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Upper limb robotic assessment: Pilot study comparing velocity dependent resistance in individuals with acquired brain injury to healthy controls

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Abstract

Introduction: Assessment of velocity dependent resistance (VDR) can provide insights into spasticity in individuals with upper motor neuron syndrome. This study investigates the relationship between Modified Ashworth scores and a biomechanical based representation of VDR using a rehabilitation robot. Comparisons in VDR are made for the upper limb (UL) between individuals with acquired brain injury and healthy controls for the para-sagittal plane.

Methods: The system manipulates the individual's limb through five flexion and extension motions at increasing speeds to obtain force profiles at different velocities. An approximation of VDR is calculated and analyzed statistically against clinical scales and tested for interactions.

Results: All individuals (aged 18–65), including healthy controls exhibited VDR greater than 0 ($P < 0.05$). MAS scores were found to be related to VDR ($P < 0.05$) with an interaction found between MAS Bicep and Tricep scores ($P < 0.01$). Considering this interaction, evidence of differences in VDR were found between several neighboring assessment score combinations.

Conclusion: The robot can detect and quantify VDR that captures information relevant to UL spasticity. Results suggests a better categorization of VDR is possible and supports further development of rehabilitation robotics for assisting spasticity assessment.

Keywords

Spasticity, assessment therapy, rehabilitation devices, motion/posture analysis, outcome measurement, robot-assisted rehabilitation

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Introduction

Spasticity is a neurophysiological phenomenon and component of the upper motor neuron (UMN) syndrome. It affects individuals with neurological injuries including multiple sclerosis, spinal cord injury, and stroke.[1,2] Characterized by Lance as "a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex",[3] spasticity impacts both, individuals with UMN syndrome as well as their caregivers. As spasticity severity is directly linked to progress in rehabilitation, it is closely monitored as clinical assessments influence treatment decisions. There exists, however, criticisms of spasticity measurement including definition, objectivity, and sensitivity.[4–7]

The increased resistance to passive movement possesses biomechanical and neurological components.[1]

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The biomechanical component changes with the muscles' physical properties and is not directly related to spasticity, whereas the neurological component does change with the hyperexcitability of the stretch reflex. The presence of both components introduces confounding when attempting to measure resistance directly from the limb. Frontline healthcare professionals, however, must regularly perform these direct measures to observe changes and apply required interventions.[8–12] Despite being a neurophysiological phenomenon, spasticity *can* be indirectly judged by quantifying resistance to passive movement in very controlled circumstances. While manually administered clinical measures possess challenges,[9] tools considering velocity with resistive force have been tested.[13–15] Force transducers[13,14] have successfully differentiated between impaired and unimpaired limbs in individuals with chronic stroke, however, evidence of velocity-dependent resistance (VDR) was not captured.

Pandyan et al.[13] concludes this lack of VDR characteristics suggests inconsistencies with the accepted neurophysiological definitions of spasticity. Motorized systems for wrist flexion/extensions[16] and lower limb joints[15,17] typically collect both biomechanical and electromyography (EMG) data. The EMG-based studies, however, have produced conflicting results regarding activation and resistance.[4,14,18] While EMG provides simple and insightful information for specific muscles, alternatives techniques are valuable due to the invasive nature of needle EMG, placement and property issues of surface EMG, and the additional skills and resources required as compared to the traditional clinical measures.[19–21]

While acknowledging component confounding, applying rehabilitation robotics can enforce controlled motions to assist VDR quantification, which may help advance towards standardized evaluations.[22] Assessment-focused rehabilitation robotics often concentrated on upper limb (UL) strength, range of motion, and function,[5,23–26] or lower limb spasticity.[15,17,27] Despite a robot's natural attributes to repeatably control motions and quantifiably measure position, velocity, and force, UL spasticity assessment studies are limited.[26,27] Building upon previous works,[26,28,29] the objective of this study, with more participants, was to validate the use of a robotic system to assist spasticity assessment using a biomechanical VDR metric. Data is collected for parasagittal elbow flexion/extension; a specific motion of functional relevance for activities of daily living (ADL) and subsequent clinical utility in rehabilitation centres. To relate forces to clinical scores, measurement is based on the direct resistance applied to a sensor at the same point of contact a healthcare professional typically uses when performing clinical assessments.

To establish that the system can detect and represent VDR in a meaningful way, the data is compared against existing clinical scales. One objective is to develop a continuous valued VDR metric as opposed to a discreet component set or a multi-dimensional vector.[27] Research questions for this first principle study are: i) Whether a relationship exists between the biomechanical VDR metric and clinical scores ii) whether an interaction exists between Bicep and Tricep assessments, and iii) whether differences in VDR values exist between healthy controls and individuals with acquired brain injury (ABI).

Methods

Participants

Eligible participants were recruited from an in-patient ABI tertiary rehabilitative program in Canada. All clinical participants were experiencing symptoms of sub-acute to chronic ABI. Inclusion criteria regarding participants were: (1) between 18 and 65 years of age, (2) greater than 10 weeks post injury, and (3) possessed Modified Ashworth Scale (MAS) scores between 0–3.

Participants were excluded if they demonstrated behavioural issues or other pain that prevented safe use of the robot. Individuals with MAS 4 were excluded as their resistive force would exceed safety limits. Prior to study participation, informed consent was collected from the participant or their guardian by a clinician not associated with the study. Healthy controls were a sample of convenience from the hospital and university. All healthy controls were between the ages of 28–65, in good condition, and were not experiencing medical issues. Data collected and output by the system would be transformed into a metric and used as an outcome measure to be statistically analyzed. This study was approved by the Hamilton Integrated Research Ethics Board (REB) of the hospital (12–347) and the University of Guelph REB (12SE020).

Assessment procedures

For all individuals with ABI, the MAS for elbow flexion and extension was first performed by a physiotherapist as it is the most commonly used clinical measure for spasticity.[1] Studies with the MAS, a six-point ordinal scale (0, 1, 1+, 2, 3, 4), have demonstrated it to correspond best for the elbow joint.[11,29] Healthy controls were grouped together and assigned their own categorical label different from all clinical data. This concept builds from previously published works that demonstrated that a difference between the two groups can be detected.[27] All clinical assessments were performed moments prior to each robotic data

collection session to ensure that all experimental data was concomitant and reflective of their most current condition. For consistency, individuals remained seated in their specialized wheelchairs placed adjacent to the robot arm shown in Figure 1. Using a standard orthosis, the individual's limb was interfaced to a force sensor that was affixed to an adapted 6 Degree of Freedom (DOF) industrial robot.[28] Force data was collected at the same point of contact on the individual that healthcare professionals would use while performing an assessment.

Protocol

For both, patient and control groups, five passive flexion and extension motions were performed consistently in the individual's para-sagittal plane as summarized in previous works[27,28] to ensure consistent within-subject speed increments. Briefly, the robot flexed and extended the elbow joint at progressively increasing speeds with each pair of flexion/extension motions.

Continuous velocity data values were used to fit the linear approximation model for each individual as discussed in the following section. A physiotherapist was present with the individual to pre-teach robot the individual's range of motion and ensure comfort.[28] An elbow rest alleviated the bulk weight of the arm, while still allowing the sensor to detect similar resistance readings to that of a clinician performing an assessment.[27,28] The final flexion/extensions motions were performed at approximately 1.5 rad/s, a value consistent with literature.[30-32] The purpose of collecting resistance readings with both, continuous values and a set of different velocities, was to obtain a better depiction of VDR. To provide rest, 2 second pauses were taken between each motion.

Outcome measures

All participants had 3-dimensional resistive force (N), position readings (mm), and time of occurrence data collected at 60 Hz. Force data was collected from a 6 DOF ATI Force/Torque sensor, and was mounted onto a FANUC F5 6DOF robot arm. This paper presents a study that tests the relationship between VDR during passive stretch and MAS scores in a two-stage statistical analysis. First, the velocity dependent component of the resistive forces was approximated from the continuous values of position, time, and force readings. For investigative purposes, a relationship between force, position, and velocity is constructed in the equation below:

$$F = aX + b\dot{X}$$

Regressing force magnitude readings (F) onto position (X) and velocity ($\dot{X}$) allows parameters a and b to be used as outcome measures representing position dependent and velocity dependent force components respectively. Specifically, b represents the change in force per unit change in velocity, where F represents resistive force readings from the sensor, X represents position readings, $\dot{}$ represents the velocity, and a and b

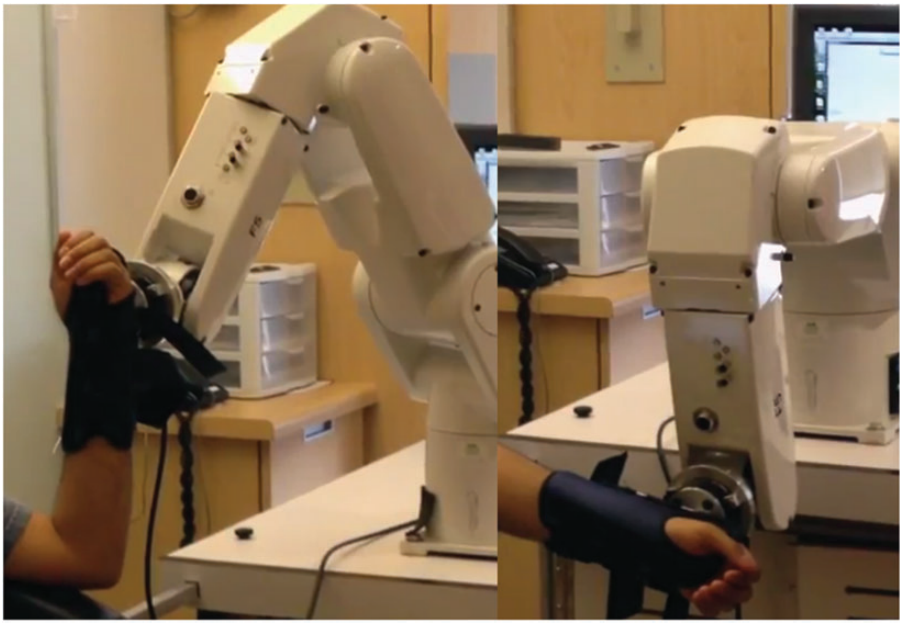

Figure 1. Robotic arm passively leads limb through elbow flexion/extension through the para- sagittal plane while attached at the wrist.

represent the position dependent and velocity dependent force components respectively.

For each affected limb of each subject, *b* was estimated using the method of least squares in MATLAB (Mathworks Inc.) where force magnitude readings were dependent on both, position readings in millimetres from initial starting position, and velocity data estimated from position and time readings. This approach also utilizes all the force and position data collected at various velocities for determining VDR.

As the sign (+\–) of b indicates direction of VDR forces, only the absolute value or magnitude of b is studied within the scope of this analysis and not the direction. In this study, a separate b-values are calculated from flexion motions, extension motions, and both flexion and extension motions together depending on the analysis presented in the following section.

Analysis

The second stage tests whether the |b|-values exhibit a relationship with the MAS scores assessed by the clinician, including a separate category representing a healthy control level of resistance. It is expected that decreases in |b|-values would correspond with lower VDR, and thus, lower clinical scores. Thus, it is expected that healthy individuals would possess the lowest |b|-values.

To test whether |b|-values were related to the MAS (Hypothesis i), three statistical analyses were performed treating the |b|-values of each subject as a response variable. A mixed linear model was fitted using the calculated |b|-value as the dependent variable and MAS score as an independent explanatory variable. First, MAS Bicep (MASB) and MAS Tricep (MAST) scores were tested separately in similar models with subject and limb nested within subjects included as random effects. Similar to the clinician assessments, |b|-values in the MASB model were calculated from extension data whereas |b|-values in the MAST model was calculated from flexion data. To test main effects of MASB and MAST scores (including the separate grouping of healthy controls), F-tests were performed for each model testing for unequal variance.

To test whether an MASB and MAST interaction was affecting VDR values (Hypothesis ii), an interaction model regressed |b|-values on all combinations of MASB and MAST scores as separate categories, referred to as MAS(B,T). For this model, new |b|-values were calculated from both flexion and extension motions as opposed to the single directions. These new |b|-values were then fit into the interaction model. This model helps determine whether differences in mean |b|-values exist between MAS(B,T) scores and healthy individuals (Hypothesis iii) as well as examine the agonist/antagonist relationship between the two assessments assumed independent of one another. Specifically, healthy individuals are contrasted to individuals with ABI. All models were analyzed in SAS (SAS Institute Inc.) using proc mixed, a specialized procedure that could adjust variances for t-tests and comparisons.

Results

Descriptive results

A total of 48 healthy individuals (18 male, 30 female) and 42 (25 male, 17 female) individuals with an ABI were recruited, confirmed eligible, and participated in the study. Characteristics of the sample of clinical participants are presented in Table 1. From this clinical group, 42 participants were able to have both their limbs tested. The data for these limbs were treated as separate data entries as they often possessed different MAS scores and were also treated in the statistical model as separate limbs grouped within a single participant.

Statistical results

The relationship between MAS scores and |b|-values (Hypothesis i), presented in Table 2, was tested using F-tests for the fixed effects were separate models fitted for MASB scores, MAST scores. Both MASB and MAST scores had an effect on mean |b|-values ($p < 0.0001$). The final row shows the F-test for the

Table 1. Demographics of clinical group with ABI.

Characteristic	Values
Age (y)	45.0 ∓ 13.9
Body mass (kg)(n = 15)	84.9 ∓ 16.9
Sex(M/F)	25/17
Unilateral/Bilateral	10/32
Etiology(T/H)	20/22

Note: Values are ∓ SD or n.
y: years; M: male; F: female; T: traumatic; H: hemorrhagic.

Table 2. Fixed effects of MAS assessment scores on |*b*|-values representing VDR.

Health-MAS(B/T)	Num DF	Estm. (Std Err.)	Stat.	P-Value
(0,0), (0,1), (1,0)	3		F = 3.01	.03
(0,0)	1	−0.81 (1.68)	t = −0.48	.634
(0,1)	1	−6.87 (3.55)	t = −0.48	.05
(1,0)	1	−5.12 (5.12)	t = −0.48	.08

interaction between MASB and MAST scores (Hypothesis ii), which is also found to be significant ($p = 0.0007$) when combined and reparametrized as an MAS(B,T) score.

The mean |b|-values for each MAS level, presented in Table 3, also demonstrates through t-tests that all levels of VDR were greater than zero; a finding consistent with literature.[14,32] This is significant as it demonstrates strong evidence of VDR being present across all categories of MAS scores including healthy subjects with notable differences in the range of each groups. The mean |b|-values for each level of MAS and their interactions were evaluated with boxplots, shown in Figures 2 and 3 respectively, as well as estimates in Table 4. By inspection, the boxplots provide evidence that a difference in VDR between the low end of the scale (H, 0, 1, 1+) and the higher scores of MAS 3 and 2. The low-end of the scale also suggests no differences between low scores. Considering data from both muscle groups together, however, the non-additive interaction effect becomes clear as an increase in MASB scores does not necessarily correspond to increases in |b|- values. With the exception of MAS (3,3), all bicep and tricep assessments that were equal (0,0), (1,1) etc. yielded relatively low |b|-values. The interaction is best typified comparing MAS(B,T) scores of (0,1) which is 16.69 ∓ 2.11, whereas (1, 0) is 5.11 ∓ 1.16

Table 3. Mean estimates of |*b*|-Values for healthy individuals and MAS scores separated by MAS Bicep and MAS Tricep. Results from t-tests confirm that each score demonstrates |*b*| greater than 0 ($p < 0.05$). Fundamentally, VDR values are generally observed to increase with the MAS scores. The trend is consistent when considering healthy limbs in addition to those from a clinical population.

Effect	F-Stat	P-value
MASB	16.25	<.001
MAST	11.55	<.001
MAS Comb.	2.82	<.001

The mean estimates suggest that two distinct groupings of VDR within the MAS, with the marked increase occurring between MAS 1+ and 2 separating low and high |b|-values. It is of

interest whether |b|-values can be used to differentiate between the different MAS groups. Thus,

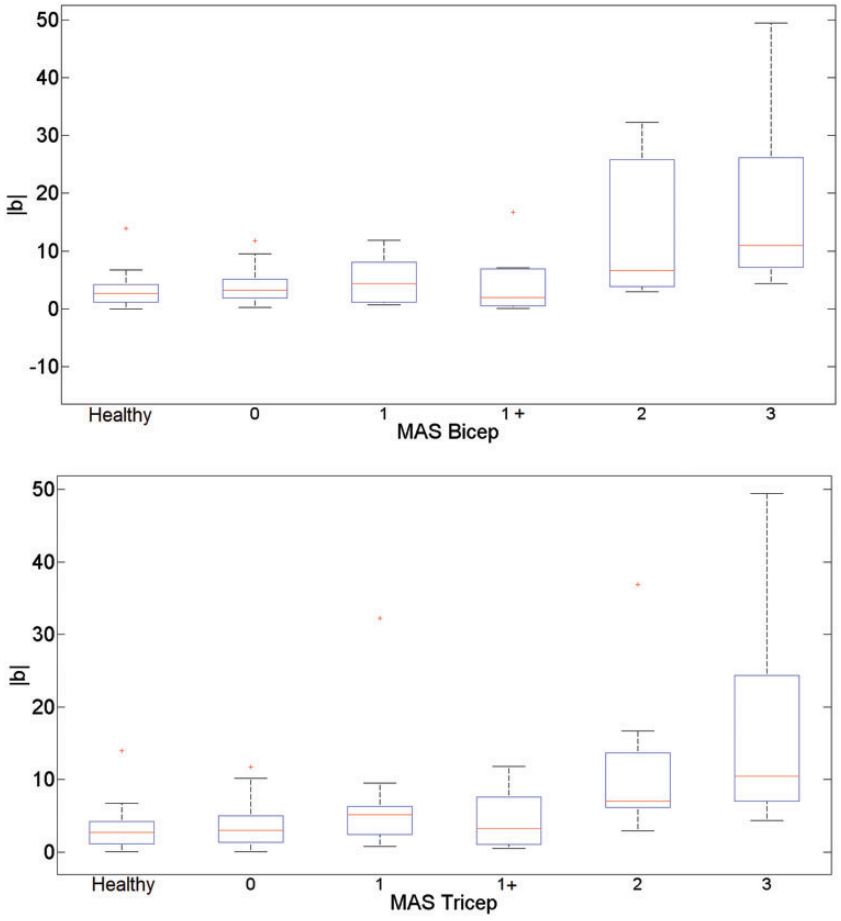

Figure 2. Boxplots of |b|-values for all MAS Bicep (above) and MAS Tricep (below).

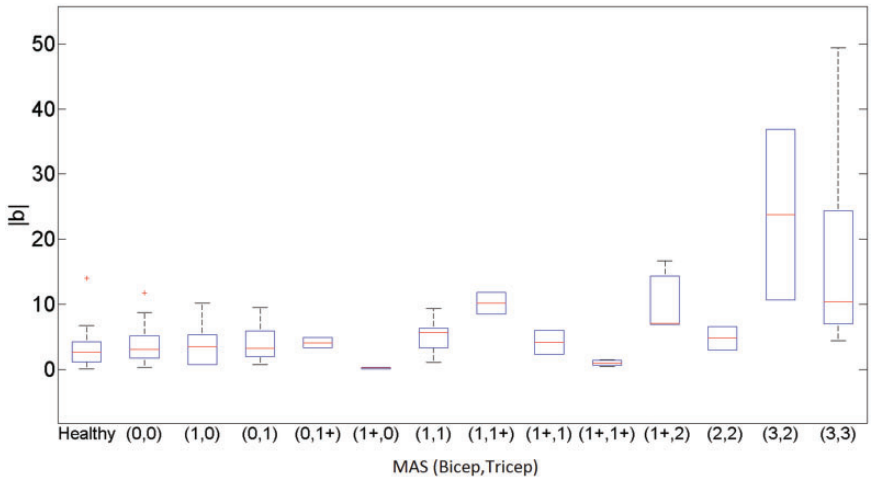

Figure 3. Boxplots of |b|-values are listed for select combinations of MAST and MASB from interaction model. Statistical tests performed help determine which groups are different from one another.

Table 4. Mean |*b*|-Value estimates for healthy controls and combinations MAS Bicep,Tricep (B,T) scores.

	MAS Bicep			MAS Tricep		
MAS	Estm.	Std. Err.	P-Value	Estm.	Std. Err.	P-Value
H	3.04	0.83	<.001	3.41	1.12	.004
0	3.77	0.82	<.001	3.55	1.01	.001
1	4.98	1.33	<.001	6.54	1.62	<.001
1+	4.24	1.73	.02	4.66	2.26	.05
2	13.39	3.16	<.001	11.69	2.12	<.001
3	17.80	1.58	<.001	16.58	1.19	<.001

differences of means and contrast comparisons, presented in Tables 5 and 6, were performed to determine whether differences exist across the neighboring assessment categories (Hypothesis iii). Several but not all MAS(B,T) groups were found to have statistically significant differences in their VDR values. Comparing means (Table 5), healthy individuals could be separated from several groups but not those theoretically closest to them on the MAS scale. Using the MAS(B/T) interaction model, F-tests compared healthy individuals to those with an MAS score of (0, 0), (0, 1), or (1, 0). Healthy individuals could be separated from those with low MAS scores grouped together, shown in Table 6, but not the individual groups themselves at the 0.05 level.

Table 5. F-test and t-tests comparing healthy individuals to individuals with an MAS 0. Denominator Degrees of Freedom = 145.

MAS (B,T)	N	Estm.	Std Err.	t-stat	P-Value
Healthy	88	9.83	1.11	8.85	<.001
0,0	74	10.63	1.26	8.42	<.001
0,1	10	16.69	3.38	4.94	<.001
0,1+	4	4.70	4.99	0.94	.35
0,2	2	26.90	7.08	3.80	<.001
1,0	18	5.11	2.49	2.06	.04
1+,0	4	4.13	5.17	0.80	.43
1,1	12	9.21	2.98	3.0	.002
1,1+	4	14.69	5.08	2.89	.004
1+,1	4	8.65	5.00	1.73	.09
1+,1+	6	25.08	4.50	5.57	<.001
1+,2	6	11.52	4.68	2.46	.02
2,1	2	11.83	7.37	1.61	.11
2,2	4	8.14	6.01	1.35	.18
3,2	4	21.34	5.42	3.94	.001
3,3	20	20.55	2.82	7.28	<.001

Discussion

Relationship between MAS and |b| (hypothesis i)

The most commonly used clinical scale for assessing spasticity,[1] the MAS, has sensitivity, objectivity, and

Table 6. Contrasts between Healthy individuals and select combinations of MAS Bicep, Tricep (B,T). Significant differences in |b|-values were often found between neighbouring MAS scores. This finding provides evidence that considering an interaction can help differentiate between Denominator Degrees of Freedom = 145.

MAS Differences (B,T) − (B,T)	Estimate	Std Err	t-stat	P-Value
Healthy–(0,1)	−6.87	3.55	−1.93	.05
Healthy–(0,2)	−17.07	7.17	−2.38	.02
Healthy–(1,0)	4.71	2.72	1.73	.09
Healthy–(1+,1,+)	−15.25	4.64	−3.29	.001
(0,0)–(1,0)	5.51	2.73	2.02	.05
(0,0)–(1+,1+)	−14.45	4.66	−3.1	.002
(0,1)–(0,1+)	11.99	6.01	2.0	.05
(0,1)–(1,0)	11.58	4.07	2.85	.005
(0,1)–(1+,0)	12.56	6.17	2.04	.04
(0,1+)–(0,2)	−22.20	8.67	−2.56	.01
(0,1+)–(1+,1+)	−20.38	6.55	−3.11	.002
(0,2)–(1+,1)	18.25	8.67	2.10	.04
(0,2)–(1+,2)	15.38	7.00	2.20	.03
(0,2)–(2,2)	18.76	9.29	2.02	.05
(1,0)–(1+,1+)	−19.97	5.10	−2.91	<.001
(1,1)–(1+,1+)	−15.87	5.33	−2.98	.003
(1+,0)–(1+,1+)	−20.95	6.85	−3.06	.003
(1+,1) − (1+,1+)	−16.43	6.70	−2.45	.02
(1+,1+) − (1+,2)	13.56	6.50	2.09	.04
(1+,1+) − (2,1)	13.25	8.63	1.54	.13
(1+,1+) − (2,2)	16.94	7.51	2.26	.03
(2,2) − (3,3)	−12.41	6.64	−1.87	.06

validity issues at measuring spasticity.[8,33–35] Small but important changes in spasticity are often not detected clinically, thus prolonging rehabilitation. Quantitative representations of spasticity and its related symptoms can alleviate this problem. Using sensors to provide quantitative values provides another level of reliability to assist clinicians.

In this study, the continuous VDR metric, "|b|", is proposed based on velocity dependent biomechanical time-series resistive force data collected from a rehabilitation robot for UL motions in the para-sagittal plane. The VDR metric was compared to clinical scores of both major UL muscle groups. MASB and MAST scores, while including healthy controls, were both found to demonstrate a relationship with the |b|-values in their respective models ($p < 0.001$). The presence of measured VDR and its relationship with the clinical scale suggests that the effects detected by the scale are also being detected by the system. These findings provide further evidence for the utility of robotics to help assist and fine-tune UL spasticity assessment and stiffness in the para-sagittal plane, in addition to other motions such as ankle plantar flexion[36] or elbow flexion/extension in the transverse plane.[37] A strength of our study is that the robot was able to obtain detailed temporal information without the need of additional invasive or time- consuming procedures such as needle EMG.

Furthermore, robotic approaches provide controlled motions and a level of repeatability to reduce subjectivity issues[32] introduced from manual based methods.[9] While not exclusively suggesting a method of evaluation, the data collected provides clinicians with preliminary estimates and ranges that form parts of new methods to help assess and monitor changes in an individual's spasticity for a functional motion pertinent to ADL.

Interaction and differences (hypothesis ii)

An interaction between the MASB and MAST scores was also found to be present in the relationship with |b|, a relationship otherwise difficult to detect by hand. This suggests the VDR experienced by the limb is influenced by the condition of both major muscle groups considered together as opposed to individually which is how they are currently performed. Fitting the new combined interaction model can produce noticeably more variability in the data, as non-additive

results can be observed by comparing estimates from Table 3 to Table 4. The non-additive effect may also explain why MAS 1+ scores went against the general trends presented in Table 4. The findings of the re-fit data, however, suggests that an interactive model can still describe this greater variability. A physiological reason for this finding could be the fact that the biceps and tricep muscle groups are already an agonist/antagonist muscle pairing. This finding may account for why previously described issues exist with manual MAS evaluations that only consider 1 muscle group.[10] The contrasts between the different MAS(B,T) interaction levels suggest a counter-intuitive non-additive structure for the scale when both scores are considered.

Discussions with physiotherapists who performed the MAS assessments yielded that a potential reason for this effect is that individuals with moderate to high MAS scores will possess a marked increase in resistance, often termed as "the catch",[38] at a specific point in their range of motion. After moving their limb past the catch, the clinicians noted these individuals will often exhibit lower resistance in their range of motion. Previous studies from this population confirmed that a catch followed by decreased resistance was observed in certain individuals.[28] While not applicable to every individual, this general observation from the study population could help explain this observation.

Evidence of muscle group interaction having an effect on VDR is significant as it suggests that VDR by a single flexion or extension motion is influenced by both muscle groups and should be considered during traditional assessment. This application of these findings could further augment and improve the sensitivity of VDR assessment.

Contrasts to healthy controls (hypothesis iii)

Individuals without spasticity, including healthy controls and MAS 0's, are anticipated to still demonstrate a natural amount of VDR behavior. Considering this, values from healthy individuals were shown to be different from combined low end of the scale where MAS (B/T) of (0, 0), (0, 1) and (1, 0) were grouped together. This add further evidence to previous studies suggesting differences from healthy participants.[27,37] Furthermore, |b|-values calculated for each limb could distinguish the VDR between the high (>1+) and low ends of the MAS (<1+). In light of the interaction between MAS Bicep and MAS Tricep score, these findings may imply that some of the ambiguity and sensitivity issues of the MAS may stem from the scale attempting to communicate multiple factors related to VDR, tone, stiffness, and spasticity. Instead, multiple dimensions or values, such as MASB and MAST scores presented together, may be required to communicate this complexity. Using robot controlled metrics, clinicians can now observe how these quantifiable VDR data change over time.

Clinical impact

This robot-collected metric provides clinicians the ability to better monitor and assess changes in stiffness and VDR for ABI subjects. Assessment is important for treatment decisions and determining the effectiveness of interventions.[37,39] This study presents findings for isolated and quantified UL VDR from individuals with ABI. The quantitative metric collected from the robot presents continuous values which can be monitored for changes over time. Demonstrating that the biomechanical VDR |b| metric has a relationship with the MAS in this plane confirms that any information captured by the MAS for this motion is also being captured by the system. The |b|-value findings provides evidence that the MAS does provides relevant information for the elbow joint in the plane of motion, while also providing a more informative representation compared to using just the MAS itself. This is significant for the rehabilitation process as limb assessments influence treatment decisions regarding UMN syndrome and contracture. This is especially important for the severe ABI population, for whom UMN syndrome can be a major barrier to recovery while possessing relatively stationary clinical scores.

The continuous valued quantities provided by an individual's |b|-value allows for a less restrictive categorical form of assessment and would reflect smaller increments of change needed to address sensitivity issues that result in delays. Finding a separation between healthy individuals |b|-values and clinical |b|-values helps healthcare professionals by providing the ability to track changes in resistance with respect to a healthy baseline. Findings of a jump in the scale could suggest that analysis techniques within both subgroups of the jumps could be treated or analyzed differently. As well, observing greater resistance in the high end of the scale compared to the low end suggests the system can assist in the study of contractures.

The interaction between the bicep and tricep muscle groups supports a case for new ways of constructing clinical scales as current gold standards consider the muscle groups independently.

The assessment techniques and data collected for healthy individuals and those with ABI, demonstrate that the system can assist elbow flexion/extension evaluation using biomechanical data. This aspect reduces complexity, costs, and set-up times of exoskeleton type systems[36,37] while making the system more favorable for front-line use in clinical settings. Quantifying VDR and directly relating it to the MAS provides clinicians with a clearer picture of changes in tone and thus treatment efficacy. Furthermore, a robotic system potentially allows for non-clinical staff to administer assessments providing further cost benefits over time whereas the MAS assessment requires clinician expertise. The increased objectivity of |b| as compared to MAS may be favourable in clinical settings where timely decisions regarding VDR are required. The controlled and repeatable nature of the robotic system's motions also helps move towards a standardized assessment process and away from issues well documented with the MAS.

Limitations

Although clinical participants remained seated in customized wheelchairs, minor measurement differences may have occurred across participants depending on wheelchair size. The participants' ability to remain relaxed during testing may also have impacted measurement quality. Efforts to reduce these influences were made by verbal encouragement. Due to the small number of participants, several of whom are chronic patients, as well as several multiple comparisons within the population, study results should be considered of a pilot nature and interpreted with care.

Prospective multi-centered studies with larger samples may include additional populations such as multiple-sclerosis and spinal cord injury to provide more insight. In this study, only the relationship between the MAS and the robot data was investigated, but not with respect to change over time both within sessions and across treatment programs. Inter and Intra-subject speed variances and differences in ROM also presents a limitation in interpretation of comparing stiffness. Future works can address this by employing velocity and position controls. In addition, while the current presentation of a MAS(B,T) as a combination of scores presents challenges, it can be considered a step towards future work in improving communication of an individual's condition and improvements in a way that clinically considers multiple muscle groups.

Prospective studies may look into all the above-mentioned aspects, normalization of arm mass, improved sampling rates, and other outcome measures.

Conclusions

This study establishes that VDR pertaining to neurological injury is detectable by a rehabilitation robotic system leading the UL through motions in the parasagittal plane. The robot-controlled biomechanical data alone can be quantified in a way that reflects VDR information also captured by the MAS scale. Findings obtained suggest natural VDR values from healthy controls can be separated from individuals with low MAS scores. This is significant as it suggests that the low end of the MAS scale does not necessarily reflect a return to full ability in terms of VDR. Evidence of patients with higher MAS (2 and 3) had increased stiffness whereas stiffness at the low end was not consistent, suggesting the ability to assist in studying contractures. Furthermore, an interaction between MAS Bicep and MAS Tricep scores was found to have a significant effect on quantified VDR values. Using a robotic system focused on VDR assessment can reduce variability introduced via clinician subjectivity or other manual assessment based techniques.

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HAA

Contributorship

NS, DJ, and HAA researched literature, conceived the study, were involved in protocol development, and gaining ethical approval. NS and OBA analyzed the data. NS and DJ wrote the first draft of the manuscript. All authors interpreted the results, contributed to and approved the final manuscript.

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References

1. Kaplan M. Upper motor neural syndrome and spasticity. In: Nesathurai S (ed.) *The rehabilitation of people with spinal cord injury*. AAP Publishing, 2001, pp. 81–86.
2. Thompson FJ, Parmer R, Reier PJ, et al. Scientific basis of spasticity: Insights from a laboratory model. *J Child Neurol* 2001; 16: 2–9.
3. Lance J. *Spasticity: disordered motor control*. Yearbook Medical Publishers: Chicago, 1980.
4. Malhotra S, Cousins E, Ward A, et al. An investigation into the agreement between clinical, biomechanical and neurophysiological measures of spasticity. *Clin Rehabil* 2008; 22: 1105–1115.
5. Malhotra S, Pandyan AD, Day CR, et al. Spasticity, an impairment that is poorly defined and poorly measured. *Clin Rehabil* 2009; 23: 651–658.
6. Biering-Sorensen F, Nielsen JB and Klinge K. Spasticity assessment: a review. *Spinal Cord* 2006; 44: 708–722.
7. Wissel J, Verrier M, Simpson DM, et al. Post-stroke spasticity: predictors of early development and considerations for therapeutic intervention. *Pm R* 2015; 7: 60–67.
8. Ward A. Spasticity treatment with botulinum toxins. *J Neural Transm (Vienna)* 2008; 115: 607–616.
9. Zorowitz R, Gillard P and Brainin M. Sequelae and burden on stroke survivors and caregivers. *Neurology* 2013; 80: S45–S52.
10. Gowland C, Stratford P, Ward M, et al. Measuring physical impairment and disability with the Chedoke-McMaster stroke assessment. *Stroke: J Am Heart Assoc* 1993; 24: 58–63.
11. Biering-Sorensen B, Iversen HK, Frederiksen IM, et al. Treatment diary for botulinum toxin spasticity treatment: a pilot study. *Int J Rehabil Res* 2017; 40: 175–184.
12. Boyd AS, Benjamin HJ and Asplund C. Principles of casting and splinting. *Am Fam Physician* 2009; 79: 16–22.
13. Pandyan AD, Price CIM, Rodgers H, et al. Biomechanical examination of a commonly used measure of spasticity. *Clin Biomech* 2001; 16: 859–865.
14. Pandyan AD, Price CIM, Barnes MP, et al. A biomechanical investigation into the validity of the modified

Ashworth scale as a measure of elbow spasticity. *Clin Rehabil* 2003; 17: 290–294.
15. Onushko T, Hyngstrom A and Schmit BD. Effects of multi-joint spastic reflexes of the legs during assisted bilateral hip oscillations in human spinal cord injury. *Arch Phys Med Rehab* 2010; 91: 1225–1235.
16. Malhotra S, Pandyan AD, Rosewilliam S, et al. Spasticity and contractures at the wrist after stroke: time course of development and their association with functional recovery of the upper limb. *Clin Rehabil* 2011; 25: 184–191.
17. Ludvig D, Visser TS, Giesbrecht H, et al. Identification of time-varying intrinsic and reflex joint stiffness. *IEEE Trans Biomed Eng* 2011; 58: 1715–1723.
18. Powers R, Marder-Meyer J and Rymer W. Quantitative relations between hypertonia and stretch reflex threshold in spastic hemiparesis. *Ann Neurol* 1988; 23: 115–124.
19. Parker P, Englehart K and Hudgins B. Myoelectric signal processing for control of powered limb prostheses. *J Electromyogr Kinesiol* 2006; 16: 541–548.
20. Hargrove L, Englehart K and Hudgins B. A comparison of surface and intramuscular myoelectric signal classification. *IEEE Trans Biomed Eng* 2007; 54: 847–853.
21. Seth N. *Robotic assessment system for spasticity in patients with acquired brain injury*, PhD Thesis, University of Guelph, CA, 2015.
22. Seth N, Johnson D and Abdullah HA. Spasticity assessment system for elbow flexors/extensors: Healthy pilot study. In: *IEEE Symposium on computational intelligence in robotic rehabilitation and assistive technologies (CIR2AT)*, . Orlando, 2014, pp. 36–41.
23. Colombo R, Pisano F, Micera S, et al. Robotic techniques for upper limb evaluation and rehabilitation of stroke patients. *IEEE Trans Neural Syst Rehabil Eng* 2005; 13: 311–324.
24. Dukelow SP, Herter TM, Bagg SD, et al. The independence of deficits in position sense and visually guided reaching following stroke. *J Neuroengineering Rehabil* 2012; 9: 72.
25. Hussain A, Balasubramanian S, Lamers I, et al. Investigation of isometric strength and control f the upper extremities in multiple sclerosis. *J Rehabil Assistive Technol Eng* 2016; 3: 2055668316663977.
26. Seth N, Johnson D and Abdullah HA. Transverse forces versus modified Ashworth scale for upper limb flexion/extension in para-sagittal plane. In: *Proceedings of IEEE international conference on rehabilitation robotics (ICORR),* London, 2017. pp. 765– 770.
27. Zhang LQ, Chung SG, Yupeng R, et al. Simultaneous characterizations of reflex and nonreflex dynamic and static changes in spastic hemiparesis. *Journal of Neurophysiology* 2013; 110: 418–430.
28. Seth N, Johnson D, Taylor GW, et al. Robotic pilot study for analysing spasticity: clinical data versus healthy controls. *J Neuroengineering Rehabil* 2015; 12: 1–13.
29. Bosecker C, Dipietro L, Volpe B, et al. Kinematic robot based evaluation scales and clinical counterparts to measure upper limb motor performance in patients with chronic stroke. *Neurorehabil Neural Repair* 2010; 24: 62–39.
30. Lebiedowska M and Fisk J. Knee resistance during passive stretch in patients with hypertonia. *Journal of Neuroscience Methods* 2009; 179: 323–330.
31. Rabita G, Dupont L, Thevenon A, et al. Quantitative assessment of the velocity-dependent increase in resistance to passive stretch in spastic plantar flexors. *Clin Biomech (Bristol, Avon)* 2005; 20: 745–753.
32. Mirbagheri M, Ness L, Patel C, et al. The effects of robot-assisted locomotor training on spasticity and volitional control. In: *Proceedings of IEEE International Conference on Rehabilitation Robotics (ICORR)*. Zurich, 2011. pp. 5975443.
33. Fleuren JF, Voerman GE, Erren-Wolters CV, et al. Stop using the Ashworth scale for the assessment of spasticity. *J Neurol Neurosurg Psychiatry* 2010 ; 81: 46–52. Jan
34. Pandyan AD, Johnson GR, Price CI, et al. A review of the properties and limitations of the Ashworth and modified Ashworth scales as measures of spasticity. *Clin Rehabil* 1999 ; 13: 373–383. Oct
35. Kumar RT, Pandyan AD and Sharma AK. Biomechanical measurement of post-stroke spasticity. *Age Ageing* 2006 ; 35: 371–375.
36. Semrau JA, Herter TM, Scott SH, et al. Inter-rater reliability of kinesthetic measurements with the kinarm robotic exoskeleton. *J Neuroeng Rehabil* 2017; 14: 42.
37. Centen A, Lowrey CR, Scott SH, et al. KAPS (kinematic assessment of passive stretch): a tool to asses elbow flexor and extensor spasticity after stroke using a robotic exoskeleton. *J Neuroengineering Rehabil* 2017; 14: 59–13. 1-
38. Bohannon RW and Smith MB. Interrater reliability of a modified ashworth scale of muscle spasticity. *Phys Ther* 1987; 67: 206–207.
39. Hosseini S and Kaplan M. Spasticity. In: Nesathurai S (ed.) *Essentials of in-patient rehabilitation*. 2nd ed. AAP Publishing, 2001, pp. 129–136. Whitinsville, MA.