across repeated trials by looking at the variability, which reflects the degree of measurement error.[117] The authors found that the CV% was 32% for both test-retest and inter-rater reliabilities.[102]

Table 3. Reliability of spasticity measures

Study	Measure	Muscle group(s)	Test-retest*	Inter-rater
Ashworth Scales				
Ansari et al.[46]	MMAS	Elbow flexors	NA	Kw=0.81
Ansari et al.[45]	MMAS	Knee extensors	K=0.82	K=0.72
Blackburn et al.[101]	MAS	Lower extremity muscles†	Tau-b=0.57	Tau-b=0.06
Gregson et al.[106]	MAS	Elbow flexors	Kw=0.83	Kw=0.84
Gregson et al.[44]	MAS	Wrist flexors	Kw=0.80-0.88	Kw=0.84-0.89
Gregson et al.[44]	MAS	Elbow flexors	Kw=0.77-0.83	Kw=0.77-0.96
Gregson et al.[44]	MAS	Ankle planter flexors	Kw=0.59-0.64	Kw=0.45-0.51
Gregson et al.[44]	MAS	Knee flexors	Kw=0.77-0.94	Kw=0.73-0.79
Kaya, et al.[104]	MMAS	Elbow flexors	NA	Kw=0.89
Kaya, et al.[104]	MAS	Elbow flexors	NA	Kw=0.87
Tardieu Scales				
Ansari et al.[54]	MTS	Ankle planter flexors	ICC=0.68	ICC=0.71
Paulis et al.[52]	TS‡	Elbow flexors	ICC=0.86	ICC=0.66
Paulis et al.[52]	TS^{x}	Elbow flexors	ICC=0.76	ICC=0.84
Singh et al.[53]	MTS	Elbow flexors	ICC=0.85	NA
Singh et al.[53]	MTS	Ankle planter flexors	ICC=0.86	NA

Study	Measure	Muscle group(s)	Test-retest*	Inter-rater
Multi-item Scales				
Barnes et al.[111]	TAS (Q4-Q9)	Elbow flexors	NA	Kw=0.66-0.94
Gregson et al.[106]	TAS (Q4-Q9)	Elbow flexors	Kw=0.59-0.86	Kw=0.79-0.92
Pomeroy et al.[113]	Categorizing Tone	Elbow flexors	NA	K=–0.046-0.56
Pomeroy et al.[113]	VAS	Elbow flexors	NA	ICC=0.56
Pomeroy et al.[113]	Categorizing Tone	Knee extensors	NA	K=–0.25–0.48
Pomeroy et al.[113]	VAS	Knee extensors	NA	ICC=0.45
Takeuchi et al.[108]	APTS	Ankle planter flexors	K=0.72-0.94	K=0.63-0.82
Worley et al.[114]	3-item, hypertonus measure	Elbow flexors	NA	rho= 0.57-0.79
Electrophysiological measures				
Calota et al.[90]	TSRT	Elbow flexors	0.46-0.68 Three raters, 2-7 day interval	0.53-0.60 Three raters
Sorinola et al.[105]	Normalized EMG by MVC	Wrist flexors	0.71 - 0.81 Six speeds 60-360 deg/s	NA
Force/torque measurements				
Gäverth et al.[102]	NeuroFlexor, (neural component)	Wrist flexors	0.90, 0.96 Two raters	0.90, 0.94 Two raters
Voerman et al.[72]	Anglo-Dutch Spasticity Measurement Tool. EMG amplitude and slope (force-angle curve)	Wrist flexors	EMG: 0.65-0.82 Slope: 0.67-0.89 Three speeds	EMG: 0.73-0.79 Slope: 0.96-0.99 Three speeds

MMAS: Modified-Modified Ashworth Scale; **MAS**: Modified Ashworth Scale; **MTS**: Modified Tardieu Scale; **TS**: Tardieu Scale; **TAS**: Tone Assessment Scale questions 4 to 9; **VAS**: Visual Analogue Scale; **APTS**: Ankle planter flexors scale; **TSRT**: Tonic Stretch Reflex Threshold; **EMG**: Electromyography; **MVC**: Maximal voluntary contraction; **Kw**: Weighted Kappa statistic; **K**; Kappa statistic; **Tau-b**: Kendall tau rank correlation coefficient; **ICC**: Intraclass correlation coefficient; **NA**: No data available; **rho**: Spearman's rank correlation coefficient.

* Test-retest reliability and intra-rater reliability were considered as one, as the reviewed studies referred to them interchangeably

† Combined values for Quadriceps femoris, Gastrocnemius and Soleus muscles.

‡ R2-R1 measured using a goniometer

x R2-R1 measured using an inertial sensor

2.3.4 RESPONSIVENESS OF THE MEASUREMENT TOOLS

Only one study evaluated the responsiveness of its spasticity measure. Chen et al.[112] who used the manual sinusoidal method, used the Wilcoxon signed-rank test to determine statistical changes before and after treatment with BTX-A. The authors found a significant reduction in the viscosity component for all participants ($p<0.05$).

2.4 DISCUSSION

The aim of our systematic review was to identify and appraise the literature on clinical measures of spasticity that have been investigated in people after stroke. We identified 15 tools (or instruments) for the measurement spasticity divided into six categories (i) Ashworth scales, (ii) Tardieu scales, (iii) multi-item scales, (iv) electrophysiological scales, (v) pendulum test, and (vi) torque/force measurement. These measures ranged from ordinal scales to biomechanical and neurophysiological tests. Thus, our review provides a comprehensive overview of all those types of spasticity assessments in patients after stroke. Overall, the systematic review indicates that little evidence exists on psychometric properties of clinical spasticity assessment tools in the patients post stroke. None of the reviewed tools demonstrated satisfactory results for all evaluated psychometric properties. For most tools, data on validity, absolute reliability and responsiveness were insufficient. We also found that the majority of the included studies (28/40) assessed the level of spasticity in upper extremity muscles. Few studies addressed spasticity in the lower extremity.

The quality appraisal of the included studies outlined in Table 1 show that the methodological quality was moderate to adequate for the majority of the studies. The item "sample" of all studies was the most limiting factor, due to the absence of adequate sample size calculations for all studies. This may be attributed to the limited reporting and understanding of error of measurement for the spasticity tools that have been used in these studies, as error of measurement is needed to estimate the proper sample size needed for the study.[91, 118] Table 1 also shows that, in general, the quality of studies that have used an instrumented measure had higher scores than studies that have used non-instrumented measures for measuring spasticity.

In the absence of a gold standard for the measurement of spasticity, the MAS is often used as the yardstick to which a new measure is compared. However, as previously mentioned, the validity of the Ashworth scales has been questioned and should probably not be the only comparator when developing a new measure of spasticity. Various studies have reported that ordinal spasticity measures, especially the Ashworth scales, are not objective and lead to grading the level spasticity inaccurately.[10-12, 33] Because the nature of ordinal scales relay on the interpretation of the assessor when measuring spasticity, it is difficult to control the assignment of the assessed spasticity degree into its proper level. In our review we noticed that the studies that aimed to investigate the validity of clinical ordinal scales, used instrumented methods to assess validity, and that studies that aimed to investigate clinical instrumented methods sought to determine their validity by comparing them the MAS. This clearly indicates that there is no universal measure of spasticity. When examining the validity of the ordinal scales in our review (i.e., Ashworth, Tardieu and multi-item scales), the results were inconclusive despite extensive investigation of some of the measures such as the MAS. In regard to the clinical instrumented measures in our review we observed that out of the six instrumented measures of spasticity, one measure (pendulum test) did not investigate the relation between that instrument with any other spasticity scale or EMG,[115] and three measures (TSRT, EMG normalized by MVC and manual sinusoidal motion) only reported the correlation between the those instruments and the MAS.[73, 90, 103, 105, 109, 112] It could be argued that the some of these measures (i.e., TSRT and EMG normalized to MVC) are obtained using EMG and do not require to be validated. Nevertheless, in the studies that we have included, the TSRT was not investigated in relation to other measures (e.g., H-reflex). Further, the approach of using EMG normalized by MVC to obtain a measure of spasticity among patients after stroke may not be accurate, as MVC is not a accurate reflection of the maximal torque generating capacity of the muscle, especially among patients post-stroke.[119, 120]

Despite the abovementioned results, our review show that the spasticity measures, in general, have the ability to discriminate between the affected and non-affected sides or assessing patients who have spasticity and non-disabled individuals. This discriminative ability was seen in all categories of measures except for the multi-item scales, as none of

the included studies have reported on this. However, it appears, as the tools are not as discriminative between different levels of spasticity. Several of the included studies reported that when using instrumented measures (e.g., H-reflex) to differentiate patient groups with different levels of spasticity measured using ordinal scales (i.e., comparing 2 groups with MAS 1 and 2 using H-reflex), there was no significant difference between those groups.[40, 107, 110]

The topic of measuring spasticity has been extensively investigated by many studies, with various systematic reviews published in this topic.[75, 89, 95-97] In 2005 the support program for assembly of database for spasticity measurement (SPASM consortium) published 3 systematic reviews on measures for spasticity and associated phenomena, and concluded that reliability evidence was missing for many of the scales reviewed.[75, 89, 97] Today, this still holds true — we noted a wide range in the level of reliability of the measurement tools and an absence of reliability testing in two of the measures (AS and manual sinusoidal method). Furthermore, only one measure (NeuroFlexor) reported on absolute reliability.[102] To interpret the strength of agreement of findings of the reliability studies, the aforementioned classification by Munro et al.[118] and the following classification by Brennan and Silman,[121] are helpful: kappa<0.21 poor; 0.21–0.40 fair; 0.41–0.60 moderate; 0.61–0.80 good; 0.81–1 very good agreement. The reliability of spasticity measures were low to very high for Ashworth scales and the multi-item scales, moderate to high for the Tardieu scales and electrophysiological measures and moderate to very high for torque/force measurements. There are large variations within the measures and this inconsistencies needs to be properly addressed such as differences between different joints, e.g. ankle and elbow and also to lack of standardization of test procedures.

Reliability was often reported in the form of the intra-class correlation coefficient (ICC). However, there are issues when using the ICC to report on the repeatability of spasticity measures. Most importantly, when the ICC is used for ordinal data, it is assumed that the spacing between each level of spasticity is equal (e.g., the level of spasticity between MAS 0 and 1 is equal to the level between MAS 1 and 1+), which is not correct.[117, 121, 122] The proper statistical method to use in this case is the Kappa statistic.[117, 121] Another issue with the ICC is when applied to data from a group of

subjects who have a wide range of the level of spasticity, reliability will appear to be higher than when applied to a group who demonstrate a narrow range of the level of spasticity.[122] Therefore, having a high ICC is not necessarily indicative of the high reliability of the measure but may be due to the sample heterogeneity. Another finding in our review is that only one of the measures reported data on responsiveness but did not use global ratings of change,[112] this is a deficiency that could be addressed through controlled trials to evaluate specific anti-spasticity interventions.

2.5 LIMITATIONS OF THIS REVIEW

One limitation of this review is the number of included studies. However, for the purpose of our review, we included studies of clinical measures as indicated by the authors of the individual articles. Although this limited the number of included studies, we found it appropriate as we set out to identify measures that could be used in clinical practice or clinical research. Another limitation of this review was that some of the measures being reviewed in this article are used to validate other measures as well (e.g., MAS is reviewed in article and used by some studies to validate their measure). However, this is expected with the issue of lacking a clinical "gold standard" for measurement of spasticity. In addition, the small sample sizes of some of the studies and the wide range in the various aspects of quality assessment of the studies limit interpretation of the results. Finally, this review was limited to studies in which muscle spasticity was measured during a relaxed state. Such condition does not necessarily reflect the level of hyper-excitability of the stretch reflex arc during functional, multi-joint movements such as walking.

2.6 RECOMMENDATIONS AND CONCLUSION

In this review we have found a number of clinical measures for assessing spasticity among patients post stroke, with different psychometric characteristics. Yet, the evidence in support of the validity of the reviewed scales in measuring spasticity was not conclusive. Further, there is a lack in the evidence of absolute reliability and responsiveness of spasticity measures. Given the importance of spasticity assessment for accurate evaluation of the functional capacity and appropriate intervention of patients after stroke, studies with larger sample sizes are required to validate these tools in this

specific population. Future research focusing on investigating the psychometric properties of clinical spasticity measures, especially validity, absolute reliability and responsiveness, is warranted. The present systematic review did not find sufficient psychometric evidence to recommend one tool over the others.

2.7 DECLARATION OF CONFLICTS OF INTEREST

One of the co-authors (JG) of this systematic review is the first author and co-author of 2 articles included in this review.[74, 102] These two articles were not reviewed or rated by JG.

CHAPTER 3

METHODS

3.1 STUDY DESIGN

This study used a single cohort design to investigate the BioTone system as part of a larger study introduced in Section 1.4.

3.2 OBJECTIVES

The overall purpose of this study was to assess, in a preliminary way, the concurrent and construct validity and test-retest reliability of the BioTone technology as a clinical measurement of spasticity among patients following stroke.

3.2.1 PRIMARY AIMS

1. Assess and compare kinematic and EMG data obtained using the BioTone and develop a framework for extracting measures of spasticity
2. Investigate the concurrent validity of the spasticity measures of the elbow and knee muscles obtained using the BioTone system
3. Examine the construct validity of the BioTone system's spasticity measures for elbow muscles
4. Assess and compare the test-retest reliability of the BioTone and clinical spasticity measures

3.2.2 SECONDARY AIMS

1. Examine the relation between active range of motion (AROM) of the elbow and BioTone and clinical spasticity measures of the elbow muscles.
2. Examine the relation between elbow flexor and extensor strength with BioTone and clinical spasticity measures.

3.3 HYPOTHESES

3.3.1 PRIMARY HYPOTHESES

H1.1. There is no difference between spastic onset time determined from kinematic data (Tk) and onset time determined from EMG signal (Te) (or $\delta T = 0$).

H1.2. Intensity kinematic variables (ΔV and ΔA) will correlate positively with corresponding stretched muscle EMG intensity (ΔStr), but will not correlate with non-stretched muscle EMG intensity ($\Delta NStr$).

H1.3. Density kinematic variables (εV and εA) will correlate positively with corresponding stretched muscle EMG intensity (εStr), but will not correlate with non-stretched muscle EMG intensity (εNStr).

H1.4. BioTone spasticity measures of elbow and knee muscles will correlate positively with the MAS and TS

H1.5. The metrics obtained from the elbow motion theoretical kinematic model (MAX-V, MAX-A, V and A) will correlate positively with the corresponding metrics from the kinematic model of the non-paretic elbow motion (MAX-V_N, MAX-A_N, V_N and A_N).

H1.6. The BioTone spasticity measures of elbow and knee muscles and clinical measures will show good test-retest reliability

3.3.2 SECONDARY HYPOTHESES

H2.1. The AROM deficit (ΔAROM) of elbow flexion will correlate positively with BioTone spasticity measures of elbow and with MAS and TS for elbow muscles.

H2.2. The elbow flexors (Δ Flexors) and elbow extensors (Δ Extensors) muscles strength deficits will correlate positively with BioTone spasticity measures of elbow and with MAS and TS for elbow muscles.

3.4 PARTICIPANTS

3.4.1 INCLUSION CRITERIA

Male or female adults who are aged 18 years or over, diagnosed with first symptomatic unilateral stroke (either ischemic or hemorrhagic). Subjects must be medically stable (as deemed by their physiatrist or neurologist), capable of providing informed consent and demonstrate some degree of spasticity in muscles crossing the elbow and/or knee joints on the hemiparetic side as measured by the MAS (see Appendix B).

3.4.2 EXCLUSION CRITERIA

Potential subjects were excluded if they were unable to sit comfortably in the wheelchair used for pendulum test or if they had excessive subcutaneous fat tissue, which impedes measurement of surface EMG signals. This exclusion criterion was

confirmed by checking the quality of the EMG signal prior to assessment. Additional exclusion criteria were: arthopathy or contracture that would prohibit objective measurement of spasticity, open skin lesions on the sites of EMG electrode application, inability to comprehend instructions in English or use of any anti-spasticity medications within the last 3-month period (e.g., Baclofen, Botulinum toxin type a [BTX-A]).

3.4.3 RECRUITMENT AND SETTING

This study was carried out at the facilities available in the NSRC. The equipment and facilities required to administer the study were available on-site.

Recruitment was facilitated via the clinical staff at the Physiotherapy Research Lab at the NSRC. Staff reviewed the recruitment process and study design and then identified potential subjects who met the inclusion and exclusion criteria of the study. Each potential subject was approached by a clinician known to them, to ask their permission to be approached by the research staff. If the potential participant agreed to be approached by research staff, the principal investigator or research assistant associated with the study, who is not involved in the patient's care, introduced the potential candidate to the study, and, if the candidate continued to express interest in participating, explained the study in detail, answered any questions and obtained written informed consent.

3.4.4 SCREENING

After obtaining the written informed consent, the subject was given an identification number (ID) and then the patient was screened to confirm the suitability of the candidate for the study. The following information/measures were collected:

1. Descriptive information obtained from the subject's hospital record consisted of date of birth, sex, pertinent medical and medication history, pre-morbid handedness, and stroke history: date of onset, type (ischemic or hemorrhagic), and location (i.e., vascular distribution and hemisphere)
2. Specific information included history of BTX-A injections (date and sites of most recent injections), spasticity medications, relevant surgeries, and any changes in recent activity or therapy

3. The study therapist determined presence of spasticity in muscles crossing the knee and/or elbow using passive range of motion of flexion and extension of each joint. The study therapist moved the joints at slow speed and high speed to determine if there is a velocity-dependent increase in tonic stretch reflexes as compared to the person's other side (non-affected side).[6, 32]
4. Weight in kilograms (kg) and height in centimeters (cm) were measured
5. Confirmation that a comfortable position in the pendulum test wheelchair was possible for the pendulum test

3.4.5 SAMPLE SIZE

For my study a sample of convenience of 15 consecutive patients who met the eligibility criteria was recruited.

3.5 PROCEDURE

After the participant provided informed consent, a two-hour appointment was scheduled for data collection. Data collection procedure was done twice for 6 participants in order to conduct a preliminary assessment of the test-retest reliability of the BioTone system. The assessment protocol is outlined below.

After the patient arrived for the session, the investigator reviewed the previously gathered information from the participant (e.g., consent, treatment changes) to confirm that there were no changes following the recruitment of the subject to the study. Then spasticity assessment was conducted on all four limbs using the MAS (see Appendix B),[32] and TS (Appendix C).[49] Subsequently, The aforementioned BioTone assessments that involves: (i) stretch tests for the elbow musculature of the affected side, (ii) elbow active ROM for the affected side, and (iii) isometric strength test for the elbow flexors and extensors of the affected side, were all conducted. Then, the pendulum test was conducted with the participant sitting in the pendulum test wheelchair with the electrogoniometer on the knee joint. The patient was asked to completely relax while the study therapist passively lifted the leg of the participant to full knee extension and confirmed that the participant was adequately relaxed to permit to the leg to swing into flexion freely. The investigator then released the leg from full extension, allowing the leg to swing freely (in a pendulum motion) until coming to rest. Three trials of the pendulum

test were recorded. Following the assessment of the paretic side, the entire procedure was repeated for the non-paretic side.

3.6 DATA PROCESSING

3.6.1 ELBOW MUSCULATURE DATA

Paretic Side

For the data obtained from the elbow muscles of the paretic side, the approach mentioned above that is used for the BioTone study, was utilized to define measures for elbow muscles spasticity.

Variables Used for Analysis

As mentioned above, two types of measures were used to represent elbow muscles spasticity: (i) Muscles reflex intensity and (ii) Muscles reflex density. Additionally, variables used to validate the theoretical model were obtained from the theoretical motion curve.

1- Muscle reflex intensity: From the kinematic data the variables used were peak magnitudes of departures from theoretical profile for velocity (Δ**V**) and acceleration (Δ**A**) (see Figure 7). From the EMG data, two discrete variables were selected which were peak, EMG at time Te for stretched muscle (Δ**Str**) and for the opposite or non-stretched muscle (Δ**NStr**)(see Figure 8).

2- Muscle reflex Density: For kinematic data, two variables were determined by quantifying the root mean square departure for angular velocity (**εV**) and acceleration (**εA**) between T1 and T2 (shaded regions in Figure 7). EMG density measures were the area under the curve between T1 and T2 for stretched (**εStr**) and non-stretched (**εNStr**) muscles (shaded region in Figure 8).

3- Variables used to validate theoretical model: Four kinematic variables, 2 discrete and 2 density variables, were used to validate the theoretical model. Discrete variables were: (**MAX-V**) which is the maximum velocity of the theoretical model and (**MAX-A**) which is the absolute maximum acceleration of the theoretical model (see Figure 7). Density variables were: (**V**) which is the root mean square of

the theoretical angular velocity and (**A**) which is the root mean square of the theoretical acceleration (hatched regions in Figure 7).

Non-Paretic Side

The main objective for assessing the non-paretic side was to further validate the BioTone system. Therefore, extracting variables from the non-paretic side to compare them with the variables obtained from the paretic side was necessary specifically for validating the theoretical model that was developed based on the kinematic data of the paretic side. The main assumption of the theoretical model is that this is the motion that would occur if there were no spastic disturbance in the affected side. Therefore, the elbow motion in the non-paretic side (i.e., non-spastic side) should significantly correlate with the theoretical model of the paretic side.

Variables Used for Analysis

Kinematic data obtained from the non-paretic UE was first low-pass filtered at 6Hz (zero-lag, 4th order Butterworth). Then the kinematic variables that correspond to the variables obtained from the theoretical model were obtained from the elbow motion curve in the non-paretic side. Using the subscript 'N' to denote the non-paretic side, discrete variables were: (MAX-V_N) which is the peak magnitude for velocity and (MAX-A_N) which is the peak magnitude for acceleration (Figure 10). The density variables were: (V_N) which is the area under the curve for velocity and (A_N) which is the area under the curve for acceleration (shaded regions in Figure 10).

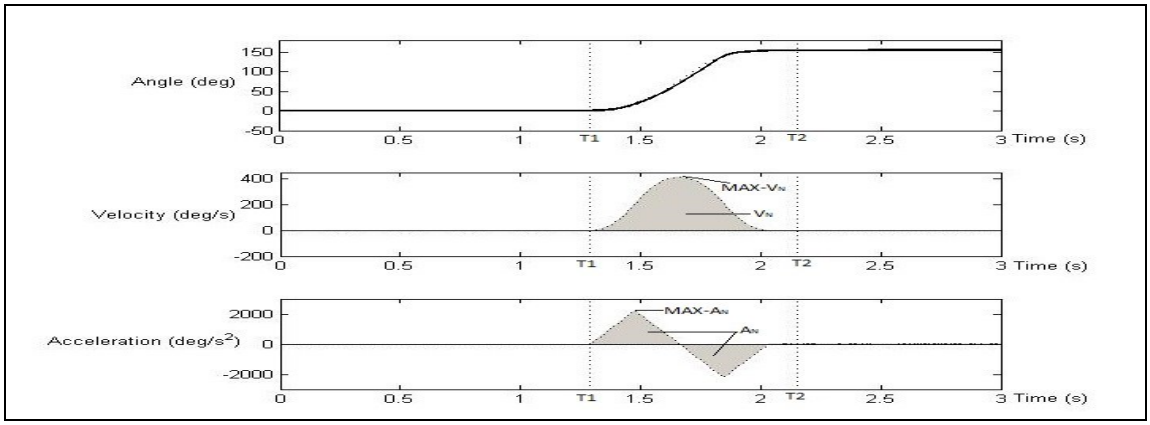

Figure 10: Variables used from the non-paretic elbow motion curve. Elbow flexion trial for participant 10 performed on the non-paretic side (MAS=0).

Strength and ROM Data

AROM Data

AROM for elbow flexion was assessed for both the paretic side and the non-paretic side. The difference in elbow flexion AROM between the non-paretic side and paretic side (**ΔAROM**) was used as a variable for the statistical analysis.

Elbow Flexion and Extension Dynamometry Data

Similar to the approach used for AROM data, "strength deficits" were obtained for elbow flexors and extensors muscles by subtracting elbow muscle strength scores of the paretic side from the scores of the non-paretic side to obtain strength deficits for elbow flexors (Δ Flexors) and elbow extensors (Δ Extensors).

3.6.2 PENDULUM TEST DATA

For the knee joint data (i.e., pendulum test data), the goniometric data was first low-pass filtered at 6Hz (zero-lag, 4th order Butterworth). The **RI** was calculated by determining the ratio of the difference between the angle at end of initial movement into flexion and angle at the start of test (i.e., magnitude of the first drop) and the difference between the angle at end of test and angle at start of test (i.e., magnitude of the initial angle) ($RI=A_1/A_0$, Figure 11). Furthermore, the first angle of pendulum motion was considered the onset angle of the spastic reaction.

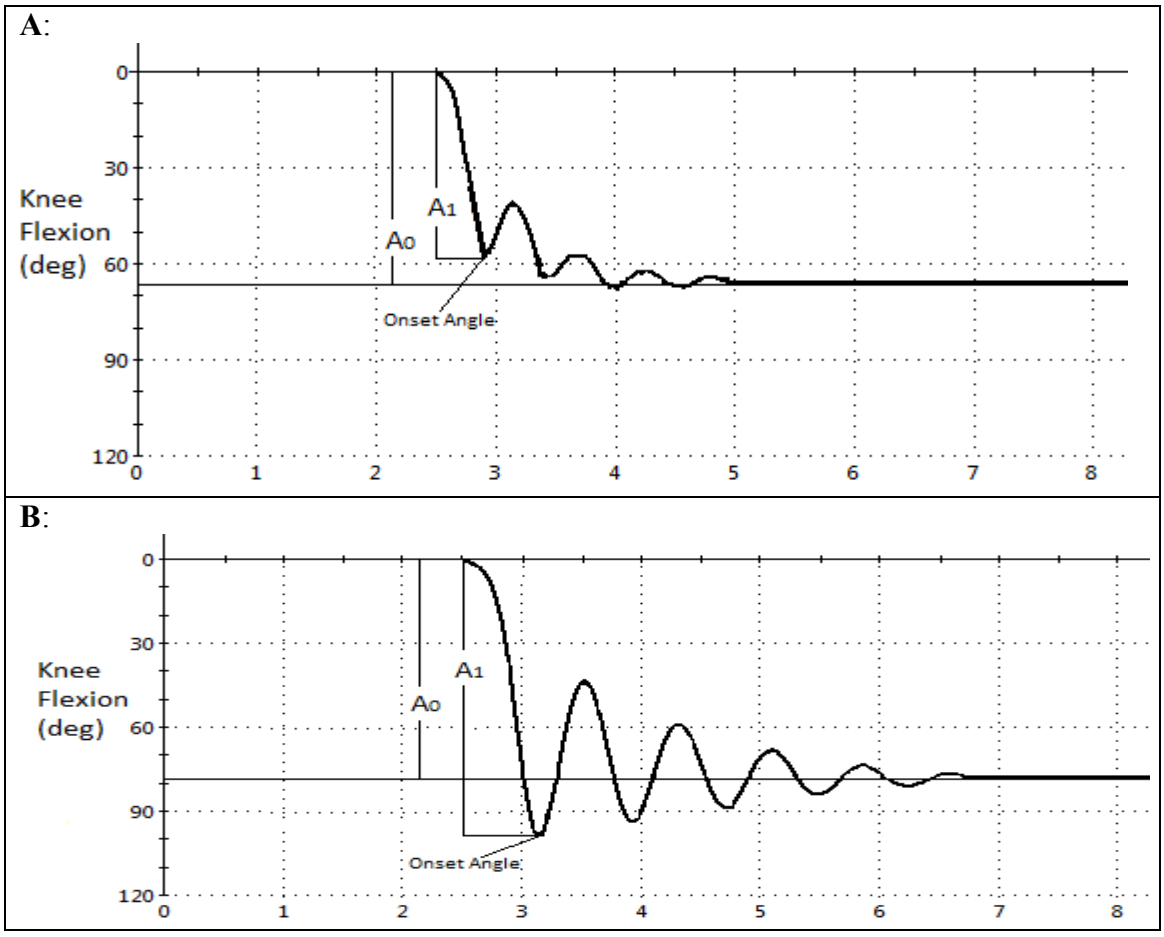

Figure 11. The pendulum test. A: Pendulum test for participant 7 with knee extensors MAS=2. B: Pendulum test for the same patient on the non-paretic side.

3.7 DATA ANALYSIS

Analysis of the data involved visually inspecting the data to identify trends and conducting descriptive statistics to further examine the data. Inferential analysis were performed to test the aforementioned hypotheses, with $\alpha = 0.05$ for statistical significance. SPSS version 20 (IBM Corp.) was used for all statistical analyses. Because the MAS (0-4) include a category of 1+, the MAS scores were reassigned to a scale of (0-5) for analysis.

1. For H1.1, one-sample t-test was used to study if there was no difference between onset times ($\delta T = 0$).

2. For H1.2 and H1.3, a Spearman's rank correlation coefficient (Spearman's rho) was used to study the correlation between the kinematic and EMG variables. A Spearman's rho was used because the EMG signals are non-normally distributed.
3. For H1.4, Spearman's rho was used to assess the correlation between spasticity measures of elbow and knee muscles with the MAS and TS. Spearman's rho was used because MAS and TS are ordinal data. A Pearson product-moment correlation coefficient (Pearson's r) was used to determine the correlation between the onset angle from the BioTone and the angle of catch from the TS.
4. For H1.5, Pearson's r was used to conduct a correlation study between measures obtained from paretic side and the measures obtained from the non-paretic side.
5. For H1.6, an intraclass correlation coefficient (ICC) was calculated to determine the test-retest reliability of the BioTone spasticity measures of elbow and knee muscles and the clinical measures.
6. For H2.1 and H2.2, Spearman's rho was used to determine the correlation between AROM and strength deficits with BioTone measures and clinical measures.

CHAPTER 4

RESULTS

4.1 PARTICIPANTS

In our study, we recruited 15 patients, 6 of whom attended 2 evaluation sessions while the rest of the participants attended only one evaluation session. The evaluation session time required 1.5-2.5 hours to complete (mean=2.13 hours). Four females and eleven males were recruited, ranging in age from 51-93 years (mean=65.4 years, standard deviation [SD]=11.08 years). Characteristics of the participants are presented in Table 4. Participants who agreed to participate in the test-retest part of the study (i.e., attended two evaluation sessions) are subject 1 and subjects 11-15.

Table 4. Characteristics of participants

ID	Sex	Age (years)	Time since Stroke (years)	BMI (kg/m^2)	Hand dominance	Hemiparetic Side
01	F	76	5	22.85	R	R
02	F	56	18	25.97	R	R
03	M	59	1	29.06	R	R
04	M	63	6	25.92	R	R
05	M	66	21	23.01	L	R
06	M	59	2	26.76	R	R
07	F	64	16	28.89	R	R
08	M	93	0.16*	24.47	R	R
09	F	52	2	21.85	L	R
10	M	68	0.08 †	22.98	R	L
11	M	58	0.16 *	29.90	R	R
12	M	51	0.25 ‡	24.88	R	L
13	M	66	4	28.34	R	R
14	M	71	9	29.34	R	L
15	M	79	2	29.39	R	R
Total	4 F 11 M	65.4 (SD=11.08)	5.73 (SD=7.07)	26.24 (SD=2.79)	2 L 13 R	3 L 12 R

BMI= Body Mass Index, **F**= Female, **M**=Male, **R**=Right, **L**=Left,

* 2 months, † 1 month, ‡ 3 months

4.2 SPASTICITY MEASURES

4.2.1 CLINICAL SPASTICITY MEASURES

The MAS and TS were used for the clinical assessment of spasticity. Table 5 shows the level of spasticity in the elbow flexors and extensors and in the knee extensors as measured by the MAS and TS. The Mann-Whitney U test was used to compare the level of spasticity in the elbow flexors versus the elbow extensors muscles. Results

showed that there was no significant difference between the two muscle groups as measured by the MAS (p=0.344) and the TS (p=0.203).

Table 5. Participant's scores on clinical spasticity measures

ID	MAS Elbow Flexors	MAS Elbow Extensors	MAS Knee Extensors	TS score (angle)* Elbow Flexors	TS score (angle) Elbow Extensors	TS score (angle) Knee Extensors
01	1	1+	0	2 (10˚)	2 (80˚)	0 (†)
02	1+	0	1	2 (50˚)	2 (90˚)	2 (100˚)
03	1	1+	1	2 (45˚)	2 (110˚)	1 (†)
04	1	1+	0	1 (†)	1 (†)	0 (†)
05	1+	1+	0	2 (70˚)	2 (80˚)	0 (†)
06	1+	1+	1	2 (40˚)	2 (70˚)	2 (80˚)
07	1	2	1	2 (50˚)	2 (55˚)	2 (40˚)
08	1	1	Not tested	2 (70˚)	1 (†)	Not tested
09	1	1+	1	2 (50˚)	2 (70˚)	2 (80˚)
10	1+	2	3	2 (30˚)	2 (80˚)	4 (25˚)
11	3	2	1	2 (30˚)	2 (90˚)	1 (†)
12	1	1	1	2 (60˚)	2 (80˚)	1 (†)
13	1	1	1+	1 (†)	1 (†)	2 (30˚)
14	1+	1	1	2 (70˚)	1 (†)	1 (†)
15	1	1	1	2 (25˚)	1 (†)	1 (†)

MAS: Modified Ashworth Scale; **TS**: Tardieu Scale

* Angle of catch, † No onset angle (catch) was detected for TS assessment

Despite that there was no significant difference in spasticity, as measured by MAS and TS, between the elbow flexors and extensors, visual inspection of the data revealed that there was no variability in the MAS for elbow flexors, as most MAS score were '1' and '1+' except for one subject (ID=11) who scored '3' (Figure 12-A). Moreover, the TS scores for elbow flexors and extensors show a trend of increased tone in elbow flexors. This is illustrated in Figure 12-B.

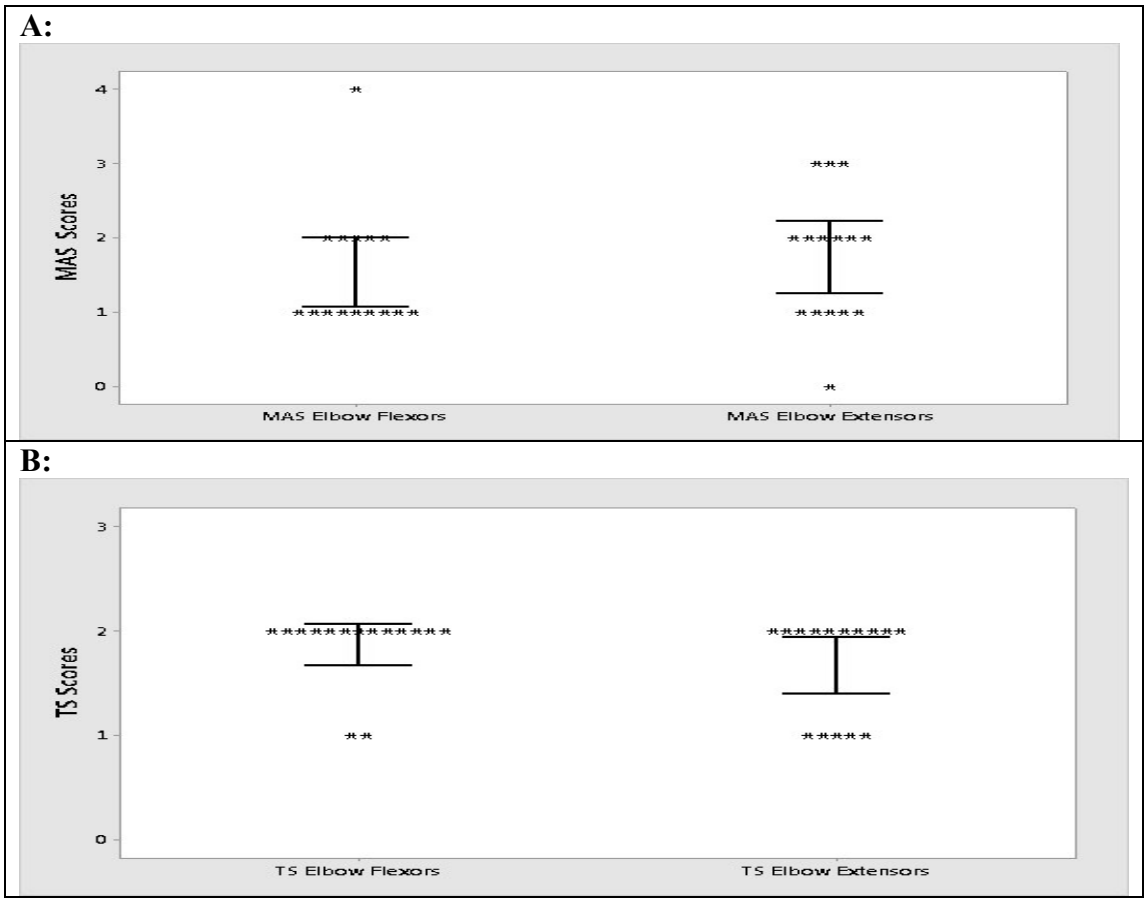

Figure 12: Interval plots of clinical spasticity measures for elbow flexors and elbow extensors. A: MAS scores for elbow flexors and extensors and their interval plot of the 95% confidence interval of the mean. B: TS scores for elbow flexors and extensors and their interval plot of the 95% confidence interval of the mean.

4.2.2 BIOTONE SPASTICITY MEASURES

Prediction of Spastic Onset Time

After determining the spastic onset times from the kinematic data and from the EMG data, the difference in time between the two onsets was determined (δT). Table 6 shows the estimated onset time for elbow flexors and extensors.

Table 6. Spastic onset times

ID	Elbow Flexors			Elbow Extensors		
	Te	Tk	δT*	Te	Tk	δT
01	-0.25	0.27	-0.52	-0.39	-0.31	-0.08
02	0.26	0.29	-0.03	0.41	0.34	0.07
03	0.38	0.33	0.05	-0.44	-0.28	-0.16
04	0.27	0.14	0.13	0.68	0.34	0.34
05	0.31	0.26	0.05	0.36	0.19	0.17
06	0.26	0.28	-0.02	-0.38	-0.22	-0.16
07	0.28	0.34	-0.06	-0.25	-0.17	-0.08
08	0.30	0.22	0.08	0.44	0.32	0.12
09	-0.64	0.29	-0.93	0.21	0.16	0.05
10	0.46	-0.16	0.62	0.26	0.18	0.08
11	0.22	0.22	0.00	0.23	0.26	-0.03
12	-1.19	0.30	-1.49	-0.18	-0.13	-0.05
13	0.18	0.16	0.02	0.31	0.13	0.18
14	0.22	0.23	-0.01	-0.21	-0.09	-0.12
15	0.20	0.35	-0.15	0.31	0.12	0.19

Te: Spastic onset time determined from EMG data; **Tk**: Spastic onset time determined from kinematic data; **δT**: Difference in onset times between EMG and Kinematic data sets.

* δT= Te-Tk

Quantifying BioTone Spasticity Measures

For the elbow musculature data, four discrete and four density variables were identified that potentially could be used as indicators of spasticity. These indicators are presented in Tables 7 and 8 for elbow flexors and extensors, respectively. In addition, Table 9 shows the RI for knee extensors muscles.

Table 7. Indicators of elbow flexors spasticity

ID	MAS	TS-Score	TS-Angle	ΔV	ΔA	ΔStr	ΔNStr	Onset-Angle	εV	εA	εStr	εNStr
01	1	2	10	112.05	2411.46	0.0145	-0.0014	67.76	36.22	620.57	0.0078	0.0052
02	1+	2	50	229.66	3367.06	0.0606	0.0112	71.04	62.83	830.99	0.0166	0.0083
03	1	2	45	139.92	2007.54	0.1095	0.0107	75.82	41.07	562.92	0.0269	0.0141
04	1	1	*	164.85	3105.49	0.2129	-0.0072	116.01	45.67	799.55	0.0584	0.062
05	1+	2	70	283.87	3290.82	0.1	0.007	67.6	86.85	1022.09	0.0236	0.0054
06	1+	2	40	264.68	4135.37	0.0135	-0.0017	57.37	60.71	1092.97	0.0054	0.0039
07	1	2	50	192.36	2663.04	0.0746	0.0037	44.09	45.07	809.12	0.0243	0.0042
08	1	2	70	207.06	2696.09	0.1442	0.0049	103.5	80.86	976.43	0.0457	0.0084
09	1	2	50	194.18	2584.47	0.0177	0.0008	59	54.94	863.13	0.0166	0.0143
10	1+	2	30	76.27	1161.85	0.1033	-0.0061	63.04	32.71	391.47	0.0272	0.0132
11	3	2	30	214.21	3387.6	0.0191	0.0045	77.61	61.3	795.19	0.0084	0.0081
12	1	2	60	155.41	4000.98	0.0018	0.0005	19.65	25.68	637.55	0.0055	0.0015
13	1	1	*	230.2	3530.53	0.0988	0.0226	90.85	60.72	1052.37	0.0148	0.006
14	1+	2	70	489.4	9059.51	0.0623	0.021	52.75	85.91	1632.61	0.0102	0.0149
15	1	2	25	194.67	5207.96	0.0239	0.0299	41.35	41.39	909.55	0.0056	0.0048

MAS= modified Ashworth Scale for elbow flexors; **TS-Score**= Tardieu scale score for elbow flexors; **TS-Angle**= Tardieu scale angle of catch; **ΔV**= peak magnitude of departure in velocity from reference profile (theoretical curve); **ΔA**= peak magnitude of departure in acceleration from reference profile; **ΔStr**= EMG intensity at onset time (Te) for stretched muscle (elbow flexors); **ΔNStr**= EMG intensity at onset time (Te) for non-stretched muscle (i.e., shortened muscle – elbow extensors); **Onset-Angle**= spasticity onset angle determined by BioTone; **εV**= root mean square departure from reference profile for angular velocity; **εA=** root mean square departure from reference profile for acceleration; **εStr**= EMG departures from "reference" (area under the curve between T1-T2) for stretched muscle (elbow flexors); **εNStr**= EMG departure from "reference" (area under the curve between T1-T2) for non-stretched muscle (elbow extensors)

* No onset angle (catch) was detected for TS assessment.

Table 8. Indicators of elbow extensors spasticity

ID	MAS	TS-Score	TS-Angle	ΔV	ΔA	ΔStr	ΔNStr	Onset-Angle	εV	εA	εStr	εNStr
01	1+	1+	80	287.51	3325.79	0.0512	0.0195	63.94	89.78	993.56	0.0303	0.0198
02	0	1+	90	155.97	2145.42	0.0248	0.0193	39.12	68.74	653.04	0.0169	0.0184
03	1+	1+	110	152.03	1878.74	0.0702	-0.0076	64.31	58.65	659.47	0.0365	0.0141
04	1+	1	*	147.83	1750.75	0.2267	0.01	68.89	52.5	643.54	0.0879	0.0844
05	1+	1+	80	264.94	3334.27	0.1751	-0.0035	29.19	78.23	1101.34	0.0307	0.0033
06	1+	1+	70	261.89	4600.77	0.0877	0.0047	42.09	89.13	1079.39	0.0417	0.0057
07	2	1+	55	195.6	2551.83	0.0218	0.0126	40.27	51.24	773.2	0.0069	0.0137
08	1	1	*	168.16	2421.36	0.0258	0.0757	62.51	53.41	697.98	0.01	0.0393
09	1+	1+	70	305.62	4830.56	0.0928	0.005	40.36	83.97	1265.14	0.0311	0.0118
10	2	1+	80	174.32	2005.03	0.2844	0.0189	25.41	45.95	683.26	0.1555	0.0333
11	2	1+	90	198.94	3475.85	0.0446	0.0014	72.64	36.33	822.78	0.0094	0.0033
12	1	1+	80	321.41	5056.36	0.0329	0.0007	35.96	68.27	1376.74	0.0056	0.0057
13	1	1	*	106.7	1452.36	0.0113	0.0911	18.36	24.79	463.83	0.0028	0.0177
14	1	1	*	332.41	8048.78	0.1509	0.0097	28.56	71.31	1491.93	0.0173	0.0041
15	1	1	*	225.53	4357.8	0.055	0.0153	33.68	66.33	1017.6	0.0076	0.0077

MAS= modified Ashworth Scale for elbow extensors; **TS-Score**= Tardieu scale score for elbow extensors; **TS-Angle**= Tardieu scale angle of catch; **ΔV**= peak magnitude of departure in velocity from reference profile (theoretical curve); **ΔA**= peak magnitude of departure in acceleration from reference profile; **ΔStr**= EMG intensity at onset time (Te) for stretched muscle (elbow extensors); **ΔNStr**= EMG intensity at onset time (Te) for non-stretched muscle (i.e., shortened muscle – elbow flexors); **Onset-Angle**= spasticity onset angle determined by BioTone; **εV**= root mean square departure from reference profile for angular velocity; **εA=** root mean square departure from reference profile for acceleration; **εStr**= EMG departures from "reference" (area under the curve between T1-T2) for stretched muscle (elbow extensors); **εNStr**= EMG departure from "reference" (area under the curve between T1-T2) for non-stretched muscle (elbow flexors)

* No onset angle (catch) was detected for TS assessment.

Table 9. Indicators of knee extensors spasticity					
ID*	MAS	TS-score	TS-Angle	Relaxation index	Onset angle- pendulum test
1	0	0	†	1.36	92.63
2	1	2	100	1.00	69.10
3	1	1	†	1.18	77.50
4	0	0	†	1.34	65.57
5	0	0	†	1.44	80.13
6	1	2	80	1.31	101.07
7	1	2	40	0.89	59.73
9	1	2	80	1.23	95.57
10	3	4	25	1.16	46.13
11	1	1	†	1.39	70.80
12	1	1	†	1.29	76.60
13	1+	2	30	1.25	63.10
14	1	1	†	0.98	70.33
15	1	1	†	1.35	91.17

MAS: Modified Ashworth Scale; **TS**: Tardieu Scale

* Note that only 14 subjects underwent the pendulum test procedure (subject ID# 8 was not included)
† No onset angle (catch) was detected for TS assessment

4.3 COMPARING KINEMATIC AND EMG DATA SETS

4.3.1 DIFFERENCE BETWEEN SPASTIC ONSET TIMES

For hypothesis H1.1 "*there is no difference between spastic onset time determined from kinematic data (Tk) and onset time determined from EMG signal (Te) (or $\delta T = 0$)*", the null hypothesis was accepted as the results of the one-sample t-test showed that there was no significant difference between onset times for elbow flexors ($p=0.257$) and for elbow extensors ($p=0.361$).

4.3.2 CORRELATION BETWEEN KINEMATIC AND EMG DATA SETS

For hypotheses H1.2 "*intensity kinematic variables (ΔV and ΔA) will correlate positively with corresponding stretched muscle EMG intensity (ΔStr), but will not correlate with non-stretched muscle EMG intensity ($\Delta NStr$)*" and H1.3 "*density kinematic variables (εV and εA) will correlate positively with corresponding stretched muscle EMG intensity (εStr), but will not correlate with non-stretched muscle EMG intensity ($\varepsilon NStr$)*", the null hypotheses were not accepted. None of the hypothesized significant correlations were seen in the kinematic and EMG intensity and density variables for both elbow flexion and extension trials. Only two significant correlations were found and they were not anticipated to be significant, which were $\Delta NStr$ with ΔV for elbow flexors and $\varepsilon NStr$ with εA for elbow extensors (Table 10).

Table 10. Correlation between kinematic variables and EMG metrics

Kinematic Variables		Muscle EMG Metrics			
		Elbow Flexors		Elbow Extensors	
Intensity variables		ΔStr	ΔNStr	ΔStr	ΔNStr
ΔV	rho	-0.075	**0.525**	0.257	-0.371
	p	0.395	**0.022**	0.177	0.086
ΔA	rho	-0.393	0.436	0.154	-0.421
	p	0.074	0.052	0.292	0.059
Density variables		εStr	εNStr	εStr	εNStr
εV	rho	0.122	0.261	0.307	-0.252
	p	0.333	0.174	0.133	0.182
εA	rho	-0.241	-0.052	-0.054	**-0.714**
	p	0.193	0.425	0.425	**0.001**

ΔV= peak magnitude of departure in velocity from reference profile; **ΔA**= peak magnitude of departure in acceleration from reference profile; **ΔStr**= EMG intensity at onset time (Te) for stretched muscle; **ΔNStr**= EMG intensity at onset time (Te) for non-stretched muscle; **εV**= root mean square departure from reference profile for angular velocity; **εA=** root mean square departure from reference profile for acceleration; **εStr**= EMG departures from "reference" (area under the curve between T1-T2) for stretched muscle; **εNStr**= EMG departure from "reference" (area under the curve between T1-T2) for non-stretched muscle; **rho**=Spearman's rank correlation coefficient; **p**= p-value.

Significant findings (i.e., $p<0.05$) are in bold type

4.4 CONCURRENT VALIDITY OF BIOTONE MEASURES

The null hypothesis was partially accepted for hypotheses H1.4 "*BioTone spasticity measures of elbow and knee muscles will correlate positively with the MAS and TS*". The results showed that for elbow flexors, there was a significant correlation between MAS of elbow flexors and ΔV and εV (rho=0.49, p=0.03; rho=0.47, p=0.04, respectively). No significant correlation was found between TS of elbow flexors and BioTone measures. For elbow extensors, there was no correlation between MAS of elbow extensors and BioTone variables. Significant correlation was found between TS of elbow extensors and ΔNStr (rho=0.46, p=0.04). For knee extensors, the RI was significantly correlated with MAS (rho= – 0.54, p=0.02) and TS (rho= – 0.65, p=0.006). The results of the correlation analysis are presented in Table 11 for elbow flexors and elbow extensors and in Table 12 for knee extensors.

Table 11. Correlation between BioTone variables and spasticity clinical measures for elbow muscles

BioTone Variables		Elbow Flexors		Elbow Extensors	
		MAS	TS-Score	MAS	TS-Score
ΔV	rho	**0.487**	-0.45	-0.006	0.262
	p	**0.033**	0.436	0.492	0.173
ΔA	rho	0.256	-0.091	-0.100	0.164
	p	0.179	0.374	0.361	0.280
ΔStr	rho	-0.124	-0.409	0.351	0.033
	p	0.330	0.065	0.100	0.454
ΔNStr	rho	0.000	0.045	-0.315	**0.458**
	p	0.500	0.436	0.126	**0.043**
εV	rho	**0.470**	-0.045	-0.215	0.295
	p	**0.038**	0.436	0.221	0.143
εA	rho	0.165	-0.136	-0.032	0.196
	p	0.278	0.314	0.455	0.241
εStr	rho	-0.167	-0.273	0.410	0.229
	p	0.276	0.163	0.065	0.206
εNStr	rho	0.058	-0.272	-0.070	-0.262
	p	0.419	0.163	0.402	0.172
Onset Angle		TS-Angle		TS-Angle	
	r	-0.382		0.177	
	p	0.080		0.264	

MAS: Modified Ashworth Scale; **TS**: Tardieu Scale; **ΔV**= peak magnitude of departure in velocity from reference profile; **ΔA**= peak magnitude of departure in acceleration from reference profile; **ΔStr**= EMG intensity at onset time (Te) for stretched muscle; **ΔNStr**= EMG intensity at onset time (Te) for non-stretched muscle; **εV**= root mean square departure from reference profile for angular velocity; **εA=** root mean square departure from reference profile for acceleration; **εStr**= EMG departures from "reference" (area under the curve between T1-T2) for stretched muscle; **εNStr**= EMG departure from "reference" (area under the curve between T1-T2) for non-stretched muscle; **rho**=Spearman's rank correlation coefficient; **r**= Pearson's product-moment correlation coefficient; **p**= p-value.

Significant findings (i.e., $p<0.05$) are in bold type

Table 12. Correlation between BioTone variables and spasticity clinical measures for knee extensors

BioTone Variables		MAS	TS-Score
RI	rho	**-0.539**	**-0.645**
	p	**0.023**	**0.006**
Onset-Angle		TS-Angle	
	r	0.168	
	p	0.283	

MAS: Modified Ashworth Scale; **TS**: Tardieu Scale; **RI**= Relaxation index; **rho**=Spearman's rank correlation coefficient; **r**= Pearson's product-moment correlation coefficient; **p**= p-value

Significant findings (i.e., $p<0.05$) are in bold type

4.5 CONSTRUCT VALIDITY OF BIOTONE MEASURES

The null hypothesis for H1.5 was partially accepted "*the metrics obtained from the elbow motion theoretical kinematic model (MAX-V, MAX-A, V* and *A) will correlate positively with the corresponding metrics from the kinematic model of the non-paretic elbow motion (MAX-V_N, MAX-A_N, V_N* and *A_N)*". Results showed that some of the parameters obtained from the theoretical curve were significantly correlated with their corresponding parameters obtained from the motion curve of the non-paretic elbow. During elbow extension motion (i.e., testing elbow flexor muscles), variables MAX-A and V from the theoretical curve were significantly correlated with their corresponding parameters MAX-A_N and V_N (r=0.66, p=0.003) and (r=0.56, p=0.015), respectively. Results of the correlation analysis are shown in Table 13.

Table 13. Correlation between variables obtained from the theoretical model and metrics obtained from the motion curve of the non-paretic elbow

Elbow flexors (elbow extension motion)					
Theoretical Model		Non-paretic side motion curve			
		MAX-V_N	MAX-A_N	V_N	A_N
MAX-V	r	0.340			
	p	0.108			
MAX-A	r		**0.664**		
	p		**0.003**		
V	r			**0.562**	
	p			**0.015**	
A	r				0.377
	p				0.083
Elbow extensors (elbow flexion motion)					
Theoretical Model		Non-paretic side motion curve			
		MAX-V_N	MAX-A_N	V_N	A_N
MAX-V	r	0.401			
	p	0.069			
MAX-A	r		0.289		
	p		0.148		
V	r			0.092	
	p			0.372	
A	r				0.393
	p				0.074

MAX-V: maximum velocity of the theoretical model; **MAX-A**: absolute maximum acceleration of the theoretical model; **V**: root mean square of the theoretical angular velocity; **A**: root mean square of the theoretical acceleration; **MAX-V_N**: peak magnitude for velocity of the non-paretic limb; **MAX-A_N**: absolute maximum acceleration of the non-paretic limb; **V_N**: root mean square of the non-paretic limb angular velocity; **A_N**: root mean square of the non-paretic limb acceleration; **r**= Pearson's product-moment correlation coefficient; **p**= p-value

Significant findings (i.e., $p<0.05$) are in bold type

4.6 TEST-RETEST RELIABILITY OF SPASTICITY MEASURES

Test-retest reliability was investigated in a sub-group (n=6) of the participants recruited for the study. The null hypothesis for H1.6 "*the BioTone spasticity measures of elbow and knee muscles and clinical measures will show good test-retest reliability*" was partially accepted as some of the spasticity measures showed significant results in test-retest reliability.

4.6.1 RELIABILITY OF SPASTICITY MEASURES FOR ELBOW MUSCLES

The ICC results for elbow flexors showed significant results for MAS (ICC=0.721, p=0.034), TS-Angle (ICC=0.973, p<0.001), ΔA (ICC=0.817, p=0.012) and εA (ICC=0.793, p=0.017). For elbow extensors, ICC was significant for TS-score (ICC=1.00, p<0.001) and angle (ICC=0.990, p<0.001) and εStr (ICC=0.788, p=0.018). Results of all test-retest reliabilities are shown in Table 14.

4.6.2 RELIABILITY OF SPASTICITY MEASURES FOR KNEE EXTENSORS

The ICC results for knee extensors showed significant results for all clinical and BioTone measures. For MAS the ICC was 1.00 (p<0.001), for TS-score the ICC was 1.00 (p<0.001), for TS-Angle the ICC was 0.882 (p=0.004), for RI the ICC was 0.909 p=0.002 and for RI onset angle the ICC was 0.772 (p=0.021).

Table 14. Intra-class correlation coefficients of measures for elbow muscles

Spasticity Measures		Elbow Flexors	Elbow Extensors
MAS	ICC	**0.721**	0.600
	p	**0.034**	0.077
TS-Score	ICC	0.615	**1.00**
	p	0.071	**<0.001**
TS-Angle	ICC	**0.973**	**0.990**
	p	**<0.001**	**<0.001**
ΔV	ICC	0.650	0.056
	p	0.057	0.453
ΔA	ICC	**0.817**	-0.53
	p	**0.012**	0.89
ΔStr	ICC	-0.06	0.502
	p	0.549	0.126
ΔNStr	ICC	-0.13	0.514
	p	0.612	0.119
Onset-Angle	ICC	-0.288	0.384
	p	0.735	0.197
εV	ICC	0.304	0.327
	p	0.254	0.237

Spasticity Measures		Elbow Flexors	Elbow Extensors
εA	ICC	**0.793**	-0.41
	p	**0.017**	0.82
εStr	ICC	-0.45	**0.788**
	p	0.844	**0.018**
εNStr	ICC	-0.01	0.421
	p	0.510	0.174

MAS: Modified Ashworth Scale; **TS**: Tardieu Scale; **ΔV**= peak magnitude of departure in velocity from reference profile; **ΔA**= peak magnitude of departure in acceleration from reference profile; **ΔStr**= EMG intensity at onset time (Te) for stretched muscle; **ΔNStr**= EMG intensity at onset time (Te) for non-stretched muscle; **εV**= root mean square departure from reference profile for angular velocity; **εA=** root mean square departure from reference profile for acceleration; **εStr**= EMG departures from "reference" (area under the curve between T1-T2) for stretched muscle; **εNStr**= EMG departure from "reference" (area under the curve between T1-T2) for non-stretched muscle; **ICC**=Intraclass correlation coefficient; **p**= p-value.

Significant findings (i.e., $p<0.05$) are in bold type

4.7 SECONDARY RESULTS

4.7.1 SPASTICITY MEASURES AND ACTIVE RANGE OF MOTION

For hypothesis H2.1 "*AROM deficit (ΔAROM) of elbow flexion will correlate positively with BioTone spasticity measures of elbow and with MAS and TS for elbow muscles*", the null hypothesis was partially accepted. Results showed that AROM deficit was significantly correlated with εA of elbow flexors (rho= – 0.48, p=0.034), MAS of elbow extensors (rho=0.52, p=0.023) and TS-score of elbow extensors (rho=0.60, p=0.009). Table 15 shows the results of the correlation analysis between AROM deficit and spasticity indicators.

Table 15. Correlation between elbow active range of motion deficit and indicators of elbow muscles spasticity

Spasticity Measures for elbow flexors		Elbow AROM Deficit
MAS	rho (p)	0.399 (0.070)
TS-Score	rho (p)	0.324 (0.120)
ΔV	rho (p)	-0.313 (0.128)
ΔA	rho (p)	-0.160 (0.284)
ΔStr	rho (p)	-0.324 (0.120)
ΔNStr	rho (p)	-0.375 (0.084)
εV	rho (p)	-0.418 (0.060)
εA	rho (p)	**-0.484 (0.034)**
εStr	rho (p)	-0.242 (0.192)
εNStr	rho (p)	-0.346 (0.104)
Spasticity Measures for elbow extensors		Elbow AROM Deficit
MAS	rho (p)	**0.523 (0.023)**
TS-Score	rho (p)	**0.600 (0.009)**
ΔV	rho (p)	0.062 (0.413)
ΔA	rho (p)	0.036 (0.449)

Spasticity Measures for elbow extensors		Elbow AROM Deficit
ΔStr	rho (p)	-0.127 (0.326)
ΔNStr	rho (p)	-0.127 (0.326)
εV	rho (p)	-0.218 (0.217)
εA	rho (p)	-0.022 (0.469)
εStr	rho (p)	0.011 (0.485)
εNStr	rho (p)	-0.131 (0.321)

AROM: Active Range of Motion; **MAS**: Modified Ashworth Scale; **TS**: Tardieu Scale; **ΔV**= peak magnitude of departure in velocity from reference profile; **ΔA**= peak magnitude of departure in acceleration from reference profile; **ΔStr**= EMG intensity at onset time (Te) for stretched muscle; **ΔNStr**= EMG intensity at onset time (Te) for non-stretched muscle; **εV**= root mean square departure from reference profile for angular velocity; **εA=** root mean square departure from reference profile for acceleration; **εStr**= EMG departures from "reference" (area under the curve between T1-T2) for stretched muscle; **εNStr**= EMG departure from "reference" (area under the curve between T1-T2) for non-stretched muscle; **rho**: Spearman's rank correlation coefficient; **p**: p-value.

Significant findings (i.e., $p<0.05$) are in bold type

4.7.2 SPASTICITY MEASURES AND STRENGTH OF ELBOW MUSCULATURE

For hypothesis H2.2 "*elbow flexors (Δ Flexors) and elbow extensors (Δ Extensors) muscles strength deficits will correlate positively with BioTone spasticity measures of elbow and with MAS and TS for elbow muscles*", the null hypothesis was partially accepted. Results showed that the strength deficit of the elbow flexors was significantly correlated with TS-score of elbow flexors (rho=0.49, p=0.029), ΔA of elbow flexors (rho= – 0.48, p=0.034) and TS-score of elbow extensors (rho=0.56, p=0.016). The strength deficit of the elbow extensors was significantly correlated with ΔA of elbow flexors (rho= – 0.52, p=0.023), ΔNStr of elbow flexors (rho= – 0.54, p=0.019), εA of elbow flexors (rho= – 0.51, p=0.026), εStr of elbow flexors (rho= 0.44, p=0.050), MAS of elbow extensors (rho=0.53, p=0.020), TS-score of elbow extensors (rho=0.52, p=0.023), and εStr of elbow extensors (rho= 0.65, p=0.005). Table 16 shows the results of the correlation analysis between elbow strength deficits and spasticity indicators of the elbow muscles.

Table 16. Correlation between elbow strength deficits and indicators of spasticity			
Spasticity Measures for elbow flexors		Elbow Flexion Strength Deficit	Elbow Extension Strength Deficit
MAS	rho (p)	0.429 (0.055)	0.256 (0.179)
TS-Score	rho (p)	**0.499 (0.029)**	0.000 (0.500)
ΔV	rho (p)	-0.132 (0.319)	-0.357 (0.096)
ΔA	rho (p)	**-0.482 (0.034)**	**-0.521 (0.023)**
ΔStr	rho (p)	0.114 (0.343)	0.282 (0.154)
ΔNStr	rho (p)	-0.164 (0.279)	**-0.539 (0.019)**
εV	rho (p)	0.004 (0.495)	-0.221 (0.214)
εA	rho (p)	-0.279 (0.157)	**-0.511 (0.026)**
εStr	rho (p)	0.211 (0.225)	**0.441 (0.050)**
εNStr	rho (p)	0.029 (0.460)	0.264 (0.171)
Spasticity Measures for elbow extensors		Elbow Flexion Strength Deficit	Elbow Extension Strength Deficit
MAS	rho (p)	0.353 (0.098)	**0.534 (0.020)**
TS-Score	rho (p)	**0.556 (0.016)**	**0.524 (0.023)**
ΔV	rho (p)	-0.214 (0.222)	-0.421 (0.059)
ΔA	rho (p)	-0.225 (0.210)	-0.407 (0.066)
ΔStr	rho (p)	0.004 (0.495)	0.321 (0.121)
ΔNStr	rho (p)	-0.039 (0.445)	-0.368 (0.089)
εV	rho (p)	-0.014 (0.480)	-0.175 (0.266)
εA	rho (p)	-0.229 (0.206)	-0.411 (0.064)
εStr	rho (p)	0.389 (0.076)	**0.646 (0.005)**
εNStr	rho (p)	0.131 (0.321)	0.242 (0.193)

MAS: Modified Ashworth Scale; **TS**: Tardieu Scale; **ΔV**= peak magnitude of departure in velocity from reference profile; **ΔA**= peak magnitude of departure in acceleration from reference profile; **ΔStr**= EMG intensity at onset time (Te) for stretched muscle; **ΔNStr**= EMG intensity at onset time (Te) for non-stretched muscle; **εV**= root mean square departure from reference profile for angular velocity; **εA=** root mean square departure from reference profile for acceleration; **εStr**= EMG departures from "reference" for stretched muscle; **εNStr**= EMG departure from "reference" for non-stretched muscle; **rho**: Spearman's rank correlation coefficient; **p**: p-value.

Significant findings (i.e., $p<0.05$) are in bold type

In summary, our results show that the participants recruited had mild spasticity and there were no significant differences found in the spastic onset time determined from kinematic and EMG data sets. Further, indicators of spasticity were quantified using these data sets and when comparing the indicators obtained from the kinematic data set to the indicators obtained from the EMG data set, none of the hypothesized significant associations were found. However, when comparing these indicators to clinical measures of spasticity, some significant correlations were found for both the elbow musculature data and pendulum test data. In addition, some significant correlations were found when validating the theoretical model and comparing it to the motion curve of the non-paretic elbow. In regard to reliability, our results showed that some of the BioTone and clinical measures used to assess spasticity have good test-retest reliability. Finally, our secondary analysis showed that there are a few significant associations between the amount of spasticity in the elbow muscles and the elbow flexion AROM and strength deficits.

CHAPTER 5

DISCUSSION

5.1 DISCUSSION

The measurement of spasticity is an integral part of the rehabilitation process of patients who have increased tone after stroke.[1, 8] The BioTone system is a device that can potentially aid clinicians in the assessment of spasticity. We found that, with minimal training, the system was easy to use in the clinical setting. The assessment process for all four limbs requires approximately 2-3 hours, but time required to assess one limb is about 20-30 minutes. This seems considerably long compared to clinical scales; however, we noted in our systematic review on instrumented measures that other instrumented devices require similar time to conduct a spasticity assessment.[69] Further, the participants in our study did not express any concerns or discomfort during the assessment procedure. However, compared to commercially available instrumental measures (e.g., NeuroFlexor and Myoton-3 Myotonometer), the BioTone, in its current stage of development, involves complex data analysis, the results of which cannot be easily interpreted.

The results of this study provide preliminary information regarding the concurrent and construct validity and test-retest reliability of the BioTone system. Most participants had mild spasticity in the elbow muscles and in the knee extensor muscles (i.e., MAS scores of 1-1+ and TS scores of 1-2). Lack of variability in MAS and TS scores may have contributed to the pattern of UE spasticity that we observed. Our results showed that there was no significant difference in the level of spasticity between flexor and extensor muscle groups, which is counter to the pattern of greater flexor than extensor tone typically seen post-stroke.[1] Nonetheless, a previous study involving 665 patients post-stroke, noted exceptions to this pattern – about 15% of the participants presented with an extensor pattern of spasticity.[30]

In terms of the construct validity of the Biotone, our finding of a lack of difference between spastic onset times determined using EMG and kinematic data is consistent with the results reported in the only paper published to date on the Biotone.[83] McGibbon et al.,[83] used the device to quantify elbow muscles spasticity among nine participants who had a variety of neurological conditions (e.g., stroke, cerebral palsy, multiple sclerosis, spinal cord injury). They also noted a few significant correlations observed between the kinematic and EMG indicators of spasticity, and between the

BioTone measures of spasticity and the MAS. In contrast, we found no significant associations in the hypothesized correlations between EMG and kinematic measures of spasticity. One possible contributing factor to these disparate results was that all of our participants had spasticity secondary to stroke whereas McGibbon and colleagues[83] included participants with different neurological conditions and not all of them had spasticity.

Regarding the concurrent validity of the Biotone, comparing the BioTone measures to the clinical spasticity measures (MAS and TS) yielded mixed findings. For the knee extensor muscles, the RI was found to have a moderate (rho= –0.54) and strong (rho= –0.65) correlation with the MAS and TS, respectively. These results confirm previous studies that reported of good validity of the Pendulum Test as an assessment tool for knee extensor muscles spasticity.[62, 65] Further, using the RI as a measure of spasticity has the advantage of having smaller increments than the MAS (and TS), which may result in grading spasticity more accurately and eliminating the disagreement between MAS levels as shown in several studies.[10, 12, 33]

Only few of the BioTone measures for the elbow musculature were found to have significant correlations with the clinical spasticity scales. There are several possible explanations for this lack of association. It may be that theoretical model from which the Biotone measures were derived did not yield a true representation of the amount of spasticity in the elbow muscles. In support of the notion of a potentially flawed model we noted that not all indices from the theoretical model were correlated with the motion curve of the non-hemiparetic elbow. The estimation of the theoretical model is based on the notion that the examiner induced a "constant jerk"when passively moving the elbow of the participant. This constant jerk is a derivative of acceleration, and since the motion was induced manually by the examiner and not by a motor system, the constant jerk is does not account for the peak of acceleration. This peak of acceleration produced by the examiner is not constant and changes during each assessment of the participant. Scaling the theoretical profile to fit the actual motion curve (given that actual range is known) does not account for small differences in acceleration. The other issue with estimating a constant jerk is that it does not account for the elastic properties of the muscle, which

differ between individuals and in effect lead to achieving different acceleration peaks, which will also affect the estimation of the theoretical model.

Another reason for the lack of significant correlations between the two motion curves (theoretical and non-hemiparetic elbow motion curves) may be that our approach to validating the motion of the theoretical curve using the non-paretic side may not be valid. We based this comparison on the assumption that the non-paretic side is 'non-affected' and that the stretch reflex response is 'normal' after stroke. However, the work of Thilmann et al.[125] challenged this assumption. The investigators recorded, in 10 patients with spasticity following a single localized stroke in one hemisphere and 10 age-matched, non-disabled subjects, the stretch reflex EMG responses of bilateral biceps and triceps muscles following imposed stretches. On the affected side, the biceps response to elbow extension was exaggerated compared with normal values and displayed a reduced velocity threshold to stretch reflex. The triceps, in contrast, showed depressed responses to elbow flexion, with a much higher velocity threshold than non-disabled subjects. Further, on the purportedly "unaffected" side, the reciprocal pattern was seen, with depression of the biceps response and a rising of its velocity threshold, along with considerably exaggerated responses in the triceps muscles. The authors postulated that the increased excitability of the flexor musculature on the spastic side may be paralleled by an increase in activity in the segmental pathways responsible for modulation of agonist/antagonist activity in the ipsi- and contra-lateral limb, leading to ipsilateral extensor and contralateral flexor inhibition and contralateral extensor excitation. They concluded that the "good" side of hemiparetic patients also undergoes pathological changes, and studies of the mechanisms of spasticity post-stroke should avoid the use of the "unaffected" side as a control for monitoring pathological reflexes. In support of this interpretation we found significant correlations between the metrics from the theoretical curve and variables measured during stretch of the non-hemiparetic elbow flexors, which, if the interpretation by Thilmann et al. is correct,[125] would simulate a 'normal' (spastic-free) muscle.

Examining test-retest reliability of the spasticity measures used in our study also generated mixed results. For elbow muscles, ΔA, εA and MAS of elbow flexors and εStr and TS-score and angle for elbow extensors showed significant results for the ICC with a

range from (ICC=0.72-1.00), indicating high test-retest reliability. However, not all measures showed significant p-values which has been reported in previous studies that utilize instruments for measuring spasticity, particularly EMG.[72, 90, 105] Given that EMG is sensitive to changes in spasticity,[126] and spasticity post-stroke may present in various degrees of severity between different days and different times of the day,[1, 3] it is reasonable to find non-significant ICCs for some of the spasticity measures in our study. For knee extensor muscles, the test-retest reliability of BioTone and clinical measure was in the good to excellent range (ICCs of 0.77-1.00). Our results were similar to what Bohannon et al.,[62] reported — excellent test-retest reliability of the pendulum test with an ICC range (0.92-0.96). These findings support the use of the pendulum test as a measure of knee extensors spasticity in the clinical setting.

The results of the secondary analysis showed a few significant associations between the amount of elbow flexor spasticity and elbow flexion AROM and elbow flexor strength deficits. Similar relationships have been previously documented by other studies,[127, 128] yet, it is remains unclear if there is a causal association between spasticity and deficits in AROM and muscle strength.[129]

In summary, determining valid and reliable spasticity measurements from the data collected with the Biotone remains an issue. As mentioned above, using a theoretical model to obtain indices for spasticity may not be an accurate approach for analyzing the kinematic data. The approach described by Bohannon et al.,[62] and also by Le Cavorzin et al.,[65] of using the area under the motion curve of the spastic limb as a measure of spasticity might be more valid in quantifying spasticity using kinematic data. Further, the analysis of EMG data as shown in this could be used as a measure of spasticity when using the BioTone, especially since studies have already demonstrated that the validity and reliability of the TSRT is good.[90, 103] Nonetheless, it should be stated that, the BioTone is a user friendly and clinically usable device that with further research and development may become a practical tool for clinicians to objectively measure spasticity.

5.2 Limitations

The most substantive limitation is that it was a pilot study involving a small sample. Therefore, the results should be interpreted with caution. The findings of the on-going large multi-center study, as well as future research regarding the BioTone system, will confirm or refute our findings. In addition, the MAS and TS scales, chosen to investigate the concurrent validity of the BioTone measures, have been criticized for being too subjective.[12, 49] Future studies should investigate the validity of the BioTone against electrophysiological measures, such as the H-reflex, which is a measure of alpha motor neuron excitability and therefore, a more accurate measure of spasticity.[130, 131]

5.3 Conclusions

Despite the notable limitation of the sample size, this study provides preliminary information of aspects of construct and concurrent validity and test-retest reliability of the BioTone system. Further investigation of the clinical utility of this device as a clinical tool to objectively measure spasticity in patients post-stroke is warranted. To reduce functional, emotional, and financial burdens of a common aftermath of stroke — spasticity — a valid, reliable and user-friendly tool of objectively measuring its clinical presentation is needed. This study makes a meaningful contribution to the on-going development of a potential measurement device – the BioTone system.

REFERENCES

1. Barnes MP, Johnson GR. *Upper Motor Neurone Syndrome and Spasticity Clinical Management and Neurophysiology*. 2nd ed. Cambridge: Cambridge University Press; 2008.

2. Stein J. *Stroke recovery and rehabilitation*. New York: Demos Medical; 2009.

3. Brashear A, Elovic E. *Spasticity diagnosis and management*. New York: Demos Medical Pub.; 2011.

4. Sommerfeld DK, Gripenstedt U, Welmer AK. Spasticity after stroke: an overview of prevalence, test instruments, and treatments. *Am J Phys Med Rehabil.* Sep 2012;91(9):814-820.

5. Urban PP, Wolf T, Uebele M, et al. Occurence and clinical predictors of spasticity after ischemic stroke. *Stroke; a journal of cerebral circulation.* Sep 2010;41(9):2016-2020.

6. Lance JW. The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture. *Neurology.* Dec 1980;30(12):1303-1313.

7. Sheean G. The pathophysiology of spasticity. *Eur J Neurol.* May 2002;9 Suppl 1:3-9; dicussion 53-61.

8. Burridge JH, Wood DE, Hermens HJ, et al. Theoretical and methodological considerations in the measurement of spasticity. *Disability and rehabilitation.* Jan 7-21 2005;27(1-2):69-80.

9. van Wijck FM, Pandyan AD, Johnson GR, Barnes MP. Assessing motor deficits in neurological rehabilitation: patterns of instrument usage. *Neurorehabilitation and neural repair.* 2001;15(1):23-30.

10. Fleuren JF, Voerman GE, Erren-Wolters CV, et al. Stop using the Ashworth Scale for the assessment of spasticity. *J Neurol Neurosurg Psychiatry.* Jan 2010;81(1):46-52.

11. Malhotra S, Pandyan AD, Day CR, Jones PW, Hermens H. Spasticity, an impairment that is poorly defined and poorly measured. *Clinical rehabilitation.* Jul 2009;23(7):651-658.

12. Pandyan AD, Johnson GR, Price CIM, Curless RH, Barnes MP, Rodgers H. A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measures of spasticity. *Clinical rehabilitation.* 1999;13(5):373-383.

13. Katz RT, Rovai GP, Brait C, Rymer WZ. Objective quantification of spastic hypertonia: correlation with clinical findings. *Archives of physical medicine and rehabilitation.* Apr 1992;73(4):339-347.

14. Kim DY, Park CI, Chon JS, Ohn SH, Park TH, Bang IK. Biomechanical assessment with electromyography of post-stroke ankle plantar flexor spasticity. *Yonsei medical journal.* 2005;46(4):546-554.

15. Milanov I. Clinical and neurophysiological correlations of spasticity. *Funct Neurol.* Oct-Dec 1999;14(4):193-201.

16. Mullick AA, Musampa NK, Feldman AG, Levin MF. Stretch reflex spatial threshold measure discriminates between spasticity and rigidity. *Clin Neurophysiol.* Apr 2013;124(4):740-751.

17. Fisher M. *Stroke: Investigation and Management*. New York: Elsevier Health Sciences; 2009.

18. Ashton Acton Q. *Hemorrhagic Stroke: New Insights for the Healthcare Professional: 2012 Edition ScholarlyPaper*: Scholarly Editions; 2012.

19. *Statistics*: Heart and Stroke Foundation;2012.

20. *Tracking heart disease and stroke in Canada*: Public Health Agency of Canada;2009.

21. *The global burden of disease: 2004 update*: World Health Orginization;2008.

22. Norrving B, Kissela B. The global burden of stroke and need for a continuum of care. *Neurology.* Jan 15 2013;80(3 Suppl 2):S5-12.

23. Ada L, Canning C. Changing the way we view the contribution of motor impairments to physical disability after stroke. In: Refshauge K, Ada L, Ellis E, eds. *Science-based Rehabilitation, Theories into Practice*. Edinburgh: Butterworth Heinemann; 2005:87-106.

24. Gracies JM. Pathophysiology of spastic paresis. I: Paresis and soft tissue changes. *Muscle Nerve.* May 2005;31(5):535-551.

25. O'Dwyer NJ, Ada L. Reflex hyperexcitability and muscle contracture in relation to spastic hypertonia. *Curr Opin Neurol.* Dec 1996;9(6):451-455.

26. Schinwelski M, Slawek J. Prevalence of spasticity following stroke and its impact on quality of life with emphasis on disability in activities of daily living. Systematic review. *Neurol Neurochir Pol.* Jul-Aug 2010;44(4):404-411.

27. 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; a journal of cerebral circulation.* Jan 2004;35(1):134-139.

28. Welmer AK, von Arbin M, Widen Holmqvist L, Sommerfeld DK. Spasticity and its association with functioning and health-related quality of life 18 months after stroke. *Cerebrovascular diseases.* 2006;21(4):247-253.

29. Esquenazi A. The human and economic burden of post-stroke spasticity and muscle over-activity. *JCOM.* 2011;18(1):607-614.

30. Hefter H, Jost WH, Reissig A, Zakine B, Bakheit AM, Wissel J. Classification of posture in poststroke upper limb spasticity: a potential decision tool for botulinum toxin A treatment? *Int J Rehabil Res.* Sep 2012;35(3):227-233.

31. Ashworth B. Preliminary Trial of Carisoprodol in Multiple Sclerosis. *Practitioner.* Apr 1964;192:540-542.

32. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther.* Feb 1987;67(2):206-207.

33. Ansari NN, Naghdi S, Moammeri H, Jalaie S. Ashworth Scales are unreliable for the assessment of muscle spasticity. *Physiother Theory Pract.* Jun 2006;22(3):119-125.

34. Patrick E, Ada L. The Tardieu Scale differentiates contracture from spasticity whereas the Ashworts Scale is confounded by it. *Clinical rehabilitation.* 2006;20(2):173-181.

35. Vattanasilp W, Ada L. The relationship between clinical and laboratory measures of spasticity. *Australian Journal of Physiotherapy.* 1999;45(2):135-139.

36. Ghotbi N, Hadian MR, Olyaei GR, et al. The investigation of criterion validity of the modified Ashworth scale using the alpha motoneuron excitability indicators. *Acta medica Iranica.* 2007;45(4):290-294.

37. Naghdi S, Ansari NN, Mansouri K, Olyaei GR, Asgari A, Kazemnejad A. The correlation between Modified Ashworth Scale scores and the new index of alpha motoneurones excitability in post-stroke patients. *Electromyogr Clin Neurophysiol.* Mar 2008;48(2):109-115.

38. Pisano F, Miscio G, Del Conte C, Pianca D, Candeloro E, Colombo R. Quantitative measures of spasticity in post-stroke patients. *Clin Neurophysiol.* Jun 2000;111(6):1015-1022.

39. Cooper A, Musa IM, van Deursen R, Wiles CM. Electromyography characterization of stretch responses in hemiparetic stroke patients and their relationship with the Modified Ashworth scale. *Clinical rehabilitation.* Oct 2005;19(7):760-766.

40. Pandyan AD, Price CI, Barnes MP, Johnson GR. A biomechanical investigation into the validity of the modified Ashworth Scale as a measure of elbow spasticity. *Clinical rehabilitation.* May 2003;17(3):290-293.

41. Naghdi S, Ebrahimi I, Asgari A, et al. A preliminary study into the criterion validity of the Modified Modified Ashworth Scale using the new measure of the alpha motoneuron excitability in spastic hemiplegia. *Electromyogr Clin Neurophysiol.* May-Jun 2007;47(3):187-192.

42. Naghdi S, Ansari NN, Mansouri K, Asgari A, Olyaei GR, Kazemnejad A. Neurophysiological examination of the Modified Modified Ashworth Scale (MMAS) in patients with wrist flexor spasticity after stroke. *Electromyogr Clin Neurophysiol.* Jan-Feb 2008;48(1):35-41.

43. Brashear A, Zafonte R, Corcoran M, et al. Inter- and intrarater reliability of the Ashworth Scale and the Disability Assessment Scale in patients with upper-limb poststroke spasticity. *Archives of physical medicine and rehabilitation.* Oct 2002;83(10):1349-1354.

44. Gregson JM, Leathley MJ, Moore AP, Smith TL, Sharma AK, Watkins CL. Reliability of measurements of muscle tone and muscle power in stroke patients. *Age Ageing.* 2000;29(3):223-228.

45. Ansari NN, Naghdi S, Younesian P, Shayeghan M. Inter- and intrarater reliability of the Modified Modified Ashworth Scale in patients with knee extensor poststroke spasticity. *Physiother Theory Pract.* May-Jun 2008;24(3):205-213.

46. Ansari NN, Naghdi S, Hasson S, Mousakhani A, Nouriyan A, Omidvar Z. Inter-rater reliability of the Modified Modified Ashworth Scale as a clinical tool in measurements of post-stroke elbow flexor spasticity. *NeuroRehabilitation.* 2009;24(3):225-229.

47. Tardieu G, Shentoub S, Delarue R. [Research on a technic for measurement of spasticity]. *Rev Neurol (Paris).* 1954;91(2):143-144.

48. Held JP, Pierrot-Deseilligny E. *Reeducation mortrice des affections neurologiques*: J. B. Balliere et fils.; 1969.

49. Haugh AB, Pandyan AD, Johnson GR. A systematic review of the Tardieu Scale for the measurement of spasticity. *Disability and rehabilitation.* Aug 15 2006;28(15):899-907.

50. Boyd RN, Graham HK. Objective measurement of clinical findings in the use of botulinum toxin type A for the management of children with cerebral palsy. *European Journal of Neurology.* 1999;6(S4):s23-s35.

51. Naghdi S, Ansari NN, Abolhasani H, Mansouri K, Ghotbi N, Hasson S. Electrophysiological evaluation of the Modified Tardieu Scale (MTS) in assessing poststroke wrist flexor spasticity. *NeuroRehabilitation.* Jan 1 2014;34(1):177-184.

52. Paulis WD, Horemans HL, Brouwer BS, Stam HJ. Excellent test-retest and inter-rater reliability for Tardieu Scale measurements with inertial sensors in elbow flexors of stroke patients. *Gait Posture.* Feb 2011;33(2):185-189.

53. Singh P, Joshua AM, Ganeshan S, Suresh S. Intra-rater reliability of the modified Tardieu scale to quantify spasticity in elbow flexors and ankle

plantar flexors in adult stroke subjects. *Ann Indian Acad Neurol.* Jan 2011;14(1):23-26.

54. Ansari NN, Naghdi S, Hasson S, Rastgoo M, Amini M, Forogh B. Clinical assessment of ankle plantarflexor spasticity in adult patients after stroke: inter-and intra-rater reliability of the Modified Tardieu Scale. *Brain Inj.* 2013;27(5):605-612.

55. Levin MF, Hui-Chan CW. Relief of hemiparetic spasticity by TENS is associated with improvement in reflex and voluntary motor functions. *Electroencephalogr Clin Neurophysiol.* Apr 1992;85(2):131-142.

56. Levin MF, Feldman AG. The role of stretch reflex threshold regulation in normal and impaired motor control. *Brain research.* Sep 19 1994;657(1-2):23-30.

57. 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. *Archives of physical medicine and rehabilitation.* Aug 2005;86(8):1641-1647.

58. Wartenberg R. Pendulousness of the legs as a diagnostic test. *Neurology.* 1951;1(1):18-18.

59. Bajd T, Bowman B. Measurement of skeletal muscle spasticity. *Proc. Int. Symp. External Control of Human Extremities, Yugoslav Committee for Electronics & Automation, Belgrade.* 1981:111-119.

60. Bajd T, Bowman B. Testing and modelling of spasticity. *J Biomed Eng.* Apr 1982;4(2):90-96.

61. Bajd T, Vodovnik L. Pendulum testing of spasticity. *J Biomed Eng.* Jan 1984;6(1):9-16.

62. Bohannon RW, Harrison S, Kinsella-Shaw J. Reliability and validity of pendulum test measures of spasticity obtained with the Polhemus tracking system from patients with chronic stroke. *J Neuroeng Rehabil.* 2009;6:30.

63. Le Cavorzin P, Poudens SA, Chagneau F, Carrault G, Allain H, Rochcongar P. A comprehensive model of spastic hypertonia derived from the pendulum test of the leg. *Muscle Nerve.* Dec 2001;24(12):1612-1621.

64. Stillman B, McMeeken J. A video-based version of the pendulum test: Technique and normal response. *Archives of physical medicine and rehabilitation.* 1995;76(2):166-176.

65. Le Cavorzin P, Hernot X, Bartier O, et al. A computed model of the pendulum test of the leg for routine assessment of spasticity in man. *ITBM-RBM.* 2001;22(3):170-177.

66. Chuang LL, Wu CY, Lin KC. Reliability, validity, and responsiveness of myotonometric measurement of muscle tone, elasticity, and stiffness in patients with stroke. *Archives of physical medicine and rehabilitation.* 2012;93(3):532-540.

67. Chuang LL, Lin KC, Wu CY, et al. Relative and absolute reliabilities of the myotonometric measurements of hemiparetic arms in patients with stroke. *Archives of physical medicine and rehabilitation.* Mar 2013;94(3):459-466.

68. Chuang LL, Wu CY, Lin KC, Lur SY. Quantitative mechanical properties of the relaxed biceps and triceps brachii muscles in patients with subacute stroke: a reliability study of the myoton-3 myometer. *Stroke Res Treat.* 2012;2012:617694.

69. Malhotra S, Cousins E, Ward A, et al. An investigation into the agreement between clinical, biomechanical and neurophysiological measures of spasticity. *Clinical rehabilitation.* 2008;22(12):1105-1115.

70. Pandyan AD, Price CI, Rodgers H, Barnes MP, Johnson GR. Biomechanical examination of a commonly used measure of spasticity. *Clin Biomech (Bristol, Avon).* Dec 2001;16(10):859-865.

71. Pandyan AD, Van Wijck FM, Stark S, Vuadens P, Johnson GR, Barnes MP. The construct validity of a spasticity measurement device for clinical practice: an alternative to the Ashworth scales. *Disability and rehabilitation.* May 15 2006;28(9):579-585.

72. Voerman GE, Burridge JH, Hitchcock RA, Hermens HJ. Clinometric properties of a clinical spasticity measurement tool. *Disability and rehabilitation.* Dec 30 2007;29(24):1870-1880.

73. Lee HM, Chen JJJ, Ju MS, Lin CCK, Poon PPW. Validation of portable muscle tone measurement device for quantifying velocity-dependent properties in elbow spasticity. *Journal of Electromyography and Kinesiology.* 2004;14(5):577-589.

74. Lindberg PG, Gaverth J, Islam M, Fagergren A, Borg J, Forssberg H. Validation of a new biomechanical model to measure muscle tone in spastic muscles. *Neurorehabilitation and neural repair.* 2011;25(7):617-625.

75. Voerman GE, Gregoric M, Hermens HJ. Neurophysiological methods for the assessment of spasticity: The Hoffman reflex, the tendon reflex, and the stretch reflex. *Disability and rehabilitation.* 2005;27(1-2):33-68.

76. Matthews WB. Ratio of maximum H reflex to maximum M response as a measure of spasticity. *J Neurol Neurosurg Psychiatry.* Jun 1966;29(3):201-204.

77. Levin MF, Hui-Chan C. Are H and stretch reflexes in hemiparesis reproducible and correlated with spasticity? *Journal of neurology.* Feb 1993;240(2):63-71.

78. Calota A, Levin MF. Tonic stretch reflex threshold as a measure of spasticity: implications for clinical practice. *Top Stroke Rehabil.* May-Jun 2009;16(3):177-188.

79. Min JH, Shin YI, Joa KL, et al. The Correlation between Modified Ashworth Scale and Biceps T-reflex and Inter-rater and Intra-rater Reliability of Biceps T-reflex. *Ann Rehabil Med.* Aug 2012;36(4):538-543.

80. Fellows SJ, Ross HF, Thilmann AF. The limitations of the tendon jerk as a marker of pathological stretch reflex activity in human spasticity. *J Neurol Neurosurg Psychiatry.* May 1993;56(5):531-537.

81. Hermens H, Freriks B, Merletti R, et al. *European Recommendations for Surface ElectroMyoGraphy: Results of the SENIAM Project*: Roessingh Research and Development; 2000.

82. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. *J Electromyogr Kinesiol.* Oct 2000;10(5):361-374.

83. McGibbon CA, Sexton A, Jones M, O'Connell C. Elbow spasticity during passive stretch-reflex: clinical evaluation using a wearable sensor system. *J Neuroeng Rehabil.* 2013;10(1):61.

84. Dowling JJ. The effect of muscle mechanics on human movement outcomes as revealed by computer simulation. *Human movement science.* 1992;11(3):273-297.

85. Mohr J, Choi D, Grotta J, Weir B, Wolf P. *Stroke: Pathophysiology, diagnosis, and management*. New York: Churchill Livingstone; 2004.

86. Ward AB. A literature review of the pathophysiology and onset of post-stroke spasticity. *European Journal of Neurology.* 2012;19(1):21-27.

87. Lieber RL, Steinman S, Barash IA, Chambers H. Structural and functional changes in spastic skeletal muscle. *Muscle Nerve.* May 2004;29(5):615-627.

88. MacKenzie CR, Charlson ME. Standards for the use of ordinal scales in clinical trials. *Br Med J (Clin Res Ed).* Jan 4 1986;292(6512):40-43.

89. Wood DE, Burridge JH, van Wijck FM, et al. Biomechanical approaches applied to the lower and upper limb for the measurement of spasticity: a systematic review of the literature. *Disability and rehabilitation.* Jan 7-21 2005;27(1-2):19-32.

90. Calota A, Feldman AG, Levin MF. Spasticity measurement based on tonic stretch reflex threshold in stroke using a portable device. *Clin Neurophysiol.* Oct 2008;119(10):2329-2337.

91. Everitt BS. *Statistical methods in medical investigations*: Edward Arnold London; 1994.

92. Wilkin D, Hallam L, Doggett M-A. *Measures of need and outcome for primary health care*: Oxford University Press; 1992.

93. Adamson J, Beswick A, Ebrahim S. Is stroke the most common cause of disability? *J Stroke Cerebrovasc Dis.* Jul-Aug 2004;13(4):171-177.

94. Sheean G, McGuire JR. Spastic hypertonia and movement disorders: pathophysiology, clinical presentation, and quantification. *Pm R.* Sep 2009;1(9):827-833.

95. Bar-On L, Aertbelien E, Molenaers G, Dan B, Desloovere K. Manually controlled instrumented spasticity assessments: a systematic review of psychometric properties. *Developmental medicine and child neurology.* Mar 17 2014.

96. Flamand VH, Masse-Alarie H, Schneider C. Psychometric evidence of spasticity measurement tools in cerebral palsy children and adolescents: a systematic review. *J Rehabil Med.* Jan 2013;45(1):14-23.

97. Platz T, Eickhof C, Nuyens G, Vuadens P. Clinical scales for the assessment of spasticity, associated phenomena, and function: a systematic review of the literature. *Disability and rehabilitation.* Jan 7-21 2005;27(1-2):7-18.

98. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *Ann Intern Med.* Aug 18 2009;151(4):264-269, W264.

99. Law MC, MacDermid J. *Evidence-based rehabilitation: A guide to practice*: Slack Incorporated; 2008.

100. Roy JS, Desmeules F, MacDermid JC. Psychometric properties of presenteeism scales for musculoskeletal disorders: a systematic review. *J Rehabil Med.* Jan 2011;43(1):23-31.

101. 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.* Jan 2002;82(1):25-34.

102. Gaverth J, Sandgren M, Lindberg PG, Forssberg H, Eliasson AC. Test-retest and inter-rater reliability of a method to measure wrist and finger spasticity. *J Rehabil Med.* Jul 2013;45(7):630-636.

103. 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.* Jan 2011;33(1):62-69.

104. Kaya T, Karatepe AG, Gunaydin R, Koc A, Altundal Ercan U. Inter-rater reliability of the Modified Ashworth Scale and modified Modified Ashworth Scale in assessing poststroke elbow flexor spasticity. *Int J Rehabil Res.* Mar 2011;34(1):59-64.

105. 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. *Neurorehabilitation and neural repair.* Mar-Apr 2009;23(3):287-294.

106. Gregson JM, Leathley M, Moore AP, Sharma AK, Smith TL, Watkins CL. Reliability of the Tone Assessment Scale and the modified Ashworth scale as clinical tools for assessing poststroke spasticity. *Archives of physical medicine and rehabilitation.* Sep 1999;80(9):1013-1016.

107. Kumar RT, Pandyan AD, Sharma AK. Biomechanical measurement of post-stroke spasticity. *Age Ageing.* Jul 2006;35(4):371-375.

108. Takeuchi N, Kuwabara T, Usuda S. Development and evaluation of a new measure for muscle tone of ankle plantar flexors: the ankle plantar flexors tone scale. *Archives of physical medicine and rehabilitation.* Dec 2009;90(12):2054-2061.

109. Wu YN, Huang SC, Chen JJJ, Wang YL, Piotrkiewicz M. Spasticity Evaluation of Hemiparetic Limbs in Stroke Patients before Intervention by Using Portable Stretching Device and EMG. *Journal of Medical and Biological Engineering.* 2004;24(1):29-35.

110. Bakheit AM, Maynard VA, Curnow J, Hudson N, Kodapala S. The relation between Ashworth scale scores and the excitability of the alpha motor neurones in patients with post-stroke muscle spasticity. *J Neurol Neurosurg Psychiatry.* May 2003;74(5):646-648.

111. Barnes S, Gregson J, Leathley M, Smith T, Sharma A, Watkins C. Development and inter-rater reliability of an assessment tool for measuring muscle tone in people with hemiplegia after a stroke. *Physiotherapy.* 1999;85(8):405-409.

112. Chen JJJ, Wu YN, Huang SC, Lee HM, Wang YL. The use of a portable muscle tone measurement device to measure the effects of botulinum toxin type A on elbow flexor spasticity. *Archives of physical medicine and rehabilitation.* 2005;86(8):1655-1660.

113. Pomeroy VM, Dean D, Sykes L, et al. The unreliability of clinical measures of muscle tone: implications for stroke therapy. *Age Ageing.* May 2000;29(3):229-233.

114. Worley JS, Harmon N, Miller GJ, Hollis SB, Harlow S, Briggs V. Reliability of potential clinical measures of muscle tone in the elbows of patients after stroke. *Am J Occup Ther.* Jul-Aug 1996;50(7):554-560.

115. Lin CC, Ju MS, Lin CW. The pendulum test for evaluating spasticity of the elbow joint. *Archives of physical medicine and rehabilitation.* Jan 2003;84(1):69-74.

116. Portney L, Watkins M. Validity of measurements. *Foundations of clinical research: Applications to practice*. Upper Saddle River, NJ: Pearson/Prentice Hall; 2009:79-110.

117. Atkinson G, Nevill AM. Statistical methods for assessing measurement error (reliability) in variables relevant to sports medicine. *Sports Med.* Oct 1998;26(4):217-238.

118. Munro BH. *Statistical methods for health care research.* Vol 1: Lippincott Williams & Wilkins; 2005.

119. Horstman AM. Comments on Point: Counterpoint: The interpolated twitch does/does not provide a valid measure of the voluntary activation of muscle. *Journal of Applied Physiology.* 2009;107(1):359-366.

120. Horstman AM, Beltman MJ, Gerrits KH, et al. Intrinsic muscle strength and voluntary activation of both lower limbs and functional performance after stroke. *Clin Physiol Funct Imaging.* Jul 2008;28(4):251-261.

121. Brennan P, Silman A. Statistical methods for assessing observer variability in clinical measures. *Bmj.* Jun 6 1992;304(6840):1491-1494.

122. Bruton A, Conway JH, Holgate ST. Reliability: what is it, and how is it measured? *Physiotherapy.* 2000;86(2):94-99.

123. Bhimani RH, Anderson LC, Henly SJ, Stoddard SA. Clinical measurement of limb spasticity in adults: state of the science. *J Neurosci Nurs.* Apr 2011;43(2):104-115.

124. Siegel S. Nonparametric statistics for the behavioral sciences. 1956.

125. Thilmann AF, Fellows SJ, Garms E. Pathological stretch reflexes on the "good" side of hemiparetic patients. *J Neurol Neurosurg Psychiatry.* Mar 1990;53(3):208-214.

126. Harburn KL, Hill KM, Vandervoort AA, et al. Spasticity measurement in stroke: A pilot study. *Canadian Journal of Public Health.* 1992;83(SUPPL. 2):S41-S45.

127. Bohannon RW. Relationship between active range of motion deficits and muscle strength and tone at the elbow in patients with hemiparesis. *Clinical rehabilitation.* 1991;5(3):219-224.

128. Bohannon RW, Larkin PA, Smith MB, Horton MG. Relationship between static muscle strength deficits and spasticity in stroke patients with hemiparesis. *Phys Ther.* Jul 1987;67(7):1068-1071.

129. 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. *Disability and rehabilitation.* Jul 15-30 2006;28(13-14):891-897.

130. Angel RW, Hofmann WW. The H Reflex in Normal, Spastic, and Rigid Subjects. *Arch Neurol.* Jun 1963;9:591-596.

131. Bour LJ, Ongerboer de Visser BW, Koelman JH, van Bruggen GJ, Speelman JD. Soleus H-reflex tests in spasticity and dystonia: A computerized analysis. *J Electromyogr Kinesiol.* 1991;1(1):9-19.

APPENDIX A

Copyright Permission Letters

June 20, 2014

Johan Gäverth
School of Physiotherapy
Dalhousie University
Halifax, Nova Scotia
B3H 4R2

Dear Dr Johan Gäverth

I am preparing my Masters thesis for submission to the Faculty of Graduate Studies at Dalhousie University, Halifax, Nova Scotia, Canada. I am seeking your permission, as a co-author, to include a manuscript version of the following paper as a chapter in the thesis:

Measurement of spasticity after stroke using clinical measures: a systematic review. *Aloraini S, Gäverth J, Yeung E, MacKay-Lyons M.*

Canadian graduate theses are reproduced by the Library and Archives of Canada (formerly National Library of Canada) through a non-exclusive, world-wide license to reproduce, loan, distribute, or sell theses. I am also seeking your permission for the material described above to be reproduced and distributed by the LAC(NLC). Further details about the LAC(NLC) thesis program are available on the LAC(NLC) website (www.nlc-bnc.ca).

Full publication details and a copy of this permission letter will be included in the thesis.

Yours sincerely,

Saleh M. Aloraini

Permission is granted for:

a) the inclusion of the material described above in your thesis.

b) for the material described above to be included in the copy of your thesis that is sent to the Library and Archives of Canada (formerly National Library of Canada) for reproduction and distribution.

Name: Johan Gäverth Title: Post-Doctoral Fellow

Signature: _____ Date: _____

June 20, 2014

Ellen Yeung
School of Physiotherapy
Dalhousie University
Halifax, Nova Scotia
B3H 4R2

Dear Ms Ellen Yeung

I am preparing my Masters thesis for submission to the Faculty of Graduate Studies at Dalhousie University, Halifax, Nova Scotia, Canada. I am seeking your permission, as a co-author, to include a manuscript version of the following paper as a chapter in the thesis:

Measurement of spasticity after stroke using clinical measures: a systematic review. *Aloraini S, Gäverth J, Yeung E, MacKay-Lyons M.*

Canadian graduate theses are reproduced by the Library and Archives of Canada (formerly National Library of Canada) through a non-exclusive, world-wide license to reproduce, loan, distribute, or sell theses. I am also seeking your permission for the material described above to be reproduced and distributed by the LAC(NLC). Further details about the LAC(NLC) thesis program are available on the LAC(NLC) website (www.nlc-bnc.ca).

Full publication details and a copy of this permission letter will be included in the thesis.

Yours sincerely,

Saleh M. Aloraini

Permission is granted for:

a) the inclusion of the material described above in your thesis.

b) for the material described above to be included in the copy of your thesis that is sent to the Library and Archives of Canada (formerly National Library of Canada) for reproduction and distribution.

Name: Ellen Yeung Title: Research Assistant

Signature: _____ Date: _____

June 20, 2014

Marilyn MacKay-Lyons
School of Physiotherapy
Dalhousie University
Halifax, Nova Scotia
B3H 4R2

Dear Dr Marilyn MacKay-Lyons

I am preparing my Masters thesis for submission to the Faculty of Graduate Studies at Dalhousie University, Halifax, Nova Scotia, Canada. I am seeking your permission, as a co-author, to include a manuscript version of the following paper as a chapter in the thesis:

Measurement of spasticity after stroke using clinical measures: a systematic review. *Aloraini S, Gäverth J, Yeung E, MacKay-Lyons M.*

Canadian graduate theses are reproduced by the Library and Archives of Canada (formerly National Library of Canada) through a non-exclusive, world-wide license to reproduce, loan, distribute, or sell theses. I am also seeking your permission for the material described above to be reproduced and distributed by the LAC(NLC). Further details about the LAC(NLC) thesis program are available on the LAC(NLC) website (www.nlc-bnc.ca).

Full publication details and a copy of this permission letter will be included in the thesis.

Yours sincerely,

Saleh M. Aloraini

Permission is granted for:

a) the inclusion of the material described above in your thesis.

b) for the material described above to be included in the copy of your thesis that is sent to the Library and Archives of Canada (formerly National Library of Canada) for reproduction and distribution.

Name: Marilyn MacKay-Lyons Title: Professor

Signature: _____ Date: _____

APPENDIX B

Modified Ashworth Scale

Modified Ashworth Scale

Instructions:

1- The patient in a supine position and patient should be instructed to relax.
2- If testing a muscle that primarily flexes a joint, place the joint in a maximally flexed position and move to a position of maximal extension over one second.
3- If testing a muscle that primarily extends a joint, place the joint in a maximally extended position and move to a position of maximal flexion over one second.
4- Score based on the classification below

Scoring:

Score	Definition
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
2	More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved
3	Considerable increase in muscle tone, passive movement difficult
4	Affected part(s) rigid in flexion or extension

APPENDIX C

Tardieu Scale

Tardieu Scale

Instructions:

1- Grading should always be performed at the same time of day.
2- The patient is in a supine position with head in midline and patient should be instructed to relax.
3- When testing flexor muscle, place limb in maximal flexion position for the muscle and perform opposite movement when testing, and vice versa for extensor muscle.
4- Measurements take place at 3 velocities: V1, V2, and V3 (see table below).
5- Responses are recorded at each velocity as X/Y, with X indicating the 0 to 5 rating (see table below), and Y indicating the degree of angle at which the muscle reaction occurs, using a goniometer for detecting angle.

Velocities:

Velocity	Definition
V1	As slow as possible, slower than the natural drop of the limb segment under gravity
V2	Speed of limb segment falling under gravity
V3	As fast as possible, faster than the rate of the natural drop of the limb segment under gravity

Scoring:

Score	Definition
0	No resistance throughout the course of the passive movement
1	Slight resistance throughout the course of passive movement, no clear catch at a precise angle
2	Clear catch at a precise angle, interrupting the passive movement, followed by release
3	Fatigable clonus with less than 10 seconds when maintaining the pressure and appearing at the precise angle
4	Unfatigable clonus with more than 10 seconds when maintaining the pressure and appearing at a precise angle
5	Joint is immovable