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Translating Modified Ashworth Scale into Functional Measures and Quantitative Kinematic Values: A Pilot Study

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TRANSLATING MODIFIED ASHWORTH SCALE INTO FUNCTIONAL MEASURES AND QUANTITATIVE KINEMATIC VALUES: A PILOT STUDY

by

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ABSTRACT

TRANSLATING MODIFIED ASHWORTH SCALE INTO FUNCTIONAL MEASURES AND QUANTITATIVE KINEMATIC VALUES: A PILOT STUDY

by

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Introduction: Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes. The gold standard for assessing spasticity in stroke patients is the Modified Ashworth Scale (MAS), but the scale is highly subjective to the clinician's opinion and previous experience and lacks psychometric fidelity. Numerous studies have criticized the scale's subjectivity and lack of rater reliability. Development of a quantitative spasticity device in routine clinical care is warranted. Before doing so, however, it is important to examine how MAS scores translate into functional measures and quantitative kinematic and/or kinetic values.

Methods: Data from 20 subjects (6 female, 14 male; mean age 57 ± 10) with chronic hemiparesis secondary to a cerebrovascular accident (stroke) were used to examine the relationships between the MAS and residual impairments (active range of motion of shoulder flexion, elbow, and wrist, and muscle strength of the elbow flexion and extension), the MAS and functional limitations as measured by the Fugl-Meyer upper extremity assessment, finger to nose movement, the MAS and overall health status following stroke as measured by the Stroke Impact Scale, and to inspect whether there are potential kinematic values or physiological responses that can be used to identify the characteristics of the passive stretch (passive stretch duration, catch

angle, electromyography response). The data were collected at both the Rehabilitation Institute of Chicago and the University of Wisconsin-Milwaukee.

Results: Overall, results showed that stroke subjects who had more severe spasticity tended to have reduced range of motion at the shoulder (flexion) (Pearson correlation coefficient $r_p = -.601$; Spearman correlation coefficient $r_s = -.607$), elbow ($r_p = -.436$; $r_s = -.495$) and wrist ($r_p = -.206$; $r_s = -.305$) joints, as well as reduced muscle strength for elbow flexion ($r_p = -.547$; $r_s = -.618$). The relationship between the MAS scores and the muscle strength for elbow extension was weak ($r_p = -.160$; $r_s = -.191$). Analysis between the FM-UE subscale and MAS revealed a significant negative correlation. The strongest correlation occurred between the FM-UE total score ($r_p = -0.817$; $r_s = -0.806$), while the weakest correlation amongst all subscales occurred between coordination subscale ($r_p = -0.696$; $r_s = -0.684$). A one-way, between-subjects design ANOVA showed significant mean differences between MAS scores and all FM-UE subscales: the FM-Arm subscale ($F_{4,15} = 17.4$, $p < .001$), the FM-Wrist subscale ($F_{4,15} = 4.3$, $p < 0.016$), the FM-Hand subscale ($F_{4,15} = 4.8$, $p < 0.011$), the FM-Corr subscale (F4,15 = 4.4, $p < 0.015$) as well as FM-Total Score subscale ($F_{4,15} = 12.6$, $p < 0.001$). Overall, there was a tendency for increased levels of spasticity per scoring of the MAS to result in decreased motor performance as measured by the FM-UE subscale. There was a moderate negative correlation between MAS score and the Stroke Index Scale hand subscale ($r_p = -0.543$; $r_s = -0.576$), indicating that a higher MAS score may be indicative of the magnitude of impairment in the hand. No significant relationships were demonstrated between the remaining subscales of the Stroke Impact Scale, suggesting that there is little to no relationship between MAS scores and overall health status. In comparing EMG activity and motion capture analysis, there was a marked increase in the EMG response when the

subjects affected limb is stretched into full elbow extension, such phenomenon was not observed when stretching the unaffected limb.

Conclusion: The strong correlation between MAS scores and the residual impairments as well as the FM-UE subscale suggests that a higher MAS score may be indicative of the general stage of motor recovery following incurrence of a stroke. Additionally, there was a marked increase in EMG activity through passive stretching of the affected limb into full elbow extension; conversely, such a phenomenon was not observed in the unaffected limb.

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I. Introduction

Background

Spasticity is a feature of altered skeletal muscle performance involving hypertonia, and has been described as one of the most debilitating complications of neurologic conditions in patients with stroke, brain injury, spinal cord injury, cerebral palsy, or multiple sclerosis. Cohort studies have revealed that 20-40% of stroke survivors are spastic (Leathley et al., 2004; Sommerfeld, Eek, Svensson, Holmqvist, & von Arbin, 2004; Watkins et al., 2002). Patients with spasticity may experience painful spasms, increased muscle stiffness, and loss of joint range of motion (Botte, Nickel, & Akeson, 1988; Ivanhoe, Francisco, McGuire, Subramanian, & Grissom, 2006; Kamper, Schmit, & Rymer, 2001). Severe spasticity may cause functional challenges in various aspects of daily living such as dressing, self-cares, and eating (Bhimani & Anderson, 2014; Jagatsinh, 2009; Katz & Rymer, 1989; Zorowitz, Gillard, & Brainin, 2013). Furthermore, prolonged spasticity that typically persists for 12 months or more after stroke may lead to postural deformity and contracture. The interaction between neural and biomechanical components of spasticity and its potential impact on functional limitation has been described by Barnes & Johnson (Figure 1).

Modified Ashworth Scale

The Modified Ashworth Scale (MAS), modified from the Ashworth (AS) Scale, is the most commonly utilized clinical measure of quantifying spasticity (Table 1) (Ashworth, 1964; Bohannon & Smith, 1987). The original Ashworth Scale introduced in 1964 was constructed using a 5-point ordinal scale, with a Likert-like grade score of 0 (indicating no increase in muscle tone), 1, 2, 3, or 4 (affected part rigid in flexion or extension) to quantify spasticity. In 1987, Bohannon and Smith introduced the grade of "1+" and proposed slight changes in how each score was defined in order to increase the sensitivity of the measure and facilitate greater ease in

scoring, which prompted the renaming of the Ashworth Scale as the "Modified Ashworth Scale" (Bohannon & Smith, 1987). To evaluate elbow spasticity using the MAS, for example, the clinician quickly and passively stretches the patient's affected elbow. The clinician would support the patient's affected extremity at the elbow joint in 90 degrees of shoulder abduction while placing the other available hand on the volar aspect of the patient's wrist to provide support. The clinician begins by bringing the patient's elbow into maximum flexion, quickly stretching the elbow into maximum extension to assess flexor spasticity (i.e., stretching the biceps muscle), and finally moving the elbow back into the starting position of maximum elbow flexion to assess extensor spasticity (i.e., stretching the triceps muscle).

The MAS, although simple to administer and convenient, has been criticized. Briefly, it is highly subjective to the clinician's personal experience and the velocity at which the affected extremity is passively stretched. Some studies showed that the scale lacks inter-rater reliability (e.g., inter-rater reliability: mean = 0.56-0.76, agreement = 66%, 59-78%, intra-rater reliability = 32%, 62-72%) (Blackburn, van Vliet, & Mockett, 2002; Fleuren et al., 2010; Gregson et al., 1999; Gregson et al., 2000; Sloan, Sinclair, Thompson, Taylor, & Pentland, 1992). Studies have illustrated that the MAS also suffers from a clustering effect wherein most of the patients are grouped within the middle grades (Damiano et al., 2002). The MAS scores were not significantly associated with electromyography changes and only moderately associated with resistance. Ambiguity of wording and lack of standardized procedures limit the scale's usefulness for comparison across studies. There are no clear guidelines for stretching velocity during administration of the MAS. It is possible that variable stretching velocities can affect the elicitation of spasticity. There is also poor scale sensitivity to change for rigorous clinical and research application. Overall, studies showed that reliability differs from muscle to muscle, and

suggested that assessment technique must be standardized and adequate training is required to ensure inter-rater reliability.

Additional studies have challenged the scale's validity, contending that it does not adequately distinguish between neurological and mechanical factors that contribute to joint stiffness (Kumar, Pandyan, & Sharma, 2006; Pandyan et al., 1999; Platz, Eickhof, Nuyens, & Vuadens, 2005). Furthermore, the MAS does not account for skeletal muscle changes, such as contractures, which are not necessarily attributable to spasticity alone (Foran, Steinman, Barash, Chambers, & Lieber, 2005).

Numerous studies have explored the relationship between MAS scores and neurophysiological variables. In particular, a study led by Cooper et al. produced findings that indicated a positive correlation of MAS scores with magnitude and duration of surface electromyography response. The authors concluded that this finding is evidential that the MAS reflects spasticity in terms of surface electromyography response during passive stretch (Cooper, Musa, van Deursen, & Wiles, 2005). Similar studies have sought to corroborate this contention, but have only found moderate associations between MAS scores and electromyography, indicating that the scale may be more of a measure of hypertonia rather than spasticity (Bakheit, Maynard, Curnow, Hudson, & Kodapala, 2003; Lin & Sabbahi, 1999; Pisano et al., 2000; Skold, Harms-Ringdahl, Hultling, Levi, & Seiger, 1998). Instead, a more significant relationship was yielded in other studies that examined the relationship between MAS scores and objective measurements of resistance to passive movement, again elucidating what the MAS is actually constructed to measure. While utilization of descriptive measures such as the MAS continues, measures that provide the clinician with neurophysiological characteristics has been advocated. It is clear that discrepancies between clinical measures like the MAS and real neurophysiological

measures (i.e., surface electromyography) persist. Bridging this gap is critical to provision of appropriate anti-spasticity treatment.

Current research trends suggest that there is a substantial effort underway to discover novel therapeutic technologies and strategies to treat spasticity (Bhakta, Cozens, Chamberlain, & Bamford, 2000; Brashear et al., 2002; Phadke, On, Kirazli, Ismail, & Boulias, 2013; Pittock et al., 2003; Remy-Neris, Tiffreau, Bouilland, & Bussel, 2003; Shakespeare, Boggild, & Young, 2003). A host of clinical studies examining the effects of surgical, pharmacological, or therapeutic spasticity interventions refer to the MAS as an outcome measure (Bohannon & Smith, 1987). One particular study by Ivanhoe et al. examined the effects of intrathecal baclofen and produced positive results demonstrating a reduction in subjects' spasticity per scoring of the MAS (Ivanhoe et al., 2006). However, an ordinal scale such as the MAS may lack sensitivity and precision for noting smaller degrees of change in spasticity, thus making it difficult to corroborate the study's conclusions and subsequently limiting clinical application of its findings. Likewise, other studies examining the effect of surgical intervention in individuals with cerebral palsy have produced lower MAS scores (Butler & Campbell, 2000; McLaughlin et al., 1998), purporting that the surgical intervention was successful. The generalizability of these studies, and others alike, is inconspicuous due to the limitations of the MAS. Because the MAS is highly subjective to the clinician's personal opinion, some researchers have contended that clinicians *stop using the Ashworth Scale for the assessment of spasticity* (Fleuren et al., 2010).

Clinical Significance

Stroke is the leading cause of disability in the adult population, affecting over 4 million people in the United States alone (Hinojosa, Rittman, & Hinojosa, 2009). More than one out of every four patients develops spasticity after experiencing a stroke (Wissel et al., 2010). As a

serious detriment to daily function and quality of life, proper management of spasticity is an important component of many rehabilitation protocols. Spasticity is a complex pathophysiological phenomenon, and, as such, should not rely on a subjective scale for quantification.

Although reliability and validity of the MAS has been criticized, currently there is no clinical measurement that outperforms the MAS in quantifying spasticity (Blackburn et al., 2002; Gregson et al., 1999; Pandyan et al., 1999). One solution in advancing the spasticity measure is to improve the precision during administering the MAS by taking away the subjective factor (i.e., develop a quantitative spasticity device in routine clinical care). Increased objectivity in the quantification of spasticity has the potential to assist physicians and therapists in anti-spasticity management and more accurately illustrate the effects of interventions that have otherwise been deemed as ineffective or inconclusive.

Specific Aims

Before developing a quantitative spasticity device in routine clinical care, it is important to understand how the MAS scores translate into functional measures and quantitative kinematic and/or kinetic values. As the first step, we proposed to conduct a pilot study. Our specific aims are:

1. To examine the relationships between the MAS and residual impairments (active range of motion of shoulder flexion, elbow, and wrist, and muscle strength of the elbow flexion and extension).
2. To explore the relationships between the MAS and functional limitations as measured by Fugl-Meyer upper extremity assessment, finger to nose movement, and ability to grasp a bottle of water.

3. To investigate the relationship between the MAS and overall health status following stroke as measured by the Stroke Impact Scale.

For aims 1 to 3: Data from 20 subjects (6 female, 14 male; mean age 57 ± 10) with chronic hemiparesis secondary to a cerebrovascular accident (stroke) will be used to examine the relationships. The data were collected at the Rehabilitation Institute of Chicago.

4. To inspect whether there are potential kinematic values or physiological responses that can be used to identify the characteristics of the passive stretch (passive stretch duration, catch angle, electromyography response).

For aim 4: A case study will be used to inspect the graphical presentation of the kinematics during passive stretching. The data were collected at the University of Wisconsin-Milwaukee.

II. Literature Review

Pathophysiology of Stroke

Cerebrovascular accident (CVA), commonly known as simply stroke, is a neurological event characterized by an abrupt disruption in cerebral circulation producing an array of neurological deficits. While the neurological mechanisms of stroke are widely variable, stroke is delineated into two broad categories (ischemic stroke and hemorrhagic stroke) based on clinical manifestation of symptoms.

Ischemic strokes occur when there is a disruption in blood flow in the brain for seconds or minutes, resulting in infarction (cell death). Infarction occurs if the brain is deprived of circulation for more than a few minutes. It could due to atherosclerosis, excess plaque buildup in the aortic arch, carotid arteries, or cerebral vessels, or emboli, traveling intravascular masses in the arteries. The most common precipitating event of cardioembolic strokes is occlusion of the

middle cerebral artery or the posterior cerebral artery in the brain. Unlike atherosclerotic events, symptoms that are produced from an embolus are often more sudden, producing severe, observable neurological deficits. Regardless of etiology, the clinical presentation in cerebral ischemic/infarction events is predicated upon the site of occlusion in the artery. Common clinical presentation of ischemic stroke includes but is not limited to weakness and loss of sensation in the contralateral leg and foot, contralateral sensory loss and weakness, loss of the nasal half of vision on the opposite side and loss of the temporal half of vision on the same side, aphasia, neglect, ataxia, vertigo, hiccupping, and difficulty swallowing (Frizzell, 2005).

Hemorrhagic strokes are the third most common cause of stroke and are associated with about a 50% mortality rate. These occur when there is a leak or rupture in a vessel causing intracerebral bleeding, commonly in the basal ganglia, thalamus, pons, and cerebellum. The onset of hemorrhagic strokes can be both non-traumatic and traumatic in nature. Neurological deficits are dependent upon the site and severity of the hemorrhage and typically appear abruptly, developing over of a span of 30 to 90 minutes. The most severe deficits typically present within hours of onset, with gradual improve as edema subsides and extravascular blood is removed (Frizzell, 2005).

Pathophysiology of Spasticity

Spasticity is a form of hypertonia, or excess muscle tone, classically defined by Lance (1980) as an increased resistance to a passive stretch. The increased resistance is a result of a velocity-dependent increase in tonic stretch reflexes that occurs from hyper-excitability of the stretch reflex, which is one component of the upper motor neuron syndrome (Lance, 1980). In a 2001 interdisciplinary workshop sponsored by the National Institutes of Health, spasticity was defined as hypertonia with either one or both of the following present: (1) resistance to externally

imposed movement that increases with increasing speed of stretch and varies with the direction of joint movement or (2) resistance to externally imposed movement increases above a threshold speed or joint angle (Sanger et al., 2003). Other proposed mechanisms of spasticity include fusimotor neuron hyperactivity, hyperexcitable motor neurons, abnormal excitability of the spinal segmental and intersegmental interneurons from loss of supraspinal influences, as well as changes in the properties of the muscles themselves (Sehgal & McGuire, 1998). Newer research has refined this definition suggesting that spasticity is also a sensorimotor phenomenon with other alterations present in the central nervous system as part of the upper motor neuron syndrome (Ivanhoe & Reistetter, 2004). The velocity dependency in spasticity is a key distinguishing feature from other similar motor disorders in which there is changed resistance to passive movement at a joint, such as in the rigidity often present in people with Parkinson's disease. Normally, in an unperturbed central nervous system, any resistance observed in the full range of joint movement should be solely due to biomechanical factors and electromyography activity would not be elicited. In spasticity, however, there is damage to the corticospinal tracts, which results in an increase of stretch reflexes that are normally latent.

In essence, spasticity is manifested largely by overactive neural input to muscles, which causes excessive muscle contraction. This neural over-activity is a result of two underlying mechanisms: hyperreflexia and brainstem upper motor neuron over activity. In hyperreflexia, there is an absence of corticospinal inhibition onto lower motor neurons, which evokes a heightened reflexive response. The lack of corticospinal inhibition furthermore produces an excessive lower motor neuron response to muscle spindle input. Likewise, lesions that disinhibit the reticulospinal and/or vestibulospinal tracts result in brainstem overactivity (Ivanhoe & Reistetter, 2004). Spasticity becomes problematic when it interferes with postural alignment,

activities of daily living, sleep, or produces pain or discomfort (Lundy-Ekman, 2013). Clinically, spasticity has been delineated into both positive and negative symptoms. The conglomerate of positive and negative features of spasticity comprises the upper motor neuron syndrome. Positive aspects of spasticity include increased muscle tone, exaggerated stretch reflexes, positive Babinski sign, clonus, and flexor/extensor spasms. Negative features encompass loss of a particular functional capacity that is ordinarily controlled by the lesioned area of the brain, which include loss of strength, dexterity, and motor control (Biering-Sorensen, Nielsen, & Klinge, 2006). The intricate pathophysiology of spasticity has obscured how clinicians define and identify spasticity in the clinical setting.

The primary objective in clinical treatment approaches for spasticity management is premised on modifying the degree of muscle imbalance through weakening the contractile effects of involuntary muscle over activity in a given pattern in order to prevent irreversible soft-tissue changes and tendon contractures (e.g., flexor or extensor synergy). There are a range of both conservative treatments and interventional measures to achieve this, including occupational and physical therapies, oral and intrathecal medications, surgery, and focal chemical denervation with phenol, alcohol, and botulinum neurotoxin (BoNT).

Brunnstrom Recovery Stages

In 1970, Brunnstrom proposed sequential stages of motor recovery following stroke based on the degree of spasticity and voluntary movement. Each stage progression indicates less presence of spasticity and improved motor performance. Brunnstrom's recovery stages are the only stroke-specific assessment in tracking the progression of motor recovery following stroke. Six stages of motor recovery have been described. In stage one, flaccidity is present and no movements of the limbs can be initiated. In stage two, the basic limb synergies or some of their components may appear as associated reactions or minimal voluntary movement responses may

be present and spasticity begins to develop. In stage three, the patient gains voluntary control of the movement synergies, although full range of all synergy components does not necessarily develop and spasticity is severe. In stage four, some movement combinations that do not follow the synergies are mastered and spasticity begins to decline. In stage five, more difficult movement combinations are possible as the basic limb synergies lose their dominance over motor acts. Finally, in stage six, individual joint movements become possible (Brunnstrom, 1970). Brunnstrom's stages of recovery have been widely utilized by clinicians, giving rise to a multitude of standardized assessments used by both occupational and physical therapists in the assessment of motor performance following the onset of stroke.

III. Methods

Study Design

This was a prospective, cross-sectional research study in which all data was be collected during a single 2.5-hour experimental session. The Institutional Review Board at Northwestern University and the University of Wisconsin-Milwaukee approved the project.

Subjects

Stroke subjects were recruited from the Clinical Neuroscience Research Registry, a research database management system that is updated and maintained by clinicians at the Rehabilitation Institute of Chicago (RIC), and research flyers were distributed near the Milwaukee area.

Eligibility for participation in the study included any history of stroke without regard to time elapsed from onset, upper extremity weakness or loss of function, as well as diagnosed spasticity in the elbow flexors and extensors in adults between the ages of 18-85. Exclusion

criteria included presence of orthopedic impairment of the shoulder, elbow, wrist, or hand joints; pain in any of the aforementioned musculoskeletal regions, or severe cognitive deficits that limit ability to follow simple commands.

Clinical Assessment

Before the motion caption data collection, each subject's non-paretic and paretic arms was evaluated for clinical assessments including light touch sensation, isometric muscle strength, spasticity, motor function and overall health status following stroke.

Light touch sensation was measured at 3 locations on the arm using the Semmes-Weinstein monofilaments 5PC touch-test hand kit (2.83, 3.61, 4.31, 4.56, and 6.65) (Sammons Preston Rolyan, Germantown, WI) at the upper arm, forearm, and hand. Results from touch sensation were classified as *intact* if all sensation tests were normal; *impaired* if any of the tests indicated sensation loss; and *absent* if patients could not identify the largest monofilament at any of the testing locations.

Maximum isometric muscle strength was tested using the Lafayette hand-held muscle tester (Lafayette Instruments, Lafayette, IN) in 2 upper limb movements: elbow flexion and extension.

Severity of spasticity was assessed using the modified Ashworth Scale (MAS) (Bohannon & Smith, 1987). Each subject's elbow was quickly and passively stretched by an occupational therapist. Spasticity is manifested by a catch angle and resistance to passive stretch. The degree of spasticity was rated using ordinal response categories using a scale of 0 to 5, where "0" corresponds to no increase in muscle tone (no spasticity) and "5" indicating that the affected joint is highly rigid during flexion or extension (severe spasticity).

We evaluated the upper limb motor function using the Fugl-Meyer Assessment scale – upper extremity subscale (FM-UE) (Fugl-Meyer, Jaasko, Leyman, Olsson, & Steglind, 1975), a scale based on the theoretical framework of the Brunnstrom recovery scale describing the recovery phases following stroke and is widely used in both clinical and research settings. The FM-UE is composed of 33 tasks with a 3-point rating scale (0 to 2) that assesses reflex-activity (3 items), dynamic movement within flexor and extensor synergy patterns (15 items), wrist stability (5 items), hand function (7 items), and coordination (3 items). The score range of the FM-UE is from 0 to 66, with higher measures representing higher levels of functionality. The FM-UE subscale has excellent interrater reliability (r = 0.995-0.996) (Duncan, Propst, & Nelson, 1983; Gladstone, Danells, & Black, 2002).

The Stroke Impact Scale (SIS) was administered to subjects to evaluate how the stroke impacted the subject's health and life. The SIS is a 59-item measure that assesses eight different domains: strength (4 items), hand function (5 items), ADL/IADL (10 items), mobility (9 items), communication (7 items), emotion (9 items), memory and thinking (7 items), and participation/role function (8 items). Each item is rated in a 5-point Likert scale in terms of difficulty the patient has experienced in completing each item. An additional question asking the patient to rate the recovery from their stroke on a scale of 0-100 is included at the end of the questionnaire. The questionnaire has established test-retest reliability, ranging from adequate to excellent (ICC = 0.70-0.92, except for the emotion domain, ICC = 0.57). Inter-rater reliability is reported as excellent for the hand function (ICC = 0.82) and mobility domains (ICC = 0.80), adequate for strength (ICC = 0.61), ADL/IADL (ICC = 0.64), and the memory and thinking (ICC = 0.43) domains, and poor for the communication (ICC = 0.39), emotion (ICC = 0.17), and

social participation (ICC = 0.29) domains (Carod-Artal, Ferreira Coral, Stieven Trizotto, & Menezes Moreira, 2009; Duncan et al., 1999).

Experimental Setting

The Vicon motion capture system (7 Vicon MCam2 Cameras with 1.3 Megapixels) was used to record the motion of reflective markers affixed to the upper arms and trunk. Subjects were instructed to sit on a stationary chair with neither back nor arm support. The predominant head axis of the global coordinate system, the Y axis, is defined as the forward facing direction (front-back). The secondary X axis is the lateral axis from right to left, orthogonal to the Y axis. The third Z axis follows the right-thumb rule, pointing up (vertical to the floor) and is orthogonal to both the X and Y axes. Kinematic data was recorded at a sampling rate of 120 Hz.

Reflective Marker Placement

The Vicon Plug-In-Gait upper body model (see Appendix 1) for each subject encompassed the trunk, upper extremities, and head, and was made using twenty-nine spherical reflective markers. The markers were attached to the body using double-sided adhesive tape. Bilaterally, markers will be placed on the acromion, inferior angle of the scapula, lateral humerus, lateral epicondyle of the humerus, forearm, wrist (radial and ulnar aspects), and the base of index finger. The trunk is defined at C7 of the cervical column, T10 of the thoracic spine, the jugular notch, the sternum, and the sacrum. The hips are bilaterally defined at the anterior superior and the posterior superior iliac spine. Markers defining the bilateral anterior and posterior head will be fixed to a head band that each subject will wear.

Electromyography (EMG) Sensor Placement

A total of four EMG sensors (Delsys, Natick, MA) were affixed with adhesive tape bilaterally to the muscle bellies of the biceps and triceps of both the affected and unaffected arm

to record muscle activity. EMG placement sites were cleaned with alcohol wipes to remove dead skin cells and maintain signal strength. These two sites were selected to assess the presence of flexor or extensor spasticity, specifically.

Tasks during Motion Capture

A standard protocol was administered to each subject, and both sides of the upper body were tested. The contralateral side (non-paretic) was precedent, and subjects were told to attempt to duplicate each task with the paretic arm.

Active range of motion (AROM). The clinician instructed the subject to attempt to move the paretic arm into all arm planes of movement to assess AROM. Ranges at the shoulder joint (shoulder flexion), elbow joint (elbow flexion/extension), and wrist joint (wrist flexion/extension) were calculated.

Passive stretch of the elbow. The clinical passive stretching test was performed by the same occupational therapist who initially evaluated the subjects' spasticity. The catch angle (in degrees) was defined as the elbow position at which the therapist initially encountered resistance of the paretic arm. The kinematic data corroborated this position, for the position was also the point at which elbow angular velocity suddenly declined. The resistance to passive stretching was quantified by the stretch duration (in seconds) that the therapist needed to complete a single stretching test: from maximum elbow flexion to full extension (a test of flexor spasticity), and from full extension back to maximum flexion (a test of extensor spasticity).

Finger-to-Nose (F-to-N) Reaching Task. Reaching is the most essential movement of upper limb function involving the shoulder and elbow. Reaching performance is critical since the arm needs to be placed in the desired position to support hand activities, such as grasping and manipulating objects. A rhythmic F-to-N reaching task was used to assess the overall arm

function. For this task, a height-adjustable target was positioned at the subject's shoulder height, oriented at the center of the body, and at about 90% of the extended arm length. Subjects were instructed to touch the target with their index fingers and then touch their noses as many times as possible within a 30s period. One repetition of F-to-N is the movement from the nose to the target and then back to the nose. We define several variables of interest. The relative completeness of the reaching movement, which we will call the path ratio (PR), is the ratio of the paretic arm's maximal reaching distance in the Y-axis from nose to target to that of the contralateral arm. Maximum path ratio will be set to 1. Efficiency of arm movement is as the total time needed to complete 5 repetitions of the F-to-N movement.

Note that the experiment setup for the case study at University of Wisconsin-Milwaukee mirrored that of the experimental setup at the Rehabilitation Institute of Chicago except the following minor differences. The Vicon motion capture system (6 Vicon Bonita Cameras with 16 Megapixels) was used to record the motion of reflective markers. In addition, the clinician placed and held a mini force sensor on the medial and lateral aspects of the subject's wrists to collect the level the force exerted on the upper limb tested during administration of the passive stretch. Figure 2 shows the experimental setup, sensor placements, and beginning and end positions when performing the passive stretching during the motion capture at the University of Wisconsin-Milwaukee site.

Analysis

The motion-caption data was digitally low-pass filtered at 6Hz with a 4th-order finite-impulse response filter to attenuate high-frequency noise without altering the signal phase. To examine the relationships between the MAS and selected variables, we first inspected the scatter plots (a) between the MAS and residual impairments (active range of motion of shoulder flexion,

elbow, and wrist, and muscle strength of the elbow flexion and extension), (b) between the MAS and functional limitations as measured by Fugl-Meyer upper extremity assessment, finger to nose movement, and ability to grasp a bottle of water, and (c) between the MAS and overall health status following stroke as measured by the Stroke Impact Scale. Pearson correlation coefficients and Spearman correlation coefficients were computed. One-way ANOVA will be used to compare the overall mean differences of the selected variables by MAS ratings. The significant level was set at 0.05. Post-hoc analysis will not be used as follow-up procedures due to small sample size per MAS ratings.

To inspect whether there are potential kinematic values or physiological responses that can be used to identify the characteristics of the passive stretch (passive stretch duration, catch angle, electromyography response), a case study was used to compare the performance between affected arm and unaffected arm during passive stretch. For this thesis, Vicon data was processed following the Plug-In-Gait model within the Nexus software. The kinematic data was graphed with the elbow joint angle as a function of time. Both EMG data and force data are presented as raw data to inspect the differences between the affected arm and unaffected arm during passive stretch.

IV. Results

Relationship between MAS and residual impairments. Figure 3 shows the scatterplot between MAS scores and residual impairments (active range of motion of shoulder, elbow and wrist, and muscle strength of elbow flexion and extension). Overall, results showed that stroke subjects who had more severe spasticity tended to have reduced range of motion at the shoulder (flexion) (Pearson correlation coefficient $r_p = -.601$; Spearman correlation coefficient $r_s = -.607$),

elbow (r_p = -.436; r_s = -.495) and wrist (r_p = -.206; r_s = -.305) joints, as well as reduced muscle strength for elbow flexion (r_p = -.547; r_s = -.618). The relationship between the MAS scores and the muscle strength for elbow extension was weak (r_p = -.160; r_s = -.191).

Relationship between MAS and functional limitations. Figure 4 summarizes the results of the relationship between the MAS scores and the FM-UE subscale. Analysis between the FM-UE subscale and MAS revealed a significant negative correlation. The strongest correlation occurred between the FM-UE total score (r_p = -0.817; r_s = -0.806), while the weakest correlation amongst all subscales occurred between coordination subscale (r_p = -0.696; r_s = -0.684). A one-way, between-subjects design ANOVA showed significant mean differences between MAS scores and all FM-UE subscales: the FM-Arm subscale ($F_{4,15}$ = 17.4, $p < .001$), the FM-Wrist subscale ($F_{4,15}$ = 4.3, $p < 0.016$), the FM-Hand subscale ($F_{4,15}$ = 4.8, $p < 0.011$), the FM-Corr subscale (F4,15 = 4.4, $p < 0.015$) as well as FM-Total Score subscale ($F_{4,15}$ = 12.6, $p < 0.001$). Overall, there was a tendency for increased levels of spasticity per scoring of the MAS to result in decreased motor performance as measured by the FM-UE subscale.

Relationship between MAS and overall health status. Figure 5 demonstrated the relationship between the MAS scores and overall health status per the Stroke Impact Scale subscales. There was a moderate negative correlation between MAS score and the hand subscale (r_p = -0.543; r_s = -0.576), indicating that a higher MAS score may be indicative of the magnitude of impairment in the hand. No significant relationships were demonstrated between the remaining subscales of the Stroke Impact Scale, suggesting that there is little to no relationship between MAS scores and overall health status.

Case Study. By inspecting the motion capture raw data, there was a marked increase in the EMG response when the subjects affected limb is stretched into full elbow extension (Figure

6, supplement with one Video clip), such phenomenon was not observed when stretching the un-affected limb.

V. Discussion

The strong correlation between MAS scores and the residual impairments as well as the FM-UE subscale suggests that a higher MAS score may be indicative of the general stage of motor recovery following incurrence of a stroke. However, the severity of spasticity may not predict an individual's overall health status as measured using the Stroke Impact Scale.

To the best of our knowledge, there is no previous literature explicitly examining the correlation between the MAS and the Fugl Meyer Assessment and Stroke Index Scale. However, previous studies have ascertained similar results regarding the MAS and residual upper extremity impairment (Denham, 2008). Previous studies have proposed that effectively operationalizing the definition of quality of life and overall health status could contribute to more objective measurement of the relationship between spasticity management and health status and quality of life (Gianino JM, York MM, Paice JA, et al., 1998).

While results from this study were variable, it was evident that there were several significant correlations between MAS scores and functional impairments, as well as neurophysiological variables such as EMG through inspection of raw EMG data and the motion capture videos. One previous study, however, confounds these findings. Alibiglou et al. (2008) investigated the quantitative measures of spasticity and their relationship to the MAS demonstrated a lack of significant correlation between the MAS and quantitative measures of stroke including neural and muscular components (Alibiglou et al., 2008). In short, the study reported that the MAS does not provide reliable information about quantitative measures

associated with spasticity or about its contributing components. Because spasticity is an intricate neurophysiological phenomenon and the MAS does not have adequate objectivity in accurately quantifying clinical assessment of spasticity, it is clear that the inconspicuous results in this previous study and others alike are faced with an aggregate of challenges in validly and reliably quantifying spasticity.

The results gleaned from the case study suggested that there is significance in the amount of spasticity present and the magnitude of EMG produced during passive stretching. Though we were unable to clearly examine the effect of velocity and force on the amount of produced resistance during passive stretching, it is likely that increased force and velocity is conducive to an increased tonic reflex (catch angle) and resistance force through passive stretching.

Future directions of research to examine the MAS and functional impairments through using motion capture and EMG should increase the case study into case series by collecting stroke subjects within each MAS strata to more closely inspect these quantitative measures and produce more clear results.

VI. Conclusions

The strong correlation between MAS scores and the residual impairments as well as the FM-UE subscale suggests that a higher MAS score may be indicative of the general stage of motor recovery following incurrence of a stroke. Additionally, there was a marked increase in EMG activity through passive stretching of the affected limb into full elbow extension; conversely, such a phenomenon was not observed in the unaffected limb.

Limitations and Potential Contributions

Because this particular study has no specificity in regard to the amount of time elapsed following the onset of stroke, it may be difficult to generalize the study's findings to a large population because spasticity can evolve over the course of time. Because functional recovery following strokes typically occurs during the first three months following onset, it would be important to include a more heterogeneous group of participants for future studies. Stroke generally affects older adults, however, there has been a growing trend of younger people incurring stroke, and the sample size does not account for this emerging trend. Furthermore, the modest sample size further constrains the amount of generalizability. The use of two force sensors instead of one as well as increasing the amount of motion capture cameras to minimize the gaps in the data frames could also increase objectivity and fidelity. Utilizing an organic motion capture system and more motion capture cameras could additionally improve the findings from this study. Finally, incorporating an independent MAS clinical assessment evaluator (blind assessor) could improve the reliability of this study.

This study has the potential to reduce the subjectivity of the MAS and translate it into functional values, a highly prominent clinical assessment for spasticity in stroke patients and other populations where spasticity is present such as individuals with stroke, multiple sclerosis, cerebral palsy, traumatic brain injury, or spinal cord injury. Reducing the subjectivity will yield an increased ability for the clinician to appropriately address spasticity treatment, track progress in treatment plans, and more clearly illustrate therapeutic outcomes. Additionally, accurate measurement of spasticity as a result eradication of subjective MAS components will enable clinical researchers to more easily illustrate efficacy of studies examining novel therapeutic or

pharmacological treatment strategies for persons with spasticity while using the MAS as an outcome measure.

TABLES/FIGURES

Table 1. Ashworth Scale and Modified Ashworth Scale.

Score	Ashworth Scale (1964)	Modified Ashworth Scale Bohannon & Smith (1987)
0 (0)	No increase in tone	No increase in muscle tone
1 (1)	Slight increase in tone giving a catch when the limb was moved in flexion or extension	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
1+ (2)		Slightly increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM (range of movement)
2 (3)	More marked increase in tone but limb easily flexed	More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved
3 (4)	Considerable increase in tone – passive movement difficult	Considerable increase in muscle tone – passive movement difficult
4 (5)	Limb rigid in flexion or extension	Affected part(s) rigid in flexion or extension

Abbreviations: ROM=range of movement

Table 2. Subject demographics from Rehabilitation Institute of Chicago data collection sample (n = 20)

	Min	Max	Mean	SD
Age (years)	27.0	70.0	57.4	10.3
Time Since Onset of Stroke (months)	12.0	433.0	151.8	113.9
Height (m)	1.60	1.93	1.73	8.9
Weight (kg)	50.0	135.0	80.3	17.2
BMI (kg/m^2)	18.8	36.0	26.7	4.3

Abbreviations: SD = standard deviation; min = minimum; max = maximum; BMI = body mass index as calculated by the subject's weight in kilograms (kg) divided by the square of height in meters (m).

Table 3. Relationship between MAS scores and residual impairments.

	N	Mean	SD	Pearson (r_p) and Spearman's (r_s) Correlation	ANOVA
Shoulder Flexion (º)	19	99.8	37.655	$r_p = -.601$ $r_s = -.607$	$F_{4, 14} = 5.8$ $p < 0.006$
Elbow Flexion (º)	19	53.7	16.223	$r_p = .436$ $r_s = .495$	$F_{3, 15} = 1.4$ $p < 0.287$
Wrist Flexion (º)	17	-44.0	28.131	$r_p = .206$ $r_s = .305$	$F_{3, 13} = .427$ $p < 0.737$
Elbow Flexion (kg)	19	7.6	5.178	$r_p = -.547$ $r_s = -.618$	$F_{3, 15} = .3$ $p < 0.844$
Elbow Extension (kg)	17	10.0	3.528	$r_p = -.160$ $r_s = -.191$	$F_{3, 13} = .427$ $p < 0.737$

Table 4. Relationship between MAS and FM-UE subscales.

		N	Mean	SD	Pearson (r_p) and Spearman's (r_s) Correlation	ANOVA
FM-Arm	0	5	32.2	2.8636	r_p = -.815	$F_{4,15} = 17.4$, $p < .001$
	1	5	26.6	6.6558	r_s = -.829	
	2	1	5	.		
	4	7	15	5.164		
	5	2	5.5	3.5355		
	Total	20	20.75	10.6468		
FM-Wrist	0	5	7	4.4721	r_p = -.696	$F_{4,15} = 4.3$, $p < 0.016$
	1	5	5.8	4.0866	r_s = -.754	
	2	1	0	.		
	4	7	0.571	1.5119		
	5	2	0	0		
	Total	20	3.4	4.26		
FM-Hand	0	5	9.8	4.7645	r_p = -.714	$F_{4,15} = 4.8$, $p < 0.011$
	1	5	8.2	4.3243	r_s = -.728	
	2	1	1	.		
	4	7	2.286	2.7516		
	5	2	0.5	0.7071		

	Total	20	5.4	5.0409		
FM-Corr	0	5	3.8	2.2804	$r_p = -.696$	$F_{4,15} = 4.4$, $p < 0.015$
	1	5	3	1.8708	$r_s = -.684$	
	2	1	0	.		
	4	7	0.571	0.9759		
	5	2	0	0		
	Total	20	1.9	2.1497		
FM-Total	0	5	52.8	13.0652	$r_p = -.817$	$F_{4,15} = 12.6$, $p < 0.001$
	1	5	43.6	14.1174	$r_s = -.806$	
	2	1	6	.		
	4	7	18.429	7.5246		
	5	2	6	2.8284		
	Total	20	31.45	20.4874		

Abbreviations: FM-UE = Fugl-Meyer upper extremity; SD = standard deviation.

Table 5. Relationship between MAS scores and overall health status.

	N	Mean	SD	Pearson (r_p) and Spearman's (r_s) Correlation	ANOVA
Subscale 1 Strength	19	12.9	2.7501	$r_p = -.186$ $r_s = -.281$	$F_{4,14} = 3.1$, $p < 0.053$
Subscale 2 Cognition	19	32.9	2.6893	$r_p = .007$ $r_s = .064$	$F_{4,14} = 0.2$, $p < 0.925$
Subscale 3 Emotion	19	34.7	6.7521	$r_p = -.009$ $r_s = .073$	$F_{4,14} = 0.5$, $p < 0.724$
Subscale 4 Speech	19	34.3	1.1471	$r_p = .081$ $r_s = .223$	$F_{4,14} = 0.2$, $p < 0.944$
Subscale 5 ADL	19	40.8	5.7547	$r_p = .083$ $r_s = .114$	$F_{4,14} = 0.4$, $p < 0.819$
Subscale 6 Walk	19	40.9	3.3177	$r_p = -.190$ $r_s = -.203$	$F_{4,14} = 2.5$, $p < 0.094$
Subscale 7 Hand	19	12.7	6.7646	$r_p = -.543$ $r_s = -.576$	$F_{4,14} = 2.4$, $p < 0.097$
Subscale 8 Social	19	34.1	5.227	$r_p = .170$ $r_s = .268$	$F_{4,14} = 0.2$, $p < 0.939$
Total Score	19	2432.1	20.9858	$r_p = -.162$ $r_s = -.188$	$F_{4,14} = 0.9$, $p < 0.506$

Abbreviations: SD = standard deviation.

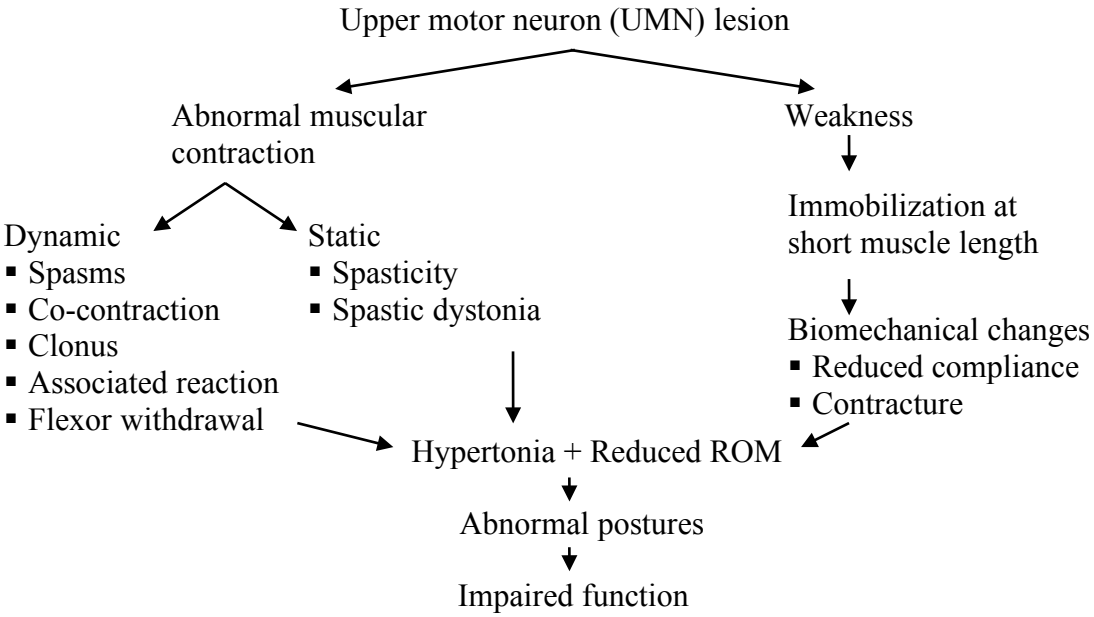


Figure 1. A model of the interaction between neural and biomechanical components of hypertonia in the upper motoneuron syndrome (Barnes & Johnson, 2001)

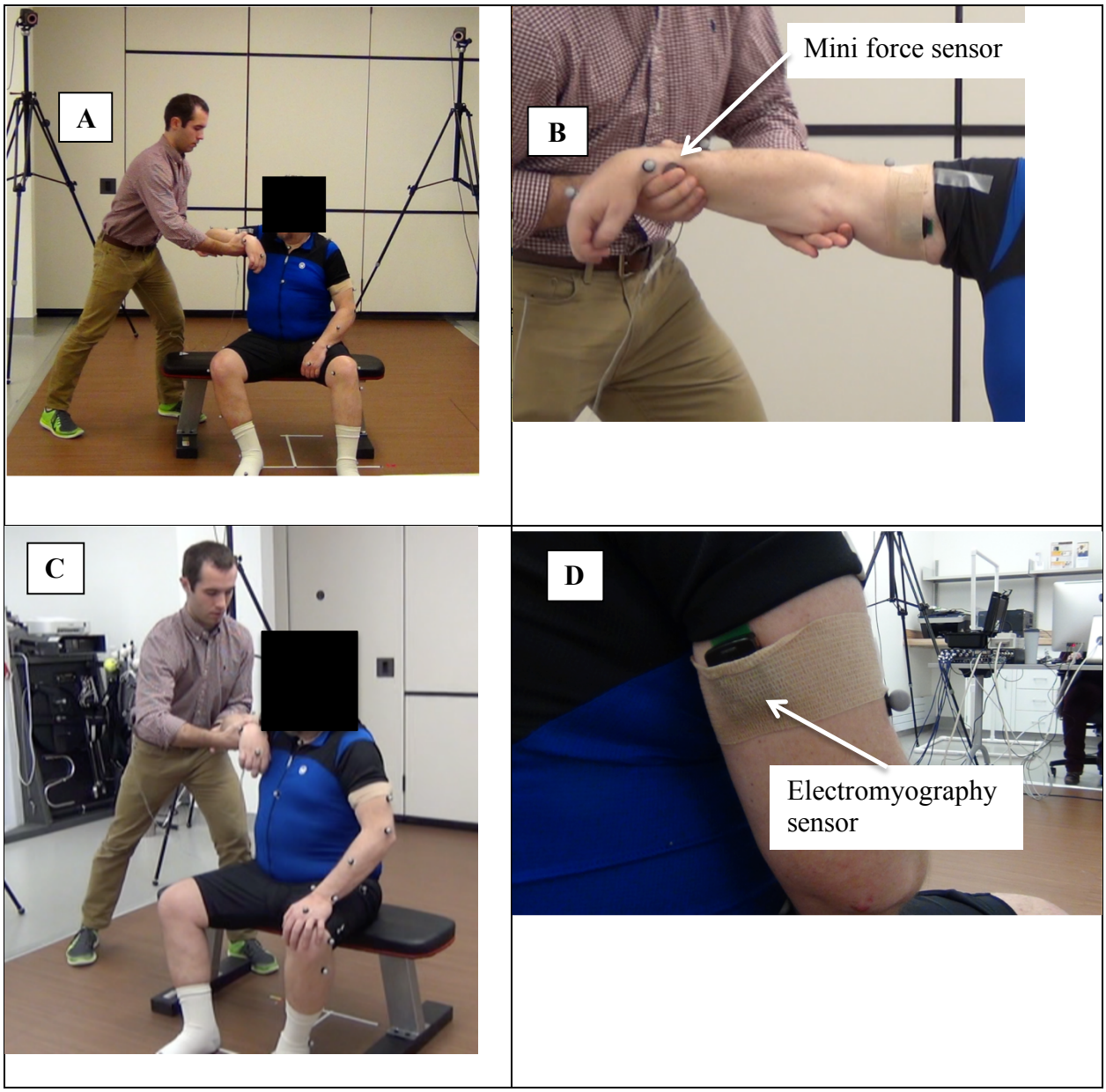


Figure 2. Motion capture experimental set-up at the University of Wisconsin-Milwaukee Innovation Accelerator motion analysis laboratory

Figure 2A. The beginning position of the MAS is displayed. The clinician supports the spastic upper extremity while passive stretching is initiated to assess for flexor/extensor spasticity. 2B. The clinician places the mini force sensor on the medial aspect of the wrist during passive stretching. 2C. Placement of reflection motion capture sensors at various anatomical landmarks of the upper extremity. 2D. Placement of the electromyography sensor on the triceps. An electromyography sensor will also be placed on the muscle belly of the biceps.

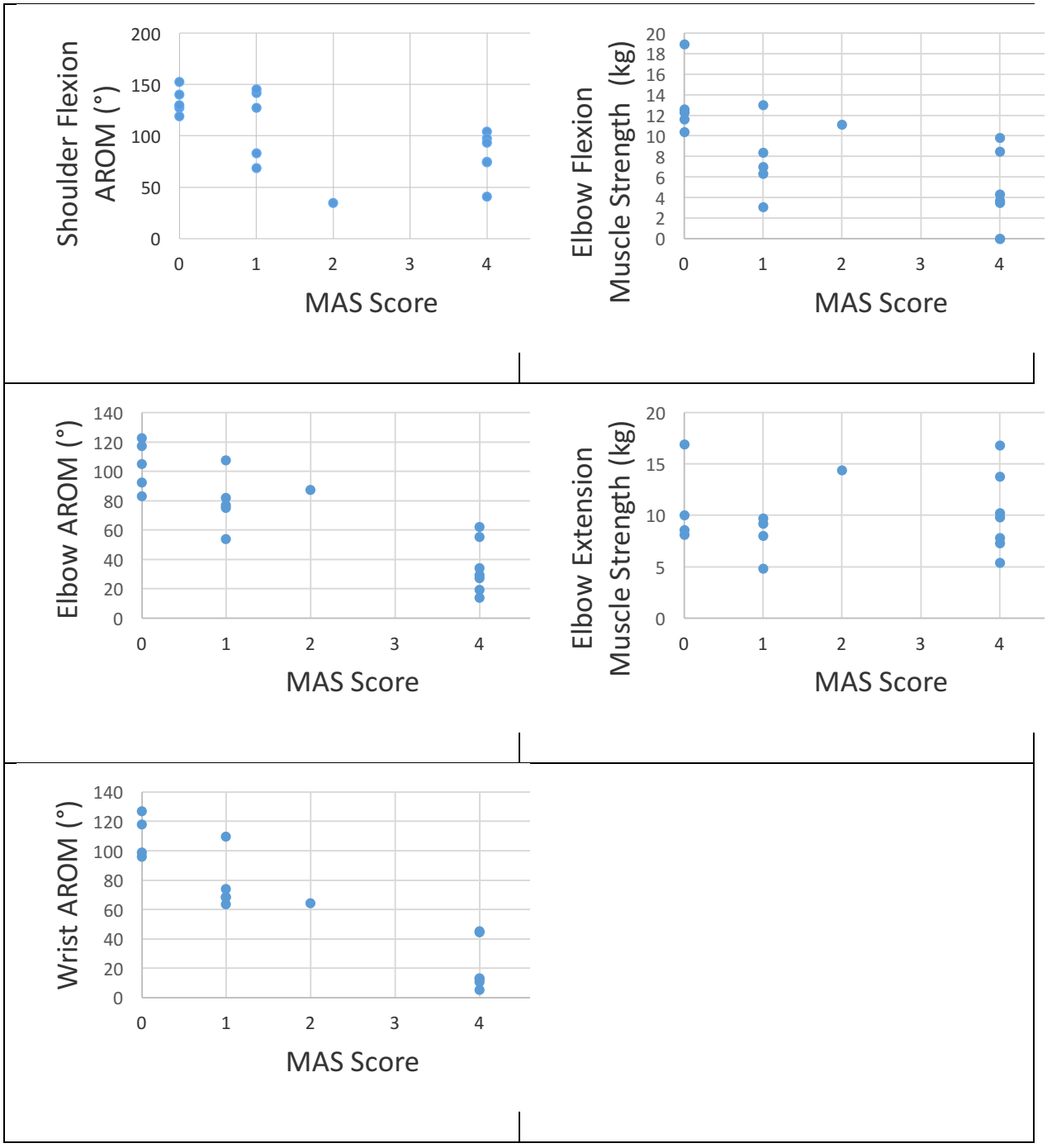


Figure 3. Relationship between Modified Ashworth Scale and residual impairments (active range of motion of shoulder flexion, elbow, and wrist, and muscle strength of elbow flexion and extension).

Abbreviations: AROM = active range of motion; MAS = Modified Ashworth Scale.

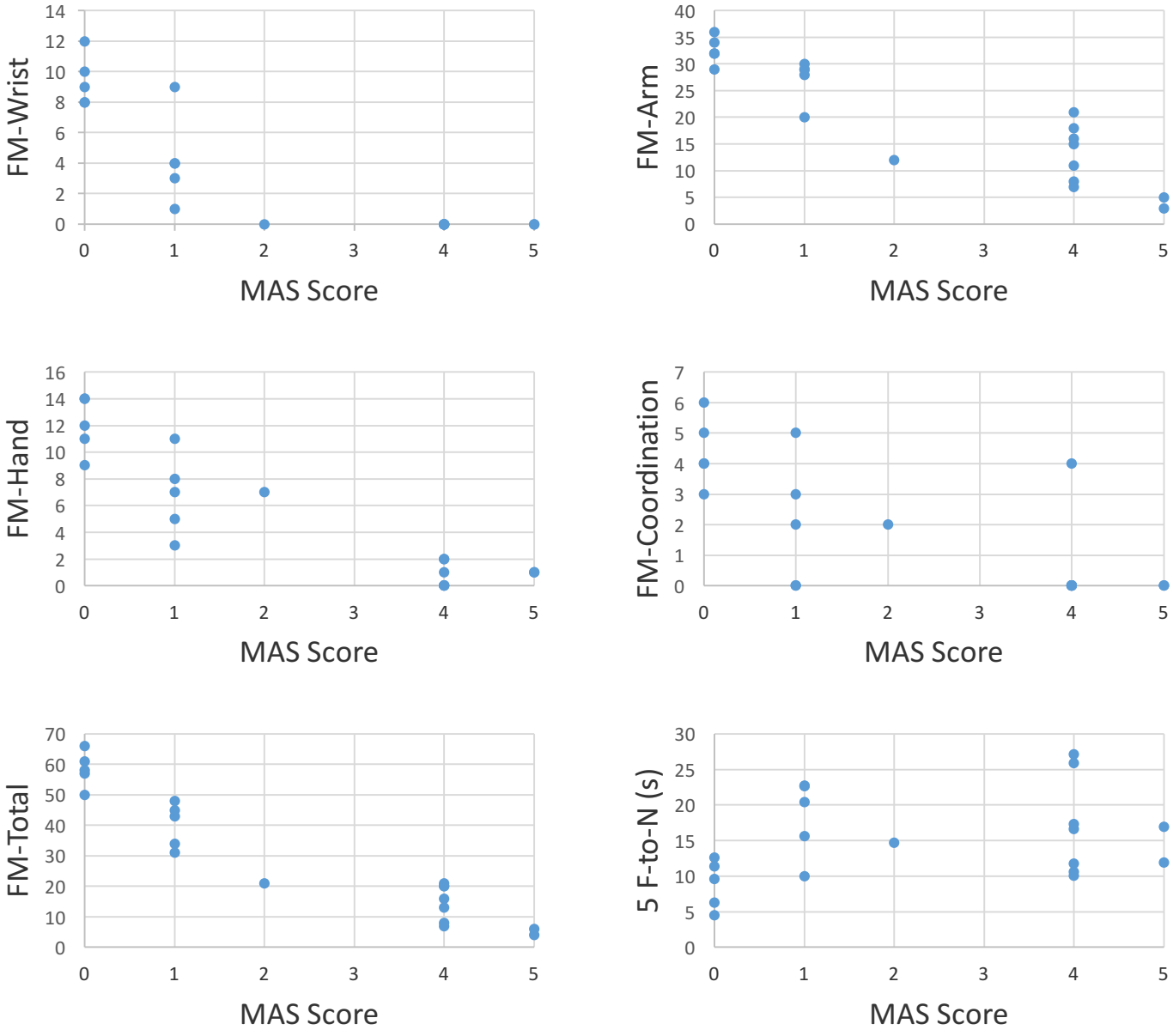
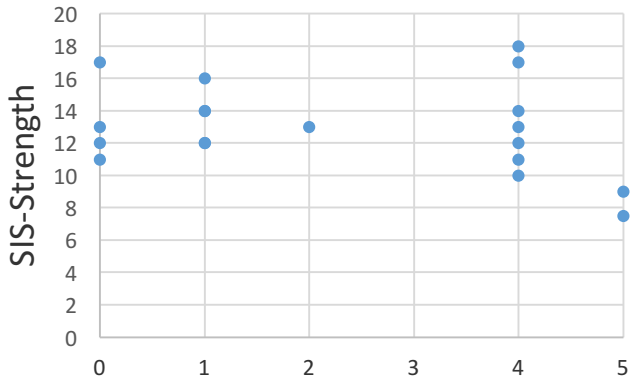
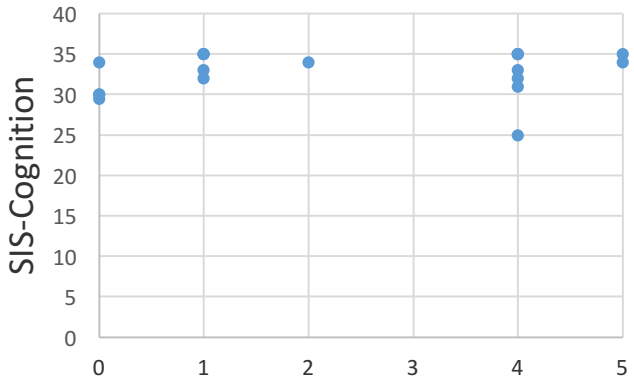
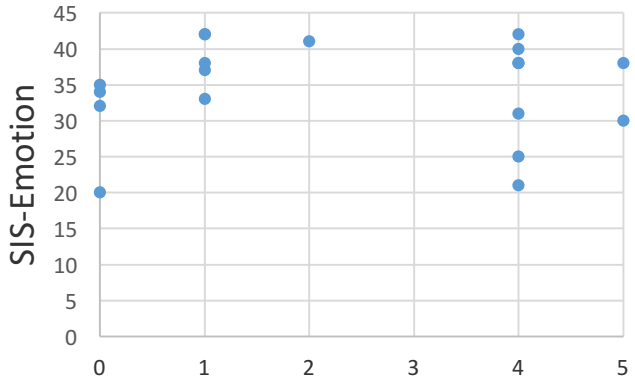


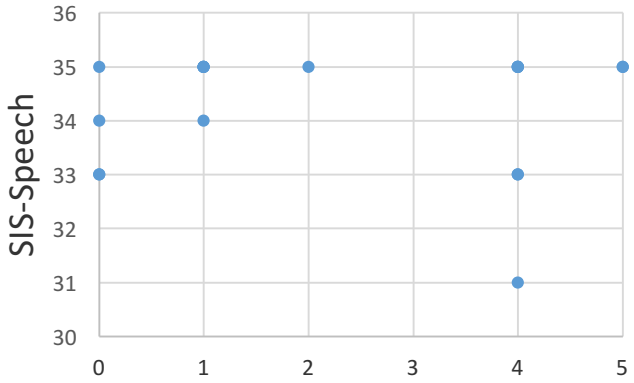
Figure 4. Relationship between MAS and functional limitations as measured by Fugl-Myer upper extremity assessment, finger to nose movement, and ability to grasp a bottle of water.

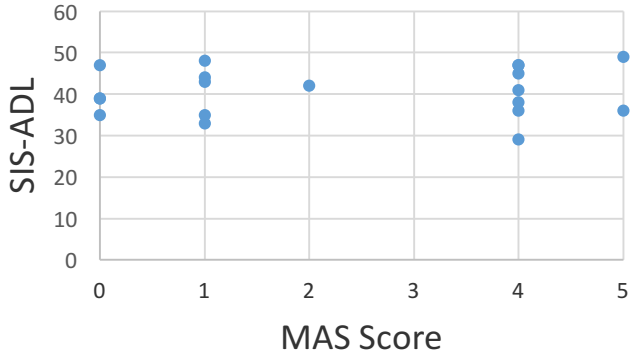
Abbreviations: FM = Fugl-Meyer upper extremity assessment subscale; MAS = Modified Ashworth Scale; F-to-N = finger-to-nose.

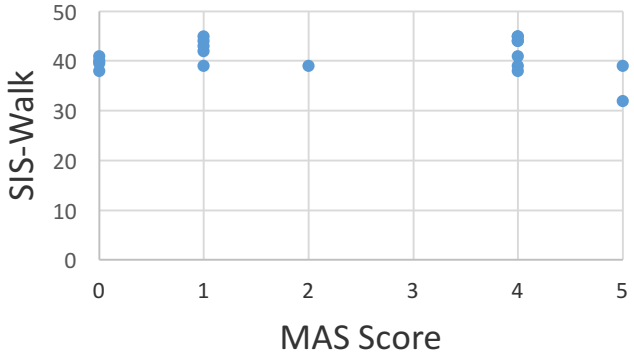

SIS-Strength
20
18
16
14
12
10
8
6
4
2
0
0 1 2 3 4 5

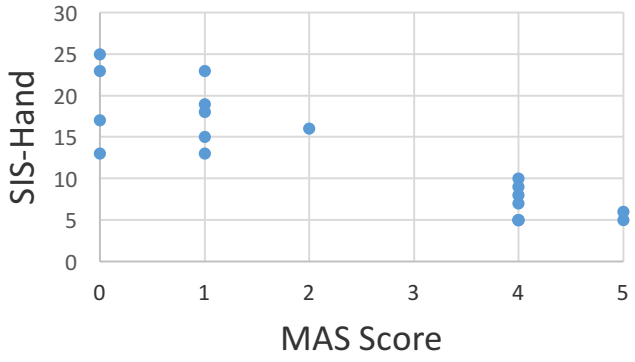

SIS-Cognition
40
35
30
25
20
15
10
5
0
0 1 2 3 4 5

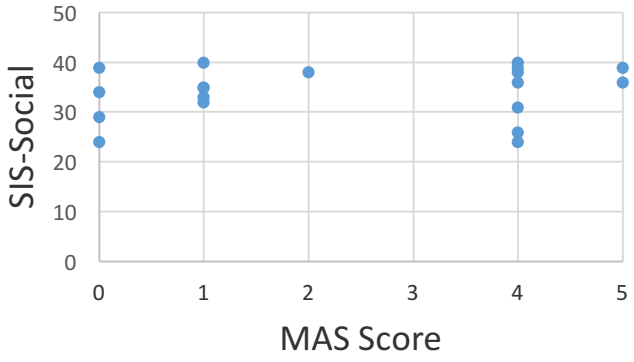

SIS-Emotion
45
40
35
30
25
20
15
10
5
0
0 1 2 3 4 5


SIS-Speech
36
35
34
33
32
31
30
0 1 2 3 4 5


SIS-ADL
60
50
40
30
20
10
0
0 1 2 3 4 5
MAS Score


SIS-Walk
50
40
30
20
10
0
0 1 2 3 4 5
MAS Score


SIS-Hand
30
25
20
15
10
5
0
0 1 2 3 4 5
MAS Score


SIS-Social
50
40
30
20
10
0
0 1 2 3 4 5
MAS Score

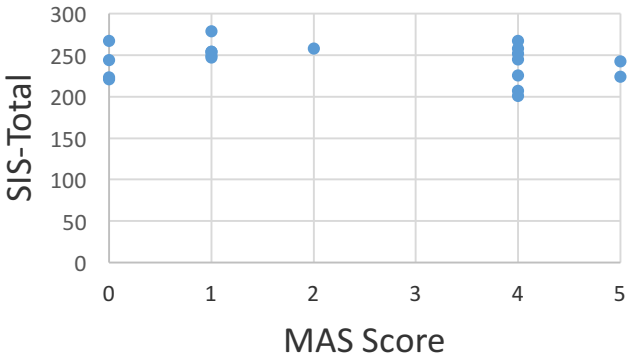


Figure 5. Relationship between MAS and overall health status following stroke as measured by the Stroke Impact Scale subscales.

Abbreviations: SIS = Stroke Impact Scale (the SIS is a 59-item measure with 8 different domains wherein each subject rated each item on a 5-point Likert scale in terms of the difficulty they experienced in completing each item; summative scores were generated for each domain with a range of 0-100 with a higher score indicating less difficulty); ADL = activities of daily living.

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Appendix

Plug-in-Gait Marker Placement

Head Markers		
LFHD	Left front head	Located approximately over the left temple
RFHD	Right front head	Located approximately over the right temple
LBHD	Left back head	Placed on the back of the head, roughly in a horizontal plane of the front head markers
RBHD	Right back head	Placed on the back of the head, roughly in a horizontal plane of the front head markers
Torso Markers		
C7	7th Cervical Vertebrae	Spinous process of the 7th cervical vertebrae
T10	10th Thoracic Vertebrae	Spinous Process of the 10th thoracic vertebrae
CLAV	Clavicle	Jugular Notch where the clavicles meet the sternum
STRN	Sternum	Xiphoid process of the Sternum
RBAK	Right Back	Placed in the middle of the right scapula. This marker has no symmetrical marker on the left side. This asymmetry helps the autolabeling routine determine right from left on the subject.
Arm Markers		
LSHO	Left shoulder marker	Placed on the Acromio-clavicular joint

LUPA	Left upper arm marker	Placed on the upper arm between the elbow and shoulder markers. Should be placed asymmetrically with RUPA
LELB	Left elbow	Placed on lateral epicondyle approximating elbow joint axis
LFRA	Left forearm marker	Placed on the lower arm between the wrist and elbow markers. Should be placed asymmetrically with RFRA
LWRA	Left wrist marker A	Left wrist bar thumb side
LWRB	Left wrist marker B	Left wrist bar pinkie side
LFIN	Left fingers	Actually placed on the dorsum of the hand just below the head of the second metacarpal
Pelvis		
LASI	Left ASIS	Placed directly over the left anterior superior iliac spine
RASI	Right ASIS	Placed directly over the right anterior superior iliac spine
LPSI	Left PSIS	Placed directly over the left posterior superior iliac spine
RPSI	Right PSIS	Placed directly over the right posterior superior iliac spine
Leg Markers		
LKNE	Left knee	Placed on the lateral epicondyle of the left knee

LTHI	Left thigh	Place the marker over the lower lateral 1/3 surface of the thigh, just below the swing of the hand, although the height is not critical.
LANK	Left ankle	Placed on the lateral malleolus along an imaginary line that passes through the transmalleolar axis
LTIB	Left tibial wand marker	Similar to the thigh markers, these are placed over the lower 1/3 of the shank to determine the alignment of the ankle flexion axis
LTOE	Left toe	Placed over the second metatarsal head, on the mid-foot side of the equinus break between fore-foot and mid-foot
LHEE	Left heel	Placed on the calcaneous at the same height above the plantar surface of the foot as the toe marker