Assessment of Upper Limb Spasticity after Stroke: Characterizing Resistance to Passive Stretch and the Impact on Active Control

by

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A thesis submitted in conformity with the requirements for the degree of Master of Science
Graduate Department of Rehabilitation Science
University of Toronto

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Abstract

Spasticity is a common phenomenon following stroke, characterized by involuntary resistance to passive stretch. Clinical assessments of spasticity are challenged as having poor sensitivity and do not describe the impact of spasticity on function. The purpose of this thesis was to objectively measure spasticity and establish the impact of spasticity on active, functional movement. Two studies were undertaken to A) assess kinematic variables that characterize spasticity during passive stretch and B) identify components of active, functional movement impacted by spasticity. Findings for Study 1 revealed that individuals with spasticity exhibit a measurable catch and release at fast velocities of stretch and increased resistance to passive movement at slow velocities. Findings for Study 2 revealed that stability during reaching and use of the more affected side were impacted by spasticity. This work provides a foundation for the development of a clinical assessment of spasticity that includes both passive and active components.
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List of Abbreviations

MAS: Modified Ashworth Scale

MTS: Modified Tardieu Scale

BTX-A: Botulinum Toxin-A

KINARM™: Kinesiological Instrument for Normal and Altered Reaching Movements
1.0 Introduction & Background

Rehabilitation after stroke aims to address any number of possible consequences with the hope of regaining function and independence. Debilitating impairments are unique to each individual and require timely treatment to improve the probability of recovery. Objective assessments aid in identifying potential targets for rehabilitation and allow for treatment to take place quickly while providing a focused approach to management or treatment of impairments. This thesis aimed to improve assessment of spasticity, specifically, and our understanding of upper limb function after stroke.

1.1 Canadian Stroke Statistics and Stroke Assessment

There are approximately 300,000 individuals living with the effects of stroke [1]. Increased incidence of key risk factors, including obesity and diabetes, is expected to contribute to a proliferation of cases of stroke. Improvements in care have led to the risk of death following stroke falling for the past 6 decades and a higher proportion of the population living with the effects of stroke and this places increased demand on community and health resources [1]. In 2000, the estimated annual health care cost and lost economic output due to premature mortality and long-term disability as a result of stroke was approximately $3.6 billion [1]. To decrease healthcare burden there is increased emphasis on recovery through rehabilitation. Recovery is optimized through objective assessments that precisely identify targets of therapy that can be individualized to the unique presentation of sensorimotor abnormalities caused by stroke. Accurate assessment of individual impairments and relative impact on active, functional movement allows therapy to be tailored to an individual's goals, whether they are general (i.e. increase strength) or specific to a task (i.e. opening a drawer to remove an item). Flexibility to adapt rehabilitation strategies to each individual relies on our capacity to objectively measure specific impairments and their impact on active, functional movement.

Recovery from stroke requires some form of rehabilitation in more than half of all stroke survivors [1]. Rehabilitation of the upper limb, historically, has been poor with only 57% of adults living with the effects of stroke achieving fair to good recovery of motor function after 4 years [2]. Compensation from the less affected arm results in motor re-organization with increased dependence on undamaged motor areas and ultimately less use of the affected side.
Rehabilitation of the upper limb must begin early after stroke, before compensation strategies develop. Simply examining the capacity to perform a task may mask the impact of impairments as individuals compensate to achieve the end goal. Measurement of the individual components of active, functional movement may unmask the unique consequences of specific impairments following stroke. Prompt identification of the specific targets of upper limb therapy is pivotal in counter-balancing compensation strategies and optimizing use and recovery of the affected side. One example of a sensorimotor impairment after stroke which is often masked by compensation and which is the focus of this thesis is spasticity. Subjective clinical assessment of spasticity and an insufficient examination of the impact of spasticity on active, functional movement have made it difficult to identify as an appropriate target for rehabilitation. Improved prognosis following stroke and increased chance for recovery of the upper limb may be facilitated through assessment of spasticity that includes both passive and active components.

1.2 Introduction to Spasticity

Spasticity occurs in 1 out of every 4 individuals following stroke [3]. The Lance definition is used extensively and describes spasticity as "a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" [4]. Lance's definition restricts our understanding of spasticity, neglecting sensory contributions and consequences to active, functional movement. A more progressive approach in defining spasticity was proposed by the Support Network for the Assembly of Database for Spasticity Measurement (SPASM) consortium: "disordered sensorimotor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained activation of muscle" [5]. Accordingly, spasticity includes a sensory and motor component with a focus on abnormal activation of muscle rather than hyperexcitability of the stretch reflex. Shifting the focus from the physiological cause of spasticity (i.e. hyperexcitability of the stretch reflex) to a sensorimotor control approach allows for simple measurement of spasticity during passive stretch and allows for an impact on active, functional movement to be established. For the purpose of this thesis, the definition proposed by the SPASM consortium was used.
Common assessment strategies for spasticity, discussed in section 1.3, involve the passive manipulation of a joint at multiple velocities followed by a grading of the muscle response (i.e. resistance to stretch). This technique provides a general overview of spasticity but may not be sensitive enough to identify varying levels of severity. Clinical signs are often termed 'catch' and 'release' referring to the involuntary activation of muscle and the behaviour that follows, respectively, and form the basis of spasticity assessments. Despite the common utilization of these terms their definitions are unclear. Bohannon and Smith, authors of a modified version of a more commonly used clinical scales for the assessment of spasticity, introduced 'catch' as "increase in muscle tone" but did not effectively define 'release' [6]. The first consideration for producing an objective measure of spasticity is to isolate and quantitatively define ‘catch’ and ‘release’.

Spasticity is associated with non-neurogenic resistance to passive stretch that includes contracture and soft-tissue changes to the joint. Contracture, defined as a change to connective tissue of muscles that may lead to reduction in range of motion, may contribute to resistance to passive stretch [7]. Similarly, soft-tissue changes or stiffness of the joint itself may play a role. Failure to isolate spasticity as neurogenic resistance to passive stretch is commonplace in spasticity assessment and can lead to exaggerated prevalence. Resistance to passive stretch is not exclusively dependent on hyperexcitability of the stretch reflex and may be associated with contracture [8]. As such, spasticity assessment should include a concurrent measure of passive range of motion. This is possible through slow velocities of stretch, below the reflex threshold where abnormal muscle activity characteristic of spasticity does not occur [9]. The second consideration for producing an objective measure of spasticity is ensuring that assessments differentiate spasticity from non-neurogenic resistance to passive stretch through inclusion of multiple velocities of movement.

Subjective clinical measures and unclear definitions of the clinical signs of spasticity make it difficult to establish the impact of spasticity on active, functional movement. Other impairments and secondary complications post-stroke may also impact active, functional movement and may confound the association if spasticity is not sufficiently isolated. There is noted disagreement and inconsistency when examining the impact of impairments on active, functional movement following stroke because the association is dependent on the accuracy and method of
impairment assessment [10]. This means that changes at the impairment level must be measureable to establish an impact on functional movement. The third consideration for producing an objective measure of spasticity is establishing the impact of spasticity on active, functional movement.

The aforementioned considerations align with the theoretical and methodological considerations for the assessment of spasticity proposed by the SPASM consortium [11]. They are 1) assessment must allow for variable velocities of displacement, 2) assessment must have both an active and passive component, 3) assessment must allow for a clearly defined protocol, and 4) assessment must have the capacity to be used in conjunction with EMG.

1.2.1 Impact of Spasticity on Active, Functional Movement

Subjective measurement of spasticity at the impairment level of the World Health Organization's International Classification of Functioning, Disability, and Health (ICF) [12] has made it difficult to draw conclusions on the impact of spasticity on active, functional movement. The two primary measurements at the impairment level, the Modified Ashworth Scale (MAS) and the Modified Tardieu Scale (MTS), do not consider active, functional movement during assessment. Nevertheless, these two scales are the primary means of measuring spasticity. Caution needs to be exercised when examining the impact of spasticity on active, functional movement when the MAS and MTS, measures with numerous concerns introduced in section 1.3 below, are used to quantify spasticity.

The existing literature to date examined the association between spasticity and function through observation of clinical changes following anti-spastic medications. Botulinum toxin-A (BTX-A) is commonly used and acts as a potent chemodenervating agent. At high doses, focal weakness may coincide with any improvements in spasticity. Therefore, it is possible that we may not see changes in function with reduction in spasticity if there is a coincidental decrease in strength. In a study examining different dosages of BTX-A, the highest dose did not coincide with improvements in function but at lower doses, function was able to improve without a significant decrease in strength [13].
There is substantial variation in the effect of BTX-A on function with some studies identifying positive change and others showing no change. A systematic review found a moderate improvement to function, although it identified greater benefit for passive rather than active function [14]. Welmer et. al. found a moderate association between spasticity reduction and scores on the Lindmark Motor Assessment Scale [15]. Other improvements have been observed in response to BTX-A, including improvements in basic tasks such as hand hygiene and dressing [16, 17], amount-of-use and quality-of-movement scores of the Motor Activity Log-30 (MAL) [18], Goal Attainment Scaling (GAS) [19], and subjective satisfaction [20]. On the other hand, reduction in spasticity has been shown to have no change on the Action Research Arm Test (ARAT) [16, 18], Box and Block Test (BBT) [18], or the Assessment of Quality of Life scale (AQoL) [19]. The substantial variation in the effect of BTX-A on function may be a product of individuals developing compensation strategies prior to treatment where, although there is an improvement in spasticity, function remains the same as they continue to compensate with the less affected side.

Several factors have been identified to explain the variation in observations of the impact of spasticity on active, functional movement. Potential causes of variation include chronicity of spasticity and timing of assessment, features of anti-spastic medication administration such as dose and site of delivery, outcome measures selected, and timing and degree of concurrent therapy [14]. Additionally, other impairments following stroke may receive more focus, such as improving strength and active range of motion, and are more likely to affect upper limb activity [10]. However, Francis identified several key factors that, if considered, will undoubtedly produce an association between a reduction in spasticity and increased function [13]. These factors are: include a measure of impairment and activity at multiple intervals, include outcomes that are relevant and acknowledge the goals of reducing spasticity (i.e. increased use of the limb, independence), and allow immediate access to rehabilitation services following anti-spastic medications to take advantage of reduced spasticity.
1.3 Evaluation of Spasticity

1.3.1 The Modified Ashworth Scale

The Ashworth Scale [21] manually grades muscle response to passive movement. A score of 0 is given for the absence of spasticity while a score of 4 is given to a rigid immovable limb. Individual muscle groups are assessed independently by passively stretching a limb through the entire range of motion and scores are assessed according to degree of resistance observed by the examiner. In an attempt to make the original Ashworth Scale more discrete, the Modified Ashworth Scale (MAS) [6] was developed by Bohannon and Smith. In both research and clinical settings, the MAS is a commonly used assessment of spasticity and has been considered the gold standard [3]. The MAS included a grade of ’1+’, effectively altering the scale to a 6-point ordinal measure that included scores 0, 1, 1+, 2, 3, and 4. A standardization procedure was developed; the muscle group being tested was stretched from maximal possible flexion to maximal possible extension in a period of one second by counting "one thousand and one" [6]. The original position of the individual being tested was supine on a bed.

The Modified Ashworth Scale has been extensively utilized in the literature and a summary of the validity and reliability can be found in Table 1. The MAS, although a valid measure of resistance to passive stretch, cannot distinguish between spasticity and biomechanical or soft-tissue changes [22] and caution is required when using the MAS as a measure of spasticity in isolation [23]. Therefore it is possible to attribute resistance to passive stretch, as measured by the MAS, to spasticity only 1-3 months post-stroke, prior to the commencement of non-neurogenic resistance to passive stretch [3]. Extensive evaluation of construct validity has rarely been performed [24]; the MAS may not be sufficiently sensitive to be a valid 6-point ordinal level measure of spasticity but may be more appropriate as a less sensitive measurement of resistance to passive stretch [25]. Using the MAS, then, can only provide a summary of the resistance to passive stretch without indicating relative contributions of spasticity and other related constructs (i.e. non-neurogenic resistance). A single study found excellent correlation between the MAS and several different approaches to measuring spastic hypertonia (i.e. EMG, H-reflex) but had a small sample size and all participants were in the chronic stage [26].
Bohannon and Smith found high reliability when they developed the MAS [6]. A review of the literature [24] found that, although high intra- and inter-rater reliability can be achieved [27, 28], this is not always the case and has been observed as very good [29] and poor to moderate [30]. The authors of the review identified a paucity of information regarding the cause of the variable reliability, citing "assessed joint" and "examiner's qualification" as possibilities. Variability of reliability estimates may be caused by poor standardization. As spasticity is velocity-dependent, velocity needs to be controlled during assessments. However, it may not be possible to control in a clinical setting, inevitably decreasing reliability [25]. The MAS may not extend further than a subjective measure of resistance to passive stretch until standardized positioning and instructions are developed. Further, standardized identification of the 'catch' and 'release' are necessary for reliability in estimating the degree of resistance.

<table>
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<th>Validity</th>
<th>Reliability</th>
<th>Standardization</th>
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Table 1: Psychometric Properties of the Modified Ashworth Scale for Post-Stroke Spasticity Assessment.

### 1.3.2 The Modified Tardieu Scale

The Modified Tardieu Scale (MTS) [31] is commonly used as an alternative to the MAS. Versions of the MTS have been employed since the 1950’s in early attempts to measure what would later be known as spasticity [32]. The final version of the MTS, developed by Jean-Michel Gracies in 2000, included three velocities of movement [31]. These velocities are standardized as: "as slow as possible", "speed of the limb segment falling under gravity", and
"as fast as possible (faster than the rate of the natural drop of the limb segment under gravity)". Through these methods, passive range of motion of the limb is assessed at the slowest velocity and the angle and degree of muscle resistance is graded at the two faster velocities. The addition of a measure of passive range of motion allows the MTS to differentiate spasticity and contracture and is therefore considered a more appropriate measure of spasticity [22].

A summary of the validity and reliability of the MTS can be found in Table 2. As a relatively new measure of spasticity, there is currently no literature assessing the content validity and no conclusion can be drawn [33]. However, one study examined convergent validity of the MTS with stretch-induced EMG and passive range of motion and found excellent correlation between the three measures [22].

As with validity data, there is a paucity of available literature examining the reliability of the MTS for the assessment of upper limb post-stroke spasticity. The available data, similar to the MAS, produces variable results [24]. One study examined individuals with severe brain injuries, including stroke, and found moderate to very good test-retest reliability for the upper limb, depending on the joint being tested [30]. The same study found poor inter-rater reliability for all joints, but, with the exception of wrist extensors, results were more favorable than the MAS. The type of tool used to determine the angle of resistance or passive range will directly influence reliability. A single study examined the use of a goniometer and found excellent test-retest reliability and fair to good inter-rater reliability [34]. More effective was the use of inertial sensors with excellent test-retest and inter-rater reliability, but this may not be a practical tool for clinical use.
Table 2: *Psychometric Properties of the Modified Tardieu Scale for Post-Stroke Spasticity Assessment.*

### 1.4 Thesis Rationale

Two primary targets of post-stroke assessment are highlighted in Figure 1: measuring changes to sensorimotor impairments and measuring changes to activity. Assessment at each of these targets allows recovery to be monitored through neurophysiological or biomechanical measures (the impairment level) and through changes in performance of active, functional movement (the activity level). The capacity to independently measure each individual impairment facilitates understanding of their role in overall disability and function [35]. This is especially true for the upper limb, where measurement needs to detect small changes as recovery of function occurs slower and to a lesser extent than the lower limb [36].

The key changes at the sensorimotor impairment level are to strength, dexterity, proprioception, and spasticity. Basic clinical tools are available and recommended for use in the measurement of strength and dexterity [37], and reliable measures of proprioception are emerging with the advancement of position sensors [38, 39]. However, an objective measure of spasticity, as it is defined, has remained elusive. Spasticity occurs as intermittent or sustained activation of muscle in response to passive movement and the response is influenced by velocity of passive stretch. Assessment of spasticity at the impairment level, then, requires observation of passive stretch at...
multiple velocities. The 2nd chapter of this thesis examines a novel approach to the assessment of spasticity during passive movement.

Components of active, functional movement are identified at the right side of Figure 1. Each component represents a general class of parameters (i.e. movement speed encompasses maximum speed, average speed, speed maxima count) during active movement that may be influenced by spasticity. These 5 general classes are related to the sensorimotor control definition of spasticity where intermittent or sustained activation of muscle and sensitivity to stretch may influence each component. Available literature does not examine the impact of spasticity on these underlying features of active, functional movement.

![Conceptual Model of Post-Stroke Assessment of Upper Limb Motor Control](image)

Figure 1: Conceptual Model of Post-Stroke Assessment of Upper Limb Motor Control.

Traditional clinical measurements of spasticity are often challenged as having poor sensitivity and do not describe the impact of spasticity on active, functional movement. The KINARM™ robotic exoskeleton for the upper limb (BKIN Technologies Ltd., Kingston, ON, Canada) [40] was used to measure resistance to passive stretch of the elbow flexors and extensors and to
establish the impact of spasticity on active, functional movement. Explained in more detail in subsequent chapters, the KINARM™ is a measurement tool that records kinematics during passive and active tasks. The KINARM™ was chosen as the primary assessment tool because it provided an accurate measure of joint kinematics during passive stretch and there was sufficient specificity in the tasks that are available for use with the KINARM™ that capture the components of active, functional movement presented in Figure 1.

Measuring spasticity at the impairment level and establishing the impact of spasticity on active, functional movement are paramount to optimizing overall rehabilitation goals. Quantitative, objective metrics characterizing the presence and extent of spasticity and its impact on active, functional movement establish a starting point upon which the determination of efficacy of rehabilitation can be based. These metrics may provide a practically viable alternative to the current clinical practice of hand-held goniometer and a therapist's perception of the extent and location of resistance to passive stretch. Resistance to passive stretch encompasses several similar components (i.e. neurogenic resistance, or spasticity, and non-neurogenic resistance) with drastically different treatment goals requiring effective measurement and isolation of each component. Treatment targets and determining treatment efficacy are dependent on objective measurement.

Stroke survivors report 'the use of the arm in everyday tasks' as the most important factor associated with upper limb recovery and 'not enough movement to work with' represents the greatest obstacle [41]. As spasticity may limit movement, recovery may be hindered; this thesis aims to improve our understanding of spasticity with clinical implications of identifying spasticity as a potential target for rehabilitation. This work improves upon traditional assessments with the inclusion of a highly standardized protocol with multiple velocities of passive movement and an active component. Spasticity may be better understood and managed when defined through a standardized measurement and evaluated during passive and active movement, facilitating maximal recovery.
1.5 Thesis Purpose

The purpose of this thesis was to objectively measure spasticity as a key component of resistance to passive stretch and establish the impact of spasticity on active, functional movement. This work provides a foundation for the development of a clinical assessment of spasticity that includes both passive and active components. This was accomplished through two studies presented in Chapter 2 and Chapter 3. The purpose of Chapter 2 was to assess kinematic variables that may characterize spasticity during passive stretch of the elbow flexors and extensors in individuals with upper limb spasticity ranging from no spasticity to moderate spasticity as measured by the MAS and the MTS. The purpose of Chapter 3 was to identify components of active, functional movement impacted by spasticity. Changes to active, functional movement are relevant to the individual and may provide a useful metric for assessing the impact of anti-spastic medications.
2.0 Objectively Measuring Spasticity as a Key Component of Resistance to Passive Stretch at the Elbow Following Stroke

2.1 Abstract

Involuntary resistance to passive stretch contributes to abnormal function after stroke and is caused by numerous clinical symptoms, including spasticity. Assessment requires independent observation and isolation of individual components of resistance to passive stretch. However, common clinical scales that assess spasticity may not effectively differentiate spasticity from other components (i.e. non-neurogenic) of resistance to passive movement. The purpose of this study was to objectively measure spasticity as a key component of resistance to passive stretch. This was accomplished through the assessment of kinematic variables that may characterize spasticity during passive stretch of the elbow flexors and extensors (delivered by the KINARM™ robotic exoskeleton) in stroke survivors with upper limb spasticity. The findings for this study revealed that kinematic variables observed with resistance to passive elbow flexor and extensor stretch uniquely characterized spasticity and differentiated between individuals with and without spasticity. Individuals diagnosed with spasticity, as measured by the Modified Ashworth Scale, exhibited a characteristic ‘catch’ and ‘release’ during passive stretch over an 80° range with a movement cycle of 1000ms. The catch and release appeared earlier when compared to stroke survivors without spasticity and healthy controls. Further, decreased passive range of motion (end angle) and earlier signs of resistance to passive movement (tone angle) during passive stretch over an 80° range with a movement cycle of 1500ms were observed in individuals diagnosed with spasticity as compared to stroke survivors without spasticity and healthy controls. These results indicate that this novel approach to generating passive elbow movement identifies metrics of spasticity that may contribute to the development of a more sensitive clinical assessment of spasticity in future studies.
2.2 Introduction

Neurorehabilitation programs are informed by accurate assessment of changes in impairments of individuals following a stroke. The metrics obtained from these assessments permit links to be established between specific impairments and associated function. To illustrate, the nine hole peg test and the box and block test have been used to show improvements in fine and gross manual dexterity after a piano rehabilitation program [42]. In another example, multiple methods of assessing weakness have been used to show improvements in strength after a strength training rehabilitation program that had a direct influence on function [43]. In contrast, there is a lack of an objective measure of spasticity, which is present in 20-25% of individuals within twelve months of first-ever stroke [3]. Traditional clinical scales may not accurately reflect improvements in response to treatment, limiting our understanding of the link between spasticity and function.

Spasticity has been recently redefined as "disordered sensorimotor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained activation of muscles" [5]. Importantly, this definition does not consider non-neurogenic components of resistance to passive movement unrelated to velocity-sensitive muscle activation, such as contracture or soft-tissue changes. Spasticity can be overestimated if non-neurogenic components of resistance to passive movement are included in the assessment [3]. The most commonly used clinical measure of spasticity, the Modified Ashworth Scale (MAS) [6], may be confounded by non-neurogenic resistance (i.e. contracture) [22]. An alternative to the MAS, the Modified Tardieu Scale (MTS) [31], differentiates spasticity from increased resistance caused by contracture [22] but does not quantify increased resistance caused by alterations in the viscoelastic properties of the joint. It has been suggested that resistance to passive movement and range of passive movement are influenced by the level of activity in the alpha motor neuron of agonist and antagonist muscle groups and by the viscoelastic properties of the soft tissues and joints themselves [23].

Sensitivity to velocity of stretch differentiates spasticity from non-neurogenic components of resistance to passive movement. The MAS and the MTS may not appropriately differentiate spasticity from other components of resistance to passive movement and therefore may not be
appropriate for use as a standalone measure of spasticity. While the MAS is used broadly, there is an implicit assumption that any changes in resistance to passive movement is exclusively a result of spasticity [23]. It has been shown to be invalid as a 6-point ordinal level measure [25] and grades on the lower end of the scale (1, 1+, 2) may not have the discriminative capacity to distinguish between different levels of spasticity [24]. The MTS, in contrast, has been shown to differentiate spasticity from contracture [22] but no conclusions can be drawn on the validity of the measure [33]. Both scales use terminology in grading, such as 'catch' and 'release', without clear definitions which adds subjectivity to the rating. Improvements in assessment can be achieved through defining 'catch' and 'release' in objective terms [23].

Prior work has described approaches to measuring the different components of elevated resistance to passive stretch. Lindberg and colleagues [44] proposed a model for capturing all of the components of resistance to passive stretch, collectively known as hypertonicity, in a single measure. The authors estimated the force contributions of muscle, passive elasticity, muscle viscosity, and inertial properties of the wrist joint to resistance to passive stretch and concluded that their model may be capable of separating the neural component of resistance from resistance caused by non-neurogenic properties of the muscle and joint. This approach, which focuses on kinetics, can potentially be built upon with the inclusion of kinematics. Classifying the key components of resistance to passive stretch in terms of kinematics provides information that is easily replicated in a clinical setting (i.e. using an accelerometer). Kinematics have been used to assess upper limb spasticity during active, functional movement [18, 45] but there have been no studies that use kinematics to sufficiently examine upper limb spasticity during passive stretch at multiple velocities. Pandyan and colleagues [25, 46] successfully developed a prototype system to quantify resistance to passive stretch that identified a 'catch' as a transient increase in force that opposed passive movement. A trained therapist was responsible for passively moving the participant's limb, closely mimicking the MAS. It may be possible to build on this work through the inclusion of multiple velocities of movement, kinematic analysis, and a standardized point of application of the force with respect to the elbow joint.

The purpose of the present study was to objectively measure spasticity as a key component of resistance to passive stretch of the elbow flexors and extensors. This work provides a foundation for the development of a clinical measure of spasticity that is sensitive to change. The primary
objective was to assess kinematic variables that may characterize spasticity during passive elbow flexion and extension across an 80° range occurring over different movement cycles (times) to differentiate between individuals with and without spasticity as determined by clinical scales. It was expected that kinematic variables observed with resistance to passive elbow flexor and extensor stretch would uniquely characterize spasticity and differentiate individuals with and without spasticity. The secondary objective was to identify the sensitivity of each measure to velocity of stretch. It was expected that passive range of motion (end angle) and the earliest sign of resistance to passive stretch (tone angle) would not be affected by velocity while the characteristic catch and release would appear sooner with decreased duration of movement (i.e. increased velocity).

2.3 Methods

2.3.1 Participants

Individuals with stroke (n=42) were recruited to participate in this study from the Toronto Rehabilitation Institute (TRI) and Sunnybrook Health Sciences Centre (SHSC). Participants were excluded (n=8) from analysis if they were within the effective period (3 months) of spasticity management (Botulinum toxin-A) (n=4), unable to follow instructions (n=2), unable to complete the study due to equipment malfunction (n=1), or participated during pilot testing only (n=1). The remaining 34 individuals were attending inpatient (n=9) or outpatient (n=25) services at either of the two centres. Healthy controls (n=12) also participated in the study through a convenience sample from TRI and Queen’s University. All patient research and 33% of healthy control research (n=4) was conducted at TRI and ethical clearance was granted from the TRI Research Ethics Board and the University of Toronto Research Ethics Board. Additional healthy control research (n=8) was conducted at Queen’s University and was approved by the Queen’s University Ethics Research Board. All individuals provided written informed consent.

Adults attending either inpatient or outpatient services for stroke rehabilitation were eligible to participate in this study. Of the 34 eligible participants with complete data, 33 had experienced a 1st-ever stroke while a single participant had multiple strokes. It has been noted that previous
stroke is not a risk factor for the development of spasticity [35] and the latter individual was included in analysis. The 34 eligible participants were split into two groups representing those with spasticity [SPAS] and those without spasticity [NO SPAS] as determined by the MAS. Although the MAS may not be sensitive enough to detect differences in severity of spasticity, it has been shown to provide a general summary of resistance to passive movement and was used to group participants. An additional control group of healthy individuals, [HEALTHY] was also assessed. Demographics, clinical scores, and stroke information for each group can be found in Table 3.

<table>
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<th>Stroke</th>
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<td>HEALTHY (n = 12)</td>
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<td>9:3</td>
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<td>171 (160-183)</td>
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<td>12:0</td>
</tr>
<tr>
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<td>5:7</td>
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<td>7:5</td>
<td></td>
</tr>
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<td>Time Since Stroke (m)</td>
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<td>7 (1-43)</td>
<td></td>
</tr>
<tr>
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<td>2.5 (2-6)</td>
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<td>'0' = 12</td>
<td></td>
</tr>
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</table>

Table 3: Demographic Information for Study Participants in the SPAS, NO SPAS, and HEALTHY Groups (Chapter 2). Unless specified, all values are presented as mean (range).
2.3.2 Experimental Setup

Passive stretch of the upper limb was delivered using the KINARM™ robotic exoskeleton for the upper limb (BKin Technologies Ltd., Kingston, ON, Canada) [40] (Figure 2). This device has been used previously in research examining upper limb sensorimotor function in the stroke population [38, 47, 48]. Passive movement was controlled using a real-time computer and Dexterit-E™ (versions 3.2.3 to 3.2.5) data acquisition software. Hand trajectories were relayed back to the examiner and provided precise and reliable feedback during data collection.

Figure 2: KINARM™ Robotic Exoskeleton for the Upper Limb (Without Display Unit). Note the full weight support for the upper limbs, wheel-chair designed seating, and fully customizable exoskeleton links to match the body shape and size of each participant.

Participants were seated in the wheel-chair base of the KINARM™. Seat height was adjusted so that the bottom of the arm troughs, which provide full weight support for the limbs, were aligned to shoulder height. The position of the exoskeleton was adjusted to align the vertical axis of the shoulder while in approximately 90° shoulder abduction. The experimental set up permits shoulder and elbow movement to occur in the horizontal plane about the joint centre. During testing, the shoulder angle was stabilized at a position of 120° (Figure 3) so that movement could only occur at the elbow.
### 2.3.3 Task Description

During the passive stretch task, the KINARM™ was programmed to move the limb segment below the elbow joint through an 80° range over five different movement durations: 1500ms, 1200ms, 1000ms, 800ms, and 600ms. For the patient groups [SPAS and NO SPAS], the more affected arm was tested first, followed by the less affected arm. For the [HEALTHY] group the non-dominant arm was tested first followed by the dominant arm. Each participant was given the following instructions:

"In this task, the robot is going to move your arm. We are going to start with the [more affected] side first and then do the opposite side. All you need to do is relax as much as possible. The robot will move your arm from one position [show approximate extension position] to another [show approximate flexion position] at different speeds. If at any time the robot moves too quickly I will stop the task. You will relax as much as possible for the duration of the task. Do not try to actively move your arm. You may close your eyes if that helps you to relax but you may not speak. Any questions? Are you ready? The trial will begin, please relax while the robot moves your [active arm]."

In the horizontal plane, the elbow was extended to the starting position of 145° where 180° represents full extension. The servomotors changed the elbow angle from 145° to 65° (flexion movement) and from 65° to 145° (extension movement) separated by a 3000ms interval, completing one full trial (Figure 3). A total of 3 trials at 5 durations of movement were completed. The task was intended to drive the arm to the specified end angle in a specific duration, indirectly affecting the velocity of movement. The position controller of the KINARM™ uses joint kinematics, robot inertias, subject weight, and the position control set point to calculate the motor torques required to reach the end angle in the specified duration. Figure 4 illustrates the desired trajectory over a 1000ms period.
Figure 3: Passive Task Protocol. Each trial began in extension (145°). The arm was passively driven to the flexion position (65°), there was a 3000ms wait, and finally driven back to the extension position. This represented a single trial.

Figure 4: Modeled Passive Movement Characteristics. Movement is delivered by the KINARM™ during a flexion trial at 1000ms. The position controller attempts to minimize the error between desired force and actual force. Force output is proportional to the error where a larger error results in greater force production within a safe range.

A 1000ms duration was selected to mimic the standardization of the Modified Ashworth Scale where the assessor manually moves the affected arm through full passive range at the elbow in 1000ms by counting 'one-thousand-and-one' [6]. A systematic review of the MAS and MTS showed variable intra- and inter-rater reliability [24], possibly due to poor standardization, and
as such, 4 other durations were included in this study. In addition, the specific angles of 145° and 65° were selected to allow for behaviour that is characteristic of spasticity to be observed within a safe range.

In instances where 145° or 65° exceeded the passive range of the participant, the KINARM™ position controller continued to drive the motors to produce torques up to a safety limit of 15Nm in an attempt to reach the end angle. The trial ended when the target angle was reached or after a maximum of 6000ms had elapsed. Thus, a total of 9000ms was possible between movements (6000ms + 3000ms wait time between trials). In these instances, the new 'start angle' was set as the current position.

2.3.4 Outcome Measures

Kinematics, including position, velocity, and acceleration at the elbow were recorded at 1 kHz. Data was analyzed for 2500ms from the beginning of a flexion or extension movement. End angle was calculated as the final elbow angle at 2500ms. Catch angle was calculated as the angle of the 1st velocity minimum after the maximum velocity. This point was selected as a simple, repeatable metric that would be present in all participants when resistance occurs. If the 1st velocity minimum was negative, the angle at which velocity reached 0°/s was recorded as the catch angle. The range of release was measured as the difference in catch angle and end angle with a positive value indicating a release. The earliest sign of resistance to passive movement, defined in this study as the tone angle, was measured as the angle at which the trajectory falls outside of 3 standard deviations of the mean trajectory for the HEALTHY group. 3 standard deviations was chosen as the benchmark as preliminary analysis suggested that the healthy control trajectory variability was relatively low. If the trajectory of an individual does not fall outside of 3 standard deviations of the mean healthy trajectory, a value equal to the minimum angle at which a difference could occur was assigned. Each measure is represented in Figure 5.
Figure 5: Outcome Measures Characterizing Resistance to Passive Movement. End angle (A), catch angle (B), and range of release (C, difference between A and B) are identified for sample participants in the SPAS group in blue and the NO SPAS group in red (left panel). The tone angle (D) is identified as the angle at which the average trajectory exceeds 3 SD of the HEALTHY group (where the dashed green line represents ± 3 SD and the solid green line represents the mean of the HEALTHY group trajectory (right panel).

The Modified Ashworth Scale (MAS) and the Modified Tardieu Scale (MTS) were measured at the elbow for both flexors and extensors on the more affected side. For the duration of the study, a total of three assessors were used; two trained therapists and one study investigator, all trained in administering these scales. Each participant was only assessed by one assessor. All attempts were made to minimize the time-of-day effects that lead to variability of spasticity and the stretch reflex [49]. This means that MAS and MTS measurements were taken immediately before or immediately after the KINARM™ assessment.

2.3.5 Statistical Analysis

Descriptive statistics were used to characterize the study groups. A one-way ANOVA was used to test the hypothesis that end angle, catch angle, and range of release would distinguish individuals in the SPAS group from those in the NO SPAS and HEALTHY groups. In cases where the assumption of normality was violated, as assessed by the Shapiro-Wilk Test, a non-parametric Kruskal-Wallis Test was used. In cases where the data was normally distributed but
violated the assumption of homogeneity of variance as measured by Levene's Test, Welch's statistic was reported. An independent t-test was used to test the hypothesis that tone angle would distinguish between the SPAS and NO SPAS groups. In cases where the assumption of normality was violated a non-parametric Mann-Whitney U Test was used. In cases where the data was normally distributed but violated the assumption of homogeneity of variance the t-statistic for the "equal variances not assumed" condition was used.

Repeated measures ANOVA were used to probe the velocity-dependent sensitivity of end angle, catch angle, range of release, and tone angle within the SPAS group. In cases where the assumption of normality was violated a non-parametric Friedman Test was used. In cases where the assumption of sphericity was violated as assessed by Mauchly's Test, a Greenhouse-Geisser correction was applied. Statistical significance was set at $p < 0.05$. Statistical analysis was performed using SPSS v22.0 for Windows.

### 2.4 Results

Elbow flexors and elbow extensors were evaluated separately. The mean of three trials at each duration of movement was used in statistical calculations and reported as (mean ± standard error of the mean). Approximately 41% ($n=9$) of the SPAS group had spasticity in both the flexors and extensors as measured by a MAS score $\geq 1$ and were included in analysis for both muscle groups. Approximately 41% ($n=9$) of the SPAS group had spasticity in the flexors only and were included in the flexors analysis while 18% ($n=4$) of the SPAS group had spasticity in the extensors only and were included in the extensors analysis only. A total of 100% ($n=12$) of the NO SPAS group and 100% ($n=12$) of the HEALTHY group were included. Outliers were included in statistical analyses.

Between-group analysis evaluated end angle, catch angle and range of release, and tone angle. Passive range of motion should be assessed using very slow stretch to remain below the velocity threshold of the stretch reflex [50]. In the present study end angle was only analyzed during the slowest desired stretch duration of 1500ms to minimize the likelihood of initiating the stretch reflex. Catch angle and range of release were analyzed at 1000ms to closely mimic the standardization of the MAS where the assessor attempts to move the limb through the full range
in one second by counting "one thousand and one" [6]. Again, to minimize the likelihood of initiating the stretch reflex, tone angle was analyzed at 1500 ms. This was done to determine the first sign of resistance to passive movement independent of spasticity in both patient groups.

Within-group analysis evaluated end angle, catch and range of release, and tone angle of the SPAS group to identify the sensitivity of each measure to velocity of passive stretch. 600 ms, 1000 ms, and 1500 ms were included to mimic the MTS where the assessor attempts to move the limb through the full range at three speeds: "slow", "speed of gravity", and "faster than the speed of gravity" [31]. Additional durations of 800 ms and 1200 ms were included to improve sensitivity. Only the SPAS group was analyzed as the NO SPAS group, as assessed by the MAS, did not exhibit clinical velocity-sensitive resistance to passive stretch (i.e. spasticity).

2.4.1 Between-Group Analysis

There was a statistically significant difference of end angle during passive elbow extension between groups, \((H(2) = 24.18, \ p < 0.001)\). Pairwise comparisons revealed a statistically significant difference between the SPAS group and the HEALTHY group \((134.71 \pm 1.73^\circ \text{ and } 142.73 \pm 0.16^\circ, \text{ respectively, } p < 0.001)\) and between the SPAS group and the NO SPAS group \((141.03 \pm 0.52^\circ, \ p = 0.030)\), but not between the NO SPAS group and the HEALTHY group \((p = 0.108)\).

There was a statistically significant difference of end angle during passive elbow flexion between groups, \((H(2) = 21.04, \ p < 0.001)\). Pairwise comparisons using Dunn's procedure with a Bonferroni correction revealed a statistically significant difference between the SPAS group and the HEALTHY group \((77.11 \pm 2.67^\circ \text{ and } 66.97 \pm 0.53^\circ, \text{ respectively, } p < 0.001)\) and between the SPAS group and the NO SPAS group \((70.00 \pm 0.79^\circ, \ p = 0.046)\), but not between the HEALTHY group and the NO SPAS group \((p = 0.104)\). Means and standard errors of the mean for each muscle group are represented in Figure 6.
Figure 6: *Mean End Angle during 1500ms Trials across All Groups*. End angle is reported for A) passive elbow extension and B) passive elbow flexion. Error bars represent ± 1 SEM. Horizontal dotted line represents desired end angle of 145° and 65° for passive elbow extension and passive elbow flexion, respectively. Asterisks indicate statistically significant differences (p < 0.05).

There was a statistically significant difference of catch angle during passive elbow extension between groups, (H(2) = 29.66, p < 0.001). Pairwise comparisons revealed a statistically significant difference between the SPAS group and the HEALTHY group (127.70 ± 3.02° and 143.78 ± 0.30°, respectively, p < 0.001) and between the SPAS group and the NO SPAS group (141.93 ± 0.70°, p = 0.001), but not between the NO SPAS group and the HEALTHY group (p = 0.403).

There was a statistically significant difference of catch angle during passive elbow flexion between groups, (Welch's F(2, 20.449) = 16.40, p < 0.001). Games-Howell post-hoc test revealed a statistically significant decrease of 15.17° (95% CI, 7.50 to 22.84°, p < 0.001) between the SPAS group (80.86 ± 2.83°) and the HEALTHY group (65.69 ± 0.75°), a statistically significant decrease of 10.80° (95% CI, 2.97 to 18.63°, p = 0.007) between the SPAS group and the NO SPAS group (70.06 ± 1.06°), and a statistically significant decrease of 4.37° (95% CI, 1.08 to 7.66°, p = 0.008) between the NO SPAS group and the HEALTHY group. Means and standard errors of the mean of catch angle for each muscle group are represented in Figure 7 along with range of release, reported below.
There was a statistically significant difference of range of release during passive elbow extension between groups, \((H(2) = 27.47, p < 0.001)\). Pairwise comparisons revealed a statistically significant difference between the SPAS group and the HEALTHY group \((7.55 \pm 2.19^\circ \text{ and } -0.74 \pm 0.16^\circ, \text{ respectively, } p < 0.001)\) and between the SPAS group and the NO SPAS group \((-0.52 \pm 0.32^\circ, p < 0.001)\) but not between the NO SPAS group and the HEALTHY group \((p = 1.000)\).

There was a statistically significant difference of range of release during passive elbow flexion between groups, \((H(2) = 20.51, p < 0.001)\). Pairwise comparisons revealed a statistically significant difference between the SPAS group and the HEALTHY group \((4.64 \pm 0.84^\circ \text{ and } -0.71 \pm 0.15^\circ, \text{ respectively, } p < 0.001)\) and between the SPAS group and the NO SPAS group \((-0.20 \pm 0.64^\circ, p = 0.001)\) but not between the NO SPAS group and the HEALTHY group \((p = 1.000)\).
Figure 7: Mean Catch Angle (Left) and Range of Release (Right) during 1000ms Trials across All Groups. Catch angle and range of release are reported for A) passive elbow extension and B) passive elbow flexion. Error bars represent ± 1 SEM. Horizontal dotted line represents desired end angle of 145° and 65° for passive elbow extension and passive elbow flexion, respectively. Asterisks indicate statistically significant differences (p < 0.05).

There was a statistically significant difference of tone angle during passive elbow extension between the SPAS group (108.99 ± 4.31°) and the NO SPAS group (130.88 ± 4.26°), (U = 31.50, z = -3.25, p = 0.001). Similarly, there was a statistically significant difference of tone angle during passive elbow flexion between the SPAS group (97.10 ± 5.44°) and the NO SPAS group (84.01 ± 4.60°), (U = 42.00, z = -1.99, p = 0.047). Means and standard errors of the mean for each muscle group are represented in Figure 8.
2.4.2 Within-Group Analysis of Duration of Movement

There were no significant effects of movement duration on end angle during passive elbow extension in the SPAS group, ($X^2(4) = 8.37, p = 0.079$). Means and standard errors of the mean were 136.04 ± 1.55° (600ms), 135.44 ± 1.59° (800ms), 135.24 ± 1.49° (1000ms), 135.06 ± 1.64° (1200ms), and 134.71 ± 1.73° (1500ms). Similarly, there were no significant effects of duration on end angle during passive elbow flexion in the SPAS group, ($X^2(4) = 0.43, p = 0.980$) with means and standard errors of the mean of 76.51 ± 2.39° (600ms), 76.58 ± 2.39° (800ms), 76.22 ± 2.41° (1000ms), 77.05 ± 2.68° (1200ms), and 77.11 ± 2.67° (1500ms).

There were no significant effects of duration on catch angle during passive elbow extension in the SPAS group, ($X^2(4) = 3.20, p = 0.525$). Means and standard errors of the mean were 128.78 ± 2.13° (600ms), 128.00 ± 2.78° (800ms), 127.70 ± 3.02° (1000ms), 128.15 ± 3.15° (1200ms), and 124.21 ± 3.61° (1500ms). However, a statistically significant effect of duration on catch angle during passive elbow flexion was revealed, ($X^2(4) = 12.49, p = 0.014$). Pairwise comparisons with a Bonferroni correction revealed a statistically significant difference between the 800ms duration and the 1500ms duration (82.85 ± 2.79° and 79.20 ± 2.92°, respectively, $p = 0.019$). The difference between the 600ms duration (83.32 ± 2.29°) and the 1500ms duration...
approached significance \( (p = 0.064) \). Means and standard errors of the mean of catch angle for each muscle group are represented in Figure 9 along with range of release, reported below.

There were no significant effects of duration on range of release during passive elbow extension in the SPAS group, \( (X^2(4) = 5.56, p = 0.235) \). Means and standard errors of the mean were 7.26 ± 1.27° (600ms), 7.44 ± 1.95° (800ms), 7.55 ± 2.19° (1000ms), 6.91 ± 2.33° (1200ms), and 10.50 ± 3.16° (1500ms). However, a statistically significant effect of duration on range of release during passive elbow flexion was revealed, \( (X^2(4) = 30.07, p < 0.001) \). Pairwise comparisons with a Bonferroni correction revealed a statistically significant difference between the 600ms duration and the 1200ms duration (6.81 ± 0.94° and 2.90 ± 0.76°, respectively, \( p = 0.043 \)), the 600ms duration and the 1500ms duration (2.09 ± 0.51°, \( p = 0.001 \)), the 800ms duration (6.27 ± 1.06°) and the 1200ms duration (\( p = 0.004 \)), and the 800ms duration and the 1500ms duration (\( p < 0.001 \)). The difference between the 1000ms duration (4.64 ± 0.84°) and the 1500ms duration approached significance \( (p = 0.077) \).
Figure 9: Mean Catch Angle (Left) and Range of Release (Right) across All Durations of Movement for the SPAS Group. Catch angle and range of release are reported for A) passive elbow extension and B) passive elbow flexion. Error bars represent ± 1 SEM. Horizontal dotted line represents desired end angle of 145° and 65° for passive elbow extension and passive elbow flexion, respectively. Asterisks indicate statistically significant differences (p < 0.05).

A statistically significant effect of duration on tone angle during passive elbow extension was revealed, (X²(4) = 28.13, p < 0.001). Pairwise comparisons with a Bonferroni correction revealed a statistically significant difference between the 600ms duration and the 1000ms duration (118.49 ± 2.87° and 109.57 ± 3.36°, respectively, p = 0.016), the 600ms duration and the 1200ms duration (106.96 ± 3.65°, p < 0.001), the 600ms duration and the 1500ms duration (108.99 ± 4.31°, p < 0.001), and the 800ms duration (112.98 ± 2.80°) and the 1200ms duration...
Similarly, a statistically significant effect of duration on tone angle during passive elbow flexion was revealed, \((X^2(4) = 18.15, p = 0.001)\). Pairwise comparisons with a Bonferroni correction revealed a statistically significant difference between the 600ms duration and the 1200ms duration \((86.12 \pm 3.11^\circ \text{ and } 98.05 \pm 4.29^\circ, \text{ respectively, } p = 0.001)\), and between the 800ms duration \((90.75 \pm 3.71^\circ)\) and the 1200ms duration \((p = 0.043)\). The difference between the 600ms duration and the 1500ms duration \((97.10 \pm 5.44^\circ)\) approached significance \((p = 0.064)\). Means and standard errors of the mean of tone angle for each muscle group are represented in Figure 10.

![Figure 10: Mean Tone Angle across All Durations of Movement for the SPAS Group. Tone angle is reported for A) passive elbow extension and B) passive elbow flexion. Error bars represent \(\pm 1\) SEM. Asterisks indicate statistically significant differences \((p < 0.05)\).](image)

2.5 Discussion

The purpose of the present study was to objectively measure spasticity as a key component of resistance to passive stretch. The primary objective was to assess kinematic variables, namely end angle, catch angle, range of release, and tone angle, that may characterize spasticity during passive stretch over multiple movement cycles (times) which indirectly impacts velocity of movement. The secondary objective was to identify the sensitivity of each measure to velocity of movement. Kinematics were recorded during passive elbow flexion and extension covering
an 80° range and outcome measures were identified through evaluating elbow angular velocity and position throughout the trials.

2.5.1 End Angle

End angle during 1500ms trials differentiated the SPAS group from the NO SPAS group and the HEALTHY group during both passive elbow flexion and extension. This finding suggests that the presence of elbow spasticity following stroke may be associated with decreased passive elbow range. Previous reports have identified a relationship between elevated resistance to passive stretch and reduced range. As examples, Ada and colleagues [51] observed a causal effect of spasticity on contracture while O'Dwyer and colleagues [8] found a general resistance to passive stretch to be associated with decreased passive range. The findings of the present study support this established relationship between spasticity and reduced passive range. In contrast, Malhotra and colleagues [52] identified loss of function and not spasticity as the primary contributor to contracture formation. Though the present study cannot determine whether contracture per se was present in the study participants, the reduction in passive range could indicate that tissue shortening has occurred. The CMSA(arm) at admission to a rehabilitation hospital between study groups was comparable; the median score for the SPAS group was 2 and the NO SPAS group was 2.5 (median fell between 2 and 3). Thus, when controlling for the level of impairment, the present work indicates that a reduction in the end angle of passive stretch can be attributed to passive stretch resistance associated with spasticity. Further, as there were no statistical differences between the NO SPAS group and the HEALTHY group, our results attribute the reduction in passive range to passive stretch resistance associated with spasticity.

Interestingly, the present study revealed that end angle was not affected by velocity of passive elbow flexion or extension. Thus, within the context of the definition of spasticity that identifies it as being 'velocity dependent' [4, 31, 53], the present study indicates that velocity of passive movement does not influence the range of that movement. In the absence of velocity dependence, end angle cannot be attributed to spasticity. Rather, reduced passive range is associated with spasticity and end angle in the present study is an appropriate measure of
passive range of motion. This measure can be used as an evaluation of passive range of motion as one of the key components of resistance to passive stretch after stroke.

### 2.5.2 Catch and Range of Release

Catch angle and range of release during 1000ms trials differentiated the SPAS group from the NO SPAS group and the HEALTHY group during both passive elbow flexion and extension. The results of the present study suggest that manual assessments of spasticity (MAS and MTS), requiring passive stretch of the limb through the full range within the same time window [6], are able to differentiate individuals with spasticity from individuals post-stroke without spasticity or the healthy population. The metrics of catch angle and range of release measured during 1000ms passive movement durations mirror what would typically be expected through manual assessment.

The definition of catch angle in the present study was the first angle after the velocity maximum at which velocity reached a minimum or 0°/second. In some instances the first minimum occurred at or beyond the end angle suggesting that, in these cases, resistance to passive movement could be attributed to reduced passive range of motion. The NO SPAS group produced an earlier catch angle during passive elbow flexion compared to the HEALTHY group. This result is perplexing as there were no statistical differences between these two groups in end angle, suggesting the earlier catch, by our definition, is a result of a key component of resistance to passive movement not identified through traditional definitions of spasticity or contracture. Further investigation is necessary to determine the cause of the difference of catch angle between the NO SPAS group and the HEALTHY group. The results presented in this study suggest that there may be a component of resistance to passive movement beyond spasticity and contracture that appears in the NO SPAS group.

Recent evidence has shifted the focus of spasticity assessment from muscle kinetics during passive stretch to descending regulation of muscle kinematics. Levin and colleagues developed the Montreal Stretch Reflex Threshold (MSRT) measure [54] based on their previous development of the threshold control theory of motor control [55]. The developed measure is based on the hypothesis that muscle force regulation relies on shifts in spatial thresholds of reflexes [54]. The authors suggest that spasticity occurs when the tonic stretch reflex threshold
lies within the biomechanical range of motion for a specific joint and thus the location of the threshold in joint space provides insight that pure kinetic measurement does not. The measure of catch in the current study may be related to the tonic stretch reflex threshold introduced by Levin and colleagues. Thus, there is agreement that kinematic measurement may be able to objectively identify features of spasticity (i.e. angle of onset of resistance). Catch angle identified through kinematics may be more appropriate for monitoring treatment effects and rehabilitation progress in spasticity programs than current, less sensitive clinical scales.

Catch angle and range of release were both influenced by velocity of passive elbow flexion but not passive elbow extension. These findings may identify a direction-dependent level of sensitivity of the protocol. Indeed, direction-dependence is a characteristic of spasticity where upper limb spasticity is more commonly observed in muscle groups that oppose gravity (i.e. elbow flexors) [15, 56]. In the present study, 9 participants were identified as having both flexor and extensor spasticity and were included in passive elbow flexion and extension analysis. Only 4 individuals had pure extensor spasticity and 9 individuals had pure flexor spasticity. The presence of spasticity in both directions may have confounded the catch angle and range of release outcomes; it is possible that the subgroup of individuals with spasticity in both directions comprise a unique classification of individuals with elevated muscle tone.

Notwithstanding the direction-dependent effect of velocity, catch angle appeared earlier and range of release was larger with decreasing velocity of passive elbow flexion. This result suggests a velocity dependence of the outcomes. Despite the later appearance of the catch angle and decreased range of release, 61% of the SPAS group still produced a catch and release at the 1500ms duration. It is possible that the slowest duration of 1500ms may still elicit the stretch reflex. Patrick and Ada [22] found that the stretch reflex threshold was not exceeded when applied torque at the elbow was less than 2 Nm. The average imposed torque during extension trials of 1500ms duration in the current study averaged 1.64 Nm but reached a maximum of 5.08 Nm. Thus, the inclusion of a slower duration of movement may provide further sensitivity to velocity of passive movement. However, as velocity dependence was observed, catch angle and range of release can be used as an evaluation of spasticity as one of the key components of resistance to passive stretch after stroke.
2.5.3 Tone Angle

Tone angle intended to measure the first sign of resistance to passive stretch, as compared to healthy controls, independent of spasticity. As such, the first angle at which the trajectory differed from healthy controls at the slowest speed, thus minimizing the effects of spasticity, was measured. Tone angle during 1500ms trials differentiated the SPAS group from the NO SPAS group during passive elbow flexion and extension. This result suggests that spasticity is associated with elevated resistance to passive stretch at slower speeds. Tone angle cannot be attributed to spasticity or contracture in the current study as there were differences between the groups at the slowest speed of passive movement and the tone angle occurred prior to the end range. As such, tone angle may be an appropriate measure of non-neurogenic resistance to passive stretch prior to the end range. Mullick and colleagues [57] found a relationship between spasticity and rigidity; a common feature of both is the reduction of the tonic stretch reflex threshold [55] that results in inability to relax muscles at specific joint positions. The findings of the current study support the presence of non-neurogenic resistance to passive stretch prior to the end range.

A total of 100% of the SPAS group and 58% of the NO SPAS group exhibited a tone angle (i.e. trajectory different than healthy controls) during extension trials while 85% of the SPAS group and 50% of the NO SPAS group exhibited a tone angle during flexion trials. This suggests a high prevalence of elevated resistance to passive movement, independent of spasticity, following stroke that may confound current clinical measurement of resistance to passive movement or spasticity.

Tone angle appeared earlier in both directions with increased duration of movement, an unexpected result. This finding agrees with Lee and colleagues [58] who have shown that rigidity may be affected by velocity-dependent stretch reflexes. It is entirely possible, however, that the tone angle appeared earlier with increasing duration because the force produced by the KINARM™ was higher relative to the viscoelastic properties of the joint. Still, tone angle appeared earlier with the presence of spasticity suggesting that spasticity is associated with tone angle. This measure may be used as an evaluation of non-neurogenic resistance to passive stretch independent of spasticity or decreased end range.
2.6 Limitations

The primary limitation of this study was using the MAS and MTS to identify individuals with spasticity. The subjectivity of these scales may have misclassified the extent or presence of spasticity; however, these measures are the current clinical standard for assessment of spasticity. Future work could include EMG analysis to confirm the presence of muscle activity that is characteristic of spasticity. One must consider that the standardized position for the MAS and MTS involves passive movement against gravity while the KINARM™ provides full weight support for the limbs and assessment is done in the horizontal plane. Thus, further study needs to identify the impact of performing a passive movement task in different planes. A second limitation was the selection of individuals with mild to moderate spasticity. Individuals with severe spasticity were unable to complete the task. It would be expected that they would behave similarly but conclusions for severe spasticity cannot be drawn from this study. Finally, outcome measures that included a healthy control group need to be examined with caution as the HEALTHY group was not age or sex matched with the patient groups.

2.7 Conclusion

Kinematic variables observed with passive elbow flexor and extensor stretch may uniquely characterize spasticity. Individuals diagnosed with spasticity exhibit a characteristic catch and release measurable as velocity minima prior to the desired end range of motion and subsequent movement. The catch angle appeared earlier when compared to stroke survivors without spasticity and healthy controls. Decreased passive range of motion (end angle) and an earlier sign of resistance to passive movement (tone angle) were also observed in individuals diagnosed with spasticity as compared to stroke survivors and healthy controls. Kinematic analysis provides an objective measurement of spasticity as a key component of resistance to passive stretch. This novel approach to generating passive limb stretch identifies unique metrics of spasticity that may contribute to the development of a clinical measure of spasticity.
3.0 Investigating Implications of Spasticity on Upper Limb Active, Functional Movements Following Stroke

3.1 Abstract

A primary goal of assessment following stroke is to understand the impact of specific impairments on function. The Modified Ashworth Scale (MAS) and the Modified Tardieu Scale (MTS) are common clinical assessment tools for the measurement of spasticity, a relatively prevalent consequence of stroke. Neither scale considers active, functional movements, leaving the impact of spasticity on function undeterminable. The purpose of this study was to identify the impact of spasticity on components of active, functional movement in stroke survivors with upper limb spasticity. This was accomplished through three tasks performed in a virtual environment (KINARM™ robotic exoskeleton) that measured limb position sense, limb preference during bilateral activity, and movement characteristics during reaching. The findings of this study revealed that spasticity may impact active, functional movement. Stability during reaching and use of the more affected side may be impacted by spasticity during active, functional movement. Stability during reaching was assessed through initial direction error and average hand speed when the hand was supposed to be at rest within a target (posture speed). The presence of spasticity was associated with higher posture speed ($0.84 \pm 0.11 \text{ cm/s}$) compared to no spasticity ($0.50 \pm 0.09 \text{ cm/s}$) and a larger initial direction error ($8.36 \pm 1.32^\circ$ and $5.12 \pm 1.04^\circ$, respectively). During bilateral activity, the presence of spasticity was associated with less movement area with the more affected side ($940.91 \pm 53.16 \text{ cm}^2$) compared to no spasticity ($1175.85 \pm 89.39 \text{ cm}^2$). Limb position sense was not significantly impacted by the presence of spasticity. These results indicate that the tasks performed in this study identify components of active, functional movement that are influenced by spasticity.
3.2 Introduction

Survivors of stroke are faced with a range of sensorimotor impairments that limit functional participation. Because the specific presentation of these impairments is typically unique to the individual, neurorehabilitation programs must also be appropriately individualized. Stroke survivors view upper limb recovery as a critical, but often neglected process, and consider the magnitude of their upper limb functional deficits to be poorly understood or appreciated [59]. Despite the importance of upper limb recovery to individuals following stroke, neurorehabilitation programs have historically been less successful for the upper limb, with only 57% of stroke survivors achieving fair to good recovery of arm motor function after 4 years [2].

A decreased ability to use the arm in everyday tasks and not having enough movement to work with have been identified as critical delimiters on recovery of post-stroke movement control [41]. Thus, identifying the precise impairments that reduce movement or use of the upper limb may help direct neurorehabilitation programs that aim to facilitate functional recovery.

Spasticity, which is observed in 20-25% of individuals within twelve months following first-ever stroke and which presents more commonly and more severely in the upper limb [3], has been shown to cause contracture [51] and decrease passive range of movement [8]. Despite the established relationship between spasticity and soft tissue changes that decrease movement (i.e. contracture), the link between spasticity and active, functional movement is less clear. Spasticity seems to contribute to activity limitations, but this is not always the case [60]. There is a need to establish which individual components of active, functional movement are influenced by spasticity to better understand the impact of post-stroke spasticity on function.

Spasticity has been recently redefined as a more broad concept: “disordered sensorimotor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained activation of muscles” [5]. Often presenting as activation of muscles in response to passive stretch, the traditional assessment of spasticity, namely the Modified Ashworth Scale (MAS) [6], does not take function into account. Although the MAS is appropriate for identifying the presence of spasticity, it is not an indicator of the impact of spasticity on active, functional movement. Scores on the MAS have been shown to correlate with traditional clinical post-stroke assessments, including grip strength, active range of motion, Fugl-Meyer arm motor test,
Quality of Upper Extremity Skills, the Gross Motor Function Measure, and the Barthel Index [24]. A moderate to high correlation between upper limb MAS scores and scores on the Lindmark Motor Assessment Scale and the Rivermead Mobility Index have also been found [61]. General, moderate improvements in function [14] and reduced disability and carer burden [62] following reduction in spasticity have been observed. However, reduction in spasticity was not associated with changes in the Action Research Arm Test [16, 18] or the Box and Block Test [18]. As ‘not enough movement to work with’ was cited as a primary factor limiting upper limb recovery [41], it is important to note that reduction in spasticity was associated with improved amount of use according to the Motor Activity Log-30 [18].

Assessment of spasticity should include an active component, warranted by the established relationship between spasticity and activity in some studies. Indeed, the SPASM consortium [11] has indicated that spasticity assessment should include an active component as a more informative measure with greater ecological validity and more sensitivity to relevant change. General classes of active, functional movement, listed on the right side of Figure 11 (adapted from Figure 1), represent groups of parameters (i.e. movement speed encompasses maximum speed, average speed, and speed maxima count) that are measureable during active movement. Identifying individual classes of active, functional movement may unmask subtle deficits not immediately observable using traditional scales. To date, the literature does not examine the impact of spasticity on these underlying components of active, functional movement. Thus, the tasks in the current study were selected to isolate the impact of spasticity on individual components of active, functional movement. This information is not readily apparent or the focus of traditional scales but may help guide rehabilitation. Spasticity may be a potential target during rehabilitation if there is a link between spasticity and components of active, functional movement that, if improved, may lead to increased use of the arm in everyday tasks and more movement to work with, key factors to upper limb recovery [41].
The purpose of the present study was to establish the impact of spasticity on active, functional movement. The primary objective was to identify unique components of active, functional movement impacted by spasticity to differentiate between individuals with and without spasticity as determined by clinical scales. Due to the paucity of literature examining the impact of spasticity on active, functional movement, the nature of spasticity directed the hypothesis in the current study. It was expected that altered sensitivity to stretch would impair spatial awareness of the arm during the limb position matching task and involuntary activation of muscle would impair stability during the visually-guided reaching task. Further, it was expected that the abnormal postures characteristic of spasticity would limit use of the arm during the bilateral object hit task.

Figure 11: Conceptual Model Indicating the Key Components of Active Movement.
3.3 Methods

3.3.1 Participants

Individuals with stroke (n=42) from the Toronto Rehabilitation Institute (TRI) and Sunnybrook Health Science Centre (SHSC) were recruited to participate in this study. Participants were excluded (n=20) from analysis if they were unable to functionally participate using their more affected side (n=18), unable to follow instructions (n=1), or within the effective period (3 months) of spasticity management (Botulinum toxin-A) (n=1). The remaining 22 individuals were attending inpatient (n=7) or outpatient (n=15) services at either of the two centres. Ethical clearance was granted from the TRI Research Ethics Board and the University of Toronto Ethics Board. Research was conducted at TRI and all individuals provided written informed consent.

Adults attending inpatient or outpatient stroke rehabilitation services were eligible to participate in this study. Individuals were excluded from participating if there was a communication barrier that limited task understanding or if their latest BTX-A injection was within 3 months of the study date. Further, individuals with managed or unmanaged neurological disorders beyond stroke were not able to participate. The 22 eligible participants were split into two groups as determined by the MAS: individuals with spasticity [SPAS] and individuals without spasticity [NO SPAS] following stroke. Demographics, including clinical scores and stroke information, for each group can be found in Table 4.
Table 4: Demographic Information for Study Participants in the SPAS and NO SPAS Groups (Chapter 3). Unless specified, all values are presented as mean (range).

### 3.3.2 Experimental Setup

Upper limb functional tasks were administered using the KINARM™ robotic exoskeleton for the upper limb (BKin Technologies Ltd., Kingston, ON, Canada) [40] (Figure 12). All tasks were controlled and relayed using a real-time computer and Dexterit-E™ (version 3.2.3 to 3.2.5) data acquisition software. Task objects (i.e. targets) were displayed in a 2-D virtual reality display unit and appeared on the same plane as the individual’s movement.
The exoskeleton was adjusted for each participant to match body shape and size while providing full weight support for the upper limbs.

Participants were seated in the KINARM™ and seat height was adjusted so that the bottom of the arm troughs were aligned to shoulder height. The shoulder remained in approximately 90° abduction throughout the duration of testing and the exoskeleton was aligned to the vertical axis of the shoulder. Shoulder and elbow movements were permitted in the horizontal plane about the joint centres. The final position of the KINARM™ in relation to the display unit allowed the individual to comfortably rest their head on a pad while viewing the tasks.

### 3.3.3 Task Descriptions

The KINARM™ has been used previously to quantify upper limb sensorimotor function in the stroke population using three tasks: limb position matching [38], object hit [48], and visually-guided reaching [47]. Automated versions of these tasks with developed measures were provided by BKIN Technologies (Figure 13) for the purposes of this study. Not all developed measures were analyzed during this preliminary work as some were deemed unlikely to be influenced by spasticity. For both groups, the more affected arm was tested first followed by the less affected arm during unilateral tasks. Where applicable, target locations were standardized to each individual’s arm length. Data was sampled at 1 kHz.
Figure 13: *Active task protocol*. From L to R: limb position matching, object hit, and visually-guided reaching. Hand trajectories represented by purple lines are only visible to the examiner.

Each participant was given the following instructions for the limb position matching task [38]:

"When I start the task the robot will be moving your [passive] arm. What I would like you to do is mirror match the position with your other arm. What I mean by this is, (show/demonstrate) if the robot moved your [passive] arm to this position, I would like you to move your [active] arm to the mirror position (watch where they put the other arm and repeat sample movements until they understand a mirror match). You will do this without being able to see your arms or hands. Once you have mirror matched the position as best you can, please give me an 'ok', 'there', 'done' or something similar. I will then hit a button to advance to the next trial. You will have lots of time to do this task."

In the horizontal plane, the KINARM™ passively moved the participant’s arm to one of four randomized locations. Vision of the upper limb and hand was blocked while the individual attempted to mirror match the passive arm’s position. A single trial was complete when all four positions had been attempted. A total of 5 trials were completed.

Each participant was given the following instructions for the object hit task [48]:

"In this task you will have green paddles at the ends of your hands. Red balls will start dropping from the top of the screen towards you. I would like you to hit the balls away from you with the paddles. You can hit the balls with either hand. The task lasts about two and a half minutes, the balls will start coming more quickly and there will be more of them as time progresses. Hit as many of the balls away from you as possible using either hand."
Objects fell randomly towards the individual from 10 spatial bins. A total of 30 objects from each bin fell throughout the duration of the task. Each subsequent object from a specific bin travelled faster than the previous. As such, multiple objects could fall at one time from different locations and at different speeds.

Each participant was given the following instructions for the visually-guided reaching task [47]:

"In this task there will be a white light that represents your fingertip. A red target will appear on the screen in front of you. What I would like you to do is move the white light into the red light. Wait until a second red light appears, then reach quickly and accurately to it. Wait there until another red target appears and then again reach as quickly and accurately as you can towards that target. Sometimes there won’t be a second red target that comes on. When that happens, just stay where you are."

Four possible targets appeared in random order. The individual would reach to the target and wait until the centralized target reappeared. A single trial was complete after all four targets have been reached. During each trial, there was one instance where a peripheral target would not appear and the individual had to remain at the centralized target. A total of 5 trials were completed.

3.3.4 Outcome Measures

Key outcome measures were specific to each task and aimed to characterize the individual components of active, functional movement required to perform the task. Spatial variables that characterized the ability to locate the arm in space during the limb position matching task were contraction/expansion ratio and shift. Contraction/expansion ratio compared the area moved by the subject to the area moved by the KINARM™. A contraction/expansion ratio < 1 indicates contraction (Figure 14A) while a contraction/expansion ratio > 1 indicates expansion (Figure 14B). Shift compared the difference between the final position (mirrored) of the arm moved by the subject and the final position of the arm moved by the KINARM™. A positive value on the X axis indicates a lateral shift (Figure 14C) and a positive value on the Y axis indicates a distal shift (Figure 14D).
Figure 14: *Outcome Measures Characterizing Limb Position Sense during Limb Position Matching*. The green lines connect the hand positions of the four target locations moved to by the KINARM™. The blue line represents the mean hand position of the arm moved by the individual and the dotted blue line is a mirroring of the mean hand positions of the arm moved by the participant. Contraction (A), expansion (B), lateral X shift (C), and distal Y shift (D) are identified for four sample participants (A, B, and C are from the SPAS group; D is from the NO SPAS group).

The percentage of objects hit during the object hit task was representative of task success during bilateral, active movement. To characterize the active contribution of the more affected side, the percentage of objects hit, the average hand speed, and the total movement area with the more affected side was recorded.

Temporal and spatial elements that were used to characterize reaching were posture speed, initial direction error, movement time, reaction time, and maximum speed. Posture speed was the mean hand speed when the hand was fixated on a target, averaged across all targets and all
trials. Initial direction error (Figure 15) was measured as the angular deviation between a straight line to the target and a straight line to the hand position of the first speed minima. Movement time was recorded as the total time elapsed from movement onset to movement offset while reaction time was recorded as the time between illumination of the destination target and the onset of movement. Maximum hand velocity was recorded independent of target and trial.

![Diagram of initial direction error](image)

Figure 15: Schematic Representing the Process of Identifying the Initial Direction Error for the Visually-Guided Reaching Task.

The Modified Ashworth Scale (MAS) and the Modified Tardieu Scale were measured by one of three assessors; two trained therapists and one trained study investigator. Attempts were made to minimize the time-of-day effects [49] by administering each scale immediately before KINARM™ testing.

3.3.5 Statistical Analysis

Descriptive statistics characterized the study groups. Independent t-tests were used for each variable to probe the components of active, functional movement that distinguish individuals in the SPAS group from those in the NO SPAS group. In cases where the assumption of normality was violated, as assessed by the Shapiro-Wilk Test, a non-parametric Mann-Whitney U Test was used. In cases where the data was normally distributed but violated the assumption of homogeneity of variance as measured by Levene’s Test, the t-statistic for the “equal variances not assumed” condition was used. Statistical significance was set at $p \leq 0.05$. Statistical
algorithms embedded within Dexterit-E™ calculated the means for each outcome measure and statistical analysis was performed using SPSS v22.0 for Windows.

### 3.4 Results

Outcome measures were averaged across all trials and group means reported as (mean ± standard error of the mean). The SPAS group (n = 11) was made up of individuals with flexor spasticity (n = 5; 45%), extensor spasticity (n = 1; 10%), or both (n = 5; 45%). A total of 100% (n = 11) of the NO SPAS group was included in analysis. Outliers were included in statistical analyses.

Between-group analysis of each outcome measure evaluated characteristics of limb position sense, limb preference during bilateral activity, and elements of reaching. Spatial characteristics (i.e. contraction and shift) of the matched area during the limb position matching task were evaluated to provide a general understanding of limb position sense as the literature examining spasticity and limb position sense and/or proprioception is negligible. Similarly, the literature examining the association between spasticity and the non-use of the more affected side is negligible. As such, outcome measures were chosen to characterize the use of the more affected side during bilateral activity. Outcome measures for the visually-guided reaching task were selected to evaluate the quality and timing of reaching similar to traditional scales (i.e. Wolf Motor Function Test [63]).

### 3.4.1 Limb Position Matching

There were no statistically significant differences between the SPAS group and the NO SPAS group for the limb position matching task. X contraction/expansion ratio was 0.75 ± 0.08 for the SPAS group and 0.76 ± 0.05 for the NO SPAS group (U = 65.00, z = 0.30, p = 0.797). Y contraction/expansion ratio was 0.98 ± 0.08 for the SPAS group and 0.92 ± 0.05 for the NO SPAS group (t(20) = 0.54, p = 0.593). Y shift was -1.80 ± 0.72 cm for the SPAS group and -1.8 ± 1.13 cm for the NO SPAS group (U = 53.00, z = -0.49, p = 0.652). The difference in X shift for the SPAS group (0.98 ± 1.44 cm) compared to the NO SPAS group (-3.44 ± 1.74 cm) approached significance (t(20) = 1.954, p = 0.065).
3.4.2 Object Hit

There was a statistically significant difference between the SPAS group (940.91 ± 53.16 cm²) and the NO SPAS group (1175.85 ± 89.39 cm²) for total area covered with the more affected side during the object hit task (t(20) = -2.26, p = 0.035). Despite this difference, there were no statistically significant differences between the SPAS group (50.70 ± 3.95 %) and the NO SPAS group (52.09 ± 2.99 %) for percentage of total objects hit (t(20) = -0.28, p = 0.781). The difference between the SPAS group (36.60 ± 2.93 %) and the NO SPAS group (43.51 ± 1.93 %) for percentage of objects hit that were hit with the more affected side approached significance (t(20) = -1.97, p = 0.063). The differences in average hand speed between the SPAS group (21.24 ± 2.45 cm/s) and the NO SPAS group (25.54 ± 2.68 cm/s) were not significant (t(20) = -1.18, p = 0.250). Means and standard errors for total objects hit and total area covered with the more affected side are represented in Figure 16.

![Figure 16](image_url)

Figure 16: Mean Total Objects Hit out of 300 (A) and Total Movement Area with the More Affected Side (B) during the Object Hit Task. Error bars represent ± 1 SEM. Asterisks indicate statistically significant differences (p < 0.05).

3.4.3 Visually-guided Reaching

There was a statistically significant difference between the SPAS group and the NO SPAS group for posture speed (0.84 ± 0.11 and 0.50 ± 0.09 cm/s, respectively) (t(20) = 2.30, p =
0.032) and initial direction error (8.36 ± 1.32 and 5.12 ± 1.04°, respectively) (U = 27.00, z = -2.20, p = 0.028). There were no statistically significant differences for reaction time (SPAS = 0.42 ± 0.36 s and NO SPAS = 0.36 ± 0.28 s) (U = 38.00, z = -1.48, p = 0.151), movement time (SPAS = 1.57 ± 0.19 s and NO SPAS = 1.34 ± 0.09 s) (U = 46.00, z = -0.95, p = 0.365), or maximum hand speed (SPAS = 20.71 ± 1.48 cm/s and NO SPAS = 22.90 ± 1.80 cm/s) (t(20) = -0.94, p = 0.358). Means and standard errors for posture speed and initial direction error are represented in Figure 17.

![Figure 17: Mean Posture Speed (A) and Initial Direction Error (B) during the Visually-Guided Reaching Task. Error bars represent ± 1 SEM. Asterisks indicate statistically significant differences (p < 0.05).](image)

### 3.5 Discussion

The purpose of the present study was to identify components of active, functional movement impacted by spasticity. Characteristics of active, functional movement were compared during three tasks: limb position matching, object hit, and visually-guided reaching. The results indicate that spasticity may not significantly influence limb position sense but may have an effect on stability during reaching and use of the more affected side. An established impact of spasticity on active, functional movement may help appropriately guide neurorehabilitation for individuals with spasticity. Further, examining the influence of spasticity on active, functional movement may help focus neurorehabilitation programs during anti-spastic interventions to take
advantage of the temporary reduction in spasticity. Access to effective programs has been shown to be a key factor for increased function following reductions in spasticity [13].

3.5.1 Limb Position Matching

Limb position matching did not differentiate the SPAS group from the NO SPAS group. Both groups actively matched a smaller area than the KINARM™ controlled arm (a contraction/expansion ratio < 1.0) and this effect was magnified in the X direction compared to the Y direction. A possible explanation for excessive contraction in the X direction only may be that lateral-medial movements for this task required more shoulder involvement than distal-proximal movements. Both groups shifted the matched area distally. However, the SPAS group shifted the matched area laterally while the NO SPAS group shifted the matched area medially and this result approached significance. This suggests that individuals with spasticity may sense their affected limb position to be further laterally than in actuality.

We expected to find a difference between groups in limb position sense given the stretch-sensitive muscle overactivity characteristic of spasticity [64]. Stretch-sensitive paresis, defined as impeded voluntary motor neuron recruitment as a result of excessive responsiveness to stretch [64], may influence positional feedback of the limb. However, no significant differences between groups for limb position sense were found in the current study. Although the available literature examining spasticity and limb position sense is scarce, our findings are consistent with the findings of Prokopenko and colleagues [65] and Tyson et. al. [66]. Prokopenko et. al. [65] concluded that a proprioceptive correction intervention resulted in no change in spasticity. Similarly, Tyson and colleagues [66] recently found evidence that spasticity did not significantly influence sensory impairment following stroke. Only one study was found that produced a positive finding [67], indicating that descending activation of gamma efferents supplying hypertonic muscles may be hypersensitive to stretch. However, this study was conducted with individuals with cerebral palsy. The results of the current study support the few studies that examine spasticity and limb position sense and found no difference associated with the presence of spasticity. It may be possible that limb position sense is not necessarily impaired during active, functional movements but only manifests during passive movement, reflecting only a sensitivity to stretch. Further, it may be possible that contraction/expansion ratio and shift
aren’t suitable to detect differences and individual target accuracy may be more appropriate, warranting further analysis.

3.5.2 Object Hit

Individual limb performance during bilateral activity differentiated the SPAS group from the NO SPAS group. Despite no significant difference in total number of objects hit or percentage of objects hit with the more affected side, movement area by the more affected side was significantly lower for the SPAS group. Average speed with the more affected side was not different between the groups, suggesting that the decrease in the area covered during the task was a result of spatial deficits (i.e. decreased range) or a decision by the individual to focus on the less affected side. Different strategies observed in the SPAS group suggest the latter may have a negative effect on use of the arm. Indeed, individuals in the SPAS group would often cease use of the more affected side as the task conditions became more challenging or ignored the more affected side from the outset despite having sufficient active movement. The individuals with spasticity in the current study had the capacity to actively participate with the more affected side as evident by participation in the unilateral tasks, but used their more affected side less. This is a prime example of the observed learned non-use of the more affected limb that is consistent with stroke recovery [68].

There appears to be a positive effect of reducing spasticity on function [14, 62]. However, few studies have examined the role of spasticity in limiting the use of the more affected side during bilateral movement. During unilateral reaching, Bensmail and colleagues [18] found a reduction in spasticity was associated with increased use of the arm that improved reaching in subsequent testing. Although this supports the findings of the current study, the authors conducted a within-subjects repeated measures design of unilateral movement. The current study, to our knowledge, is the first comparing limb selection during bilateral activity in individuals with spasticity and individuals without spasticity following stroke. Despite no difference in task score (i.e. number of objects hit), spasticity was found to impair limb selection during bilateral tasks.
3.5.3 Visually-guided Reaching

Reaction time, movement time, and maximum speed during reaching were unable to differentiate individuals with and without spasticity following stroke. However, posture speed and initial direction error were significantly higher for the SPAS group compared to the NO SPAS group. These measures may be attributed to overall stability during reaching [47]: posture speed represents the ability to stabilize on a target while initial direction error may be influenced by decreased stability. Stability during reaching is an essential element of overall independence and performance of activities of daily living, including dressing and hygiene. As such, targeting spasticity through rehabilitation strategies may improve stability during reaching and have functional benefits in real-world situations.

The influence of spasticity on reaching has been rarely examined in the literature. Contrary to the findings of the current study, Shaw and colleagues [16] found a reduction in spasticity as unlikely to improve reaching as measured by the Action Research Arm Test and the 9-hole peg test. Similarly, Leonard colleagues [69] found that increased passive muscle stiffness and decreased reflex thresholds, features associated with spasticity, were not primary factors limiting reaching. However, both of these studies mentioned examined overall success of the reach; participants in the current study were capable of reaching all the targets. Examining the spatial characteristics of the reach itself may have identified subtle deficits not found in the aforementioned studies. Very few studies have examined the influence of spasticity on individual components of reaching movements. Hingtgen and colleagues [70] found a significant difference in peak angular velocity during reaching between the affected arm and the unaffected arm in patients with spasticity. Although the current study did not find a difference between groups for peak velocity during reaching, this may be explained by examining differences between groups as opposed to within individuals using their less affected side. The current study also found no difference in movement time between the two study groups. Levin found that the level of clinical spasticity was significantly correlated with movement time during reaching [71]. As such, it is possible that the inclusion of individuals with more severe spasticity may have led to a significant difference between the SPAS group and the NO SPAS group.
3.6 Limitations

The MAS and MTS were used to identify individuals with spasticity in the current study. These scales are known to be subjective and may have misclassified the presence of spasticity. As these scales are the current clinical standards for assessment of spasticity, they were used in the current study. The second limitation was the inclusion of individuals with mild to moderate spasticity only. Although there was no exclusion criteria concerning MAS or MTS scores, 20 individuals recruited were unable to functionally participate in all three tasks with some individuals unable to reach targets during the visually-guided reaching task or use their more affected side during the object hit task. Inclusion of these individuals was not possible. As such, the results of the current study should only be applied to mild to moderate levels of spasticity. The final limitation was the absence of clinical activity or function scales that would have provided context. Further work may take advantage of established clinical scales to isolate the effect of spasticity from effects of hemiplegia or deficits in dexterity and proprioception.

3.7 Conclusion

This study identified an impact of spasticity on active, functional movement. Stability during reaching and use of the more affected side may be impacted by spasticity during active, functional movement. These observations may not be evident through common clinical assessment of activity. Spasticity did not appear to influence limb position sense during a limb position matching task. The results of the current study agree with the inclusion of an active component in spasticity assessment. Elements of an active assessment of spasticity could include stability measures during reaching and amount of use of the more affected side during bilateral movements. The inclusion of an active component may be more relevant to the individual and provides another means of monitoring progress and treatment outcomes. This novel approach to assessing active, functional movement identified components of activity impacted by spasticity that are not observable using current clinical scales for the measurement of spasticity.
4.0 General Discussion

The purpose of this thesis was to objectively measure spasticity as a key component of resistance to passive stretch and establish the impact of spasticity on active, functional movement. Three specific considerations were identified in the introductory chapter:

1. Produce an objective measure of spasticity by isolating and quantitatively defining ‘catch’ and ‘release’.

2. Produce an objective measure of spasticity by ensuring assessment differentiates spasticity from non-neurogenic resistance to passive stretch through the inclusion of multiple velocities of movement.

3. Produce an objective measure of spasticity by establishing the impact of spasticity and active, functional movement.

The KINARM™ [40] was used to address each consideration through the accurate measurement of joint kinematics during passive stretch and active, functional movement with the necessary specificity to capture the essential components of active, functional movement. This thesis was based on the methodological considerations for the measurement of spasticity outlined by Burridge and colleagues [11]: assessment must allow for variable velocities of displacement, must include both an active and passive component, and must be easily repeatable with a clearly defined protocol.

Kinematic profiles may be appropriate for identifying the presence of spasticity following stroke while differentiating spasticity from non-neurogenic resistance to passive movement. Catch angle, defined as the 1\textsuperscript{st} velocity minimum after the maximum velocity, and range of release were velocity-dependent measures that were able to identify the presence of spasticity. These measures were analyzed during 1000ms trials, closely mimicking the standardization of common clinical scales [6, 31]. End angle was not velocity-dependent and effectively differentiated spasticity from resistance to passive stretch caused by decreased passive range of motion. This novel approach to the measurement of spasticity addressed the first two considerations outlined above. Catch angle and range of release were defined in easily understood terms and isolated, providing additional insight to these terms first introduced by
Bohannon and Smith [6]. Further, we proposed that the KINARM™ was able to isolate the neurogenic properties of spasticity, effectively characterizing the key components of resistance to passive movement that are not exclusively dependent on hyperexcitability of the stretch reflex [8]. As a more objective approach, kinematic profiles of resistance to passive movement may be more appropriate than traditional clinical scales at identifying changes in severity of spasticity.

Assessment of individual components of active, functional movement was able to unmask unique consequences of spasticity following stroke. As traditional scales for the measurement of spasticity do not consider active movement during assessment, identifying the impact of spasticity on active, functional movement addressed the third consideration outlined above. It was found that spasticity impacted stability during reaching and limb selection during bilateral activity but not with sensory deficits following stroke, namely limb position sense. Traditional impairment and functional scales focus on ability to complete a task (i.e. Box and Block Test) or the time required to complete the task rather than on the components of active, functional movement that contribute to the extent of dysfunction characterized by task performance. Assessing individual components of active, functional movement provides more insight into how an impairment influences activity. Task scores during bilateral activity and capacity to reach to a target were not influenced by spasticity. However, overall movement area with the more affected side was less during bilateral movement and stability during reaching was impaired despite not hindering the ability to compete the reach.

Overall, the findings of Chapters 2 and 3 indicate that there are unique components of passive and active movement that characterize individuals with post-stroke upper limb spasticity. Typical ‘catch’ and ‘release’ patterns are observable in response to passive movement that are absent or less profound in individuals without spasticity. Thus, it is possible to quantify spasticity through kinematic analysis while standardizing the assessment. Controlled, objective measurement of spasticity may appropriately differentiate individuals with post-stroke spasticity from individuals without spasticity. This allows more definitive conclusions to be drawn when examining the association between spasticity and active, functional movement. The unique components of active, functional movement that characterize individuals with post-stroke upper limb spasticity are decreased stability during reaching and decreased use of the more affected side during bilateral tasks. These are essential elements of performance of activities of daily
living; poor stability during reaching and unilateral compensation may lead to chronic difficulties in dressing, hygiene, and overall independence. Thus, targeting spasticity during rehabilitation may be effective at improving reaching and use of the more affected side, directly influencing activities of daily living and independence, and in turn, quality of life.

4.1 Limitations

There were several limitations during the present studies. The most significant limitations were: limited data on healthy controls, using the MAS and MTS to group participants, and limiting participation to individuals with MAS or MTS \( \leq 2 \).

Resistance to passive movement included a healthy control group as this was a novel task that required some understanding of how a healthy individual would behave. Individuals were selected using a convenience sample and were not age or sex matched to the patient groups. As such, conclusions that rely on healthy group comparisons need be made with caution. In retrospect, the less affected side for the patient groups may be more appropriate in identifying a typical pattern of resistance to passive movement. This would account for any age-related changes to joints that may result in resistance to passive movement (i.e. skin elasticity). Further, although the MAS and MTS were not used on the less affected side, it is likely that both clinical scales would result in a score of 0 on the less affected side for both patient groups.

The MAS and MTS were used to group participants in the present studies. Both scales are used extensively in clinical and research settings but have notable deficiencies. Further development and validation of objective, sensitive measurement of spasticity such as that used in the 1st study may be more appropriate in future research when grouping participants. However, as no such measure has been sufficiently validated and as the MAS and MTS are extensively utilized, these scales were used in the present studies. It is important to note that both scales were used rather than just one to decrease chance of inappropriately grouping participants. There was near perfect agreement between the two scales in the present studies.

Passive manipulation of the elbow was not possible for individuals with higher levels of spasticity (MAS > 2) as force required to move the limb would have exceeded safe values. However, kinematic profiles established for lower levels of spasticity in the current study may
be applied to different measurement tools (i.e. accelerometers). For example, higher levels of spasticity may be assessed manually by an examiner while kinematics are derived from accelerometer data. Similarly, individuals with higher levels of spasticity (MAS > 2) consistently had insufficient active movement to perform functional tasks and were not included in analysis. Although there was no way of accounting for this in the present study, repeated testing during these functional tasks may still be informative. Although an individual may not be able to actively reach a target, a sensitive measure of position may be able to observe small change in position (i.e. participant is closer to the target) in subsequent visits.

4.2 Future Direction

An objective, sensitive measurement of spasticity and an established impact of spasticity on active, functional movement produced in this thesis may indicate spasticity as an appropriate target for rehabilitation. Two primary targets of post-stroke assessment were identified in Figure 1: changes in sensorimotor impairments and changes in active, functional movements. Changes in spasticity and impact of spasticity on function have not been fully understood to date. The results presented in this thesis provide an objective measurement of spasticity at the impairment level while establishing a link between spasticity and active, functional movement, namely that spasticity results in decreased stability during reaching and decreased use of the more affected side during bilateral activity. Thus, this thesis provided an improved understanding of post-stroke sensorimotor control through improved understanding of spasticity, a relatively common impairment following stroke.

Kinematic profiles observed with resistance to passive movement allows for a practical alternative to current clinical practice of hand-held goniometer and a therapist’s perception of the extent and location of spasticity. This approach is capable of measuring passive range of motion, resistance associated with spasticity, and resistance occurring when the stretch reflex threshold is not exceeded. As such, kinematic profiles may overcome the established shortcomings of current clinical scales, namely the inability of the MAS to distinguish between spasticity and soft-tissue changes [22], the insensitivity of the MAS [25], and the inability to control velocity in a clinical setting for both scales [25]. As resistance to passive movement encompasses several components (i.e. spasticity, contracture, rigidity) with drastically different
treatment goals, effective measurement and isolation of each component was necessary. Rehabilitation targets are dependent on objective measurements using a highly standardized and repeatable protocol. Future research into the examination of spasticity at the impairment level can use kinematic profiles to objectively characterize spasticity. The next step is to characterize different levels of spasticity while identifying the sensitivity of such measures to anti-spastic interventions. An objective and sensitive measure of spasticity may help identify appropriate medication levels. BTX-A has potent chemodenervating properties and as such, high doses may lead to increased weakness, ultimately resulting in no observable changes in function despite a reduction in spasticity. It has been shown that lower doses of BTX-A result in improved function without affecting strength [13]. All participants in the current thesis were outside of the effective period of BTX-A and sensitivity to BTX-A cannot be established. An objective, sensitive measure may be able to identify an optimal balance between reduction in spasticity and coincidental reduction in strength in response to BTX-A.

The theoretical and methodological considerations for the measurement of spasticity proposed by the SPASM consortium [11] identify an active component of spasticity assessment as necessary to improve ecological validity and provide a more sensitive marker of relevant change. Identifying the key elements of active movement that are impacted by spasticity may help focus neurorehabilitation programs. Optimal recovery is facilitated if there is access to rehabilitation programs in the short time period of active anti-spastic effects of medications [13]. Accordingly, during these periods of time, focus should be placed on improving components of active motor control that have been shown to be impacted by spasticity, namely use of the more affected side during bilateral movement and stability during reaching.

4.3 Conclusion

This work developed an objective measurement of spasticity during both passive and active movement. An objective measurement of spasticity has remained elusive in the literature and this work provides a stepping stone to developing a clinically sensitive measure of spasticity. Including an active component in spasticity assessment allows for sensitive monitoring of the impact of spasticity and anti-spastic medications on function. Changes to key components of
active movement are relevant to the individual and may help focus neurorehabilitation programs that facilitate upper limb recovery following stroke.
5.0 References

1. Tracking Heart Disease and Stroke in Canada. 2009, Public Health Agency of Canada.


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