Evaluation of a toolkit for standardizing clinical measures of muscle tone

Chris A McGibbon1,2, Andrew Sexton1, Glen Hughes1, Adam Wilson1, Melony Jones1, Colleen O’Connel1,2, Kim Parker1, Catherine Adans-Dester4, Anne O’Brien3 and Paolo Bonato5

1 Institute of Biomedical Engineering, University of New Brunswick, Fredericton, New Brunswick, E3B 5A3, Canada
2 Faculty of Kinesiology, University of New Brunswick, Fredericton, New Brunswick, Canada
3 Stan Cassidy Centre for Rehabilitation, Fredericton, New Brunswick, Canada
4 Nova Scotia Health Authority, Halifax, Nova Scotia, Canada
5 Spaulding Rehabilitation Hospital, Boston, MA, United States of America

Abstract

Objective: To evaluate a new portable toolkit for quantifying upper and lower extremity muscle tone in patients with upper motor neuron syndrome (UMNS). Approach: Cross-sectional, multi-site, observational trial to test and validate a new technology. Setting: Neurorehabilitation clinics at tertiary care hospitals. Participants: Four cohorts UMNS patient, >6 mo post acquired brain injury, spinal cord injury, multiple sclerosis and cerebral palsy, and a sample of healthy age-matched adult controls. Measures: Strength: grip, elbow flexor and extensor, and knee extensor; range of motion (ROM): passive ROM (contracture) and passive-active ROM (paresis); objective spasticity: stretch-reflex test for elbow, and pendulum test for knee; subjective spasticity: modified Ashworth scale scores for elbow and knee flexors and extensors. Results: Measures were acquired for 103 patients from three rehabilitation clinics. Results for patient cohorts were consistent with the literature. Grip strength correlated significantly with elbow muscle strength and all patient populations were significantly weaker in upper- and lower-extremity compared to controls. Strength and paresis were correlated for elbow and knee but neither correlated with contracture. Elbow spasticity correlated with strength and paresis but not contracture. Knee spasticity correlated with strength, and subjective spasticity correlated with contracture. Significance: The BioTone™ toolkit provided comprehensive objective measures for assessing muscle tone in patients with UMNS. The toolkit could be useful for standardizing outcomes measures in clinical trials and for routine practice.

Introduction

Effective management of motor impairment in persons with upper motor neuron syndrome (UMNS) is critical for maximizing independent function and minimizing long-term disability (Bhimani and Anderson 2014). UMNS is a complicated disorder to treat, owing to the variable time course of, and interaction between, negative signs such as weakness and loss of dexterity and control, and positive signs such as hyper-excitability of the stretch-reflex (spasticity) and increased resistance to passive motion (Yelnik et al 2010, Logan 2011). Assessment of post-injury onset of paresis, spasticity and contracture is important for directing treatment (Wissel et al 2013), and for addressing the syndrome as a whole (Ivanhoe and Reistetter 2004). However, the relationships between these factors are complex and not well understood (Gracies 2005a, 2005b, Ada et al 2013), of post-injury onset of paresis, spasticity and contracture is important for directing treatment (Wissel et al 2010, Logan 2011). Assessment of post-injury onset of paresis, spasticity and contracture is important for directing treatment (Wissel et al 2013), and for addressing the syndrome as a whole (Ivanhoe and Reistetter 2004). However, the relationships between these factors are complex and not well understood (Gracies 2005a, 2005b, Ada et al 2013).

The inability to quantify important clinical indicators of abnormal tone in a consistent and repeatable manner remains as a significant and persistent gap in neuro-rehabilitation practice and research (Pandyan et al 2006, Ansari et al 2006, Fleuren et al 2009) but until recently few alternatives have existed that can be easily integrated into a clinical workflow. Unobtrusive sensors for measuring characteristics of muscle tone
in a clinical setting using kinematic (Chen et al 2005, Calota et al 2008, Bohannon et al 2009, Paulis et al 2010, McGibbon et al 2013), force/torque (Chen et al 2005, Pandyan et al 2006, Kumar et al 2006, Malhotra et al 2008), and electromyographic (Pisano et al 2000, Chen et al 2005, Pandyan et al 2006, Calota et al 2008, Naghdi et al 2008, McGibbon et al 2013) sensors represent a possible solution.

A drawback to all these studies is the singular focus on muscle spasticity. Muscle tone assessment requires more comprehensive measures than just evaluating spasticity (Gracies 2005a). Although technologies exist for measuring strength/force development and active and passive range of movement, the likelihood of achieving standardized clinical adoption of multiple separate tools is low. To our knowledge there is no fully-integrated commercial system for standardized, objective clinical assessment of muscle tone. The objective of the present study was to validate the BioTone™ system for acquiring objective clinical measures of muscle tone using low-cost wearable sensors during routine examination at multiple clinical sites and across different patient populations with UMNS.

This paper presents the outcomes measures (elbow and knee strength, active and passive ROM and spasticity) from four patient populations across three clinical sites, and uses a mixture of descriptive and inferential statistics for (1) testing these outcomes as valid measures of muscle tone, and (2) exploring the relationships between strength, range of motion (ROM) measures (paresis and contracture) and clinical and objective measures of spasticity for the elbow and knee.

**Methods**

**Study design**

This was a cross-sectional, multi-centre, observational trial to evaluate the validity of the BioTone™ system, an integrated set of measurement tools, testing protocols, and analytics for comprehensive clinical assessment of elbow and knee muscle tone.

**Study sites**

Three sites were included in the study: the Stan Cassidy Centre for Rehabilitation (SCCR, Fredericton NB), the Nova Scotia Rehabilitation Hospital (NSRH, Halifax NS), and the Spaulding Rehabilitation Hospital (SRHB, Boston MA). The study was approved by Research Ethics Board (REB)/Institutional Review Board (IRB) for each site, and the University of New Brunswick (UNB, Fredericton NB) REB.

**Research participants**

**Recruitment**

Patients meeting the enrollment inclusion/exclusion criteria below were referred to or approached by the site coordinator. Upon providing written consent, the participant was enrolled in the BioTone™ system software by creating a new patient record with a unique identification code. Healthy control subjects were recruited locally (UNB and local community) by advertisement and word-of-mouth. All participants in the study provided informed, signed consent prior to data collection.

**Patients**

In order to establish generalizability of BioTone™ assessment, four of the most common patient populations affected by UMNS were selected.

**Inclusion criteria**: Male or female active inpatient or outpatient currently receiving services at study site for one or more of the following diagnoses: acquired brain injury (ABI, including strokes, trauma, etc), spinal cord injury (SCI, incomplete any level or complete C7 and below), multiple sclerosis (MS, meeting MacDonald criteria), and cerebral palsy (CP, hemiplegic or diplegic); current diagnosis has been assessed as medically stable by their physiatrist/physician; exhibits some degree of abnormal tone in either upper or lower limbs, specifically at the elbow and/or knee joint(s); and capable of providing informed consent. Adults 18 or older were included at all three sites, and 16 or older at the SCCR site.

**Exclusion criteria**: Bariatric; joint arthopathy (osteoarthritis, rheumatoid arthritis, etc); positive for methicillin-resistant *Staphylococcus aureus* or vancomycin-resistant *Enterococcus* bacteria; open skin lesions, and; not capable of comprehending instructions and consenting in English.

**Healthy control subjects**

Thirty healthy adult participants (15 female) were also recruited to acquire age-matched normative values for elbow and knee strength measurements. Included were adults 21 years old or older. Excluded were those with any known musculoskeletal or neurological injury, disease or condition, or any health condition that would contraindicate moderate physical activity. For the knee, 10 subjects (five female) between 21 and 67 years old were recruited for knee extensor strength measurement bilaterally. For the arm, twenty participants (10 female)
between 21 and 53 years old were recruited for elbow flexor and extensor strength measurement unilaterally (dominant side).

**Procedures**

**Clinical examination**

After enrollment, the patient underwent clinical assessment as regularly scheduled, which included the modified Ashworth scale (MAS) for spasticity assessment of elbow and/or knee flexors and extensors (Bohannon and Smith 1987). Although several authors have questioned the continued use of the MAS (Pandyan et al 2006, Ansari et al 2006, Fleuren et al 2009), there are currently few ‘low-tech’ alternatives that are scalable and easily integrated into a clinical workflow. Using a rapid stretch-reflex test the therapist rates spasticity as follows (Bohannon and Smith 1987):

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

Other information collected included age, height, weight, diagnosis group (ABI, MS, CP or SCI), affected extremities (left/right arm and/or leg), and years since injury or onset.

**BioTone™ assessment**

The BioTone™ Clinic toolkit (figure 1) consisted of a collection of integrated tools for acquiring measurements of voluntary muscle strength and joint ROM, kinematics and muscle activity during clinical examination. The BioTone™ software was programmed with clinical protocols that guide the user through the examination. Sensor measurements were displayed in real-time, and all data were stored in a database on a secure laptop computer for reviewing and analyzing off-line.

**Active and passive range of motion**

For elbow ROM tests, the participant was seated in a comfortable chair and positioned so the elbow had free . A fibre-optic goniometer (FOG) was positioned on the limb using the Neoprene/Velcro cuffs and secured in place such, as seen in figure 1(A). The FOG was then zeroed and the test began. The same process was used for the knee, but due to the seated positioning, a full flexion measurement was not possible so only the extension end of the active and passive range was used in the data analysis.

For passive range of motion (PROM), the therapist moved the participant’s limb slowly through its full range of unrestricted movement in the sagittal plane. For elbow active range of motion (AROM), the subject was instructed to move their forearm from their full flexion angle to full extension angle in the sagittal plane. For both active and passive tests, the maximum angle achieved (peak flexion angle) and the minimum angle achieved (peak extension angle) were recorded and used as described below to estimate the degree of contracture and paresis.

**Isometric muscle strength**

Elbow flexor and extensor strength were measured with a limb strength measurement device (LSMD-arm) as shown in figure 1(B). The LSMD was adjusted to ‘extensor’ or ‘flexor’ orientation and positioned on the extremity such that the subject’s joint formed an angle of approximately 90°. The subject then sat with elbow supported (by therapist or side-table) and extended or flexed with maximal effort. This was repeated three times for extension and flexion with 15 sec between trials, and approximately 1–2 min between extension and flexion trials (the time required to doff the device, re-configure for direction, and re-don the device). The same procedure was used for the knee except only extensor force was measured. Details of the system can be found elsewhere (Landry et al 2015).

Grip strength was measured with the grip strength measurement device (GSMD, figure 1(B)). This was a custom device designed to accommodate participants with limited hand function. The rigid sensor provided no force feedback to the participant. The subject first held the grip tester and the hand grip size was adjusted to the subject’s hand size such that a full grip could be achieved. To measure grip strength the subject sat with elbow resting on table top and flexed at approximately 90° with forearm in neutral position. The subject then squeezed the GSMD with maximal effort. This was repeated three times each for right and left sides with 5 s between trials.
Muscle spasticity
Spasticity data for this study and associated testing protocols have been published elsewhere for the elbow (McGibbon et al 2013, McGibbon et al 2016) and for the knee (Whelan et al 2018). For the elbow, an instrumented stretch-reflex test was conducted where the therapist moved the forearm slowly through elbow flexion, and then rapidly through extension to stretch the elbow flexors. This was repeated for elbow extensors. Stretch-reflex tests were repeated at least three times for each muscle. Only the most involved arm was tested.

For the knee, the Wartenberg pendulum test was used. The participant was positioned in a seated posture with legs hanging freely over the edge of a plinth and torso reclined to approximately 30°. The therapist then slowly raised the lower leg to full extension (or passive extension limit) and held the leg horizontal until the participant was completely relaxed, as indicated by real-time EMG display. The participant’s lower-leg was then released and allowed to oscillate until coming to rest. Pendulum tests were repeated at least three times for the right and/or left legs.

Full time records of FOG and EMG sensors were stored for off-line analysis, as described elsewhere (McGibbon et al 2016, Whelan et al 2018).

Data analysis
All statistical analyses were carried out with SPSS (v21 IBM Inc.). These consisted of ANOVA tests for comparing groups (healthy controls to patients) and Pearson correlation analysis for examining relationships between variables. Significance was set at \( \alpha = .05 \).

BioTone™ measurements
Contracture and paresis
PROM is a typical measure of contracture (Ada et al 2006) and is normally quantified as the difference in PROM between a patient’s impaired and non-impaired side (Bohannon 1987). We did not measure PROM bilaterally for upper extremities, therefore we used anatomical norms for PROM of the elbow (0 to 150°) to estimate contracture, as illustrated in figure 1(A). Although PROM as a single measure is useful, we chose to analyze it by end-range, which is therefore specific to extensor contracture (inability to passively flex the elbow, \( C_{ext} = 150 - \max \text{(PROM)} \)) or flexor contracture (inability to passively extend the elbow, \( C_{flx} = \min \text{(PROM)} \)). Values of \( C \) near zero represent no contracture while increasing positive values (in degrees) indicate increasing contracture.
AROM reflects both the passive limits of movement and the deficiency in voluntary muscle force to achieve end-limits of motion (i.e. at its shortest length). Therefore, the difference between passive and active ROM should be a measure of paresis that is independent of contracture and dependent on voluntary strength. The passive-active limit angles were computed for extensors \( (P_{\text{ext}} = \min(\text{AROM}) - \min(\text{PROM})) \) and flexors \( (P_{\text{flx}} = \max(\text{PROM}) - \max(\text{AROM})) \). Values of \( P \) near zero therefore represent low relative paresis (can make use of their full passive range) while increasing positive values (again in degrees) indicate inability to reach available end-points.

The same analysis was applied to the knee except only \( P_{\text{ext}} \) and \( P_{\text{flx}} \) were computed since the knee passive and active ranges were only tested in extension.

**Voluntary muscle strength**

Muscle strength for extensors \( (S_{\text{ext}}) \) and flexors \( (S_{\text{flx}}) \), and grip strength \( (S_{\text{grip}}) \) were assessed using the instruments in figure 1(B) and normalized to body weight in Newtons. For convergent validity we evaluated the correlation between grip strength and elbow flexor and extensor strength for the pooled sample. Furthermore we expect that muscle paresis as measured by passive–active range limit angles \( (P) \) will correlate inversely with muscle strength \( (S) \); in other words, patients with higher degree of muscle paresis (large passive-active limit angle) will be weaker than those with less paresis (small passive-active limit angle).

For concurrent validity we compared elbow and knee strength from the healthy control subjects measured with the LSMD-arm and LSMD-leg to the patient population studied here, with the expectation that the patient population would have significantly lower strength than an adult sample without neurological impairments. We also compared strength of elbow flexors and extensors and knee extensors between patient groups.

**Inter-relationships between contracture, paresis and spasticity**

Detailed analyses of objective spasticity metrics from manual stretch-reflex data for elbows and pendulum test data for knees can be found elsewhere (McGibbon et al 2016, Whelan et al 2018). Correlation analysis was used to examine the relationship between spasticity measures (clinical and objective) and voluntary muscle strength, passive-active limit angle (paresis), and passive limit angle (contracture), for the elbow and the knee.

**Results**

**Patient sample**

One hundred and three patients participated, consisting of 54 with ABI, 23 with SCI, 14 with MS and 12 adults with CP. The sample was mostly male (71 men versus 32 women). The age range of the sample spanned from as young as 16 to as old as 93. Patient characteristics are shown in table 1.

Of the 103 tested patients, 79 (77%) were evaluated for the involved upper extremity or the most involved side. Of these patients, most affected side was roughly equivalent for right and left extremities; 42 (53%) were right side affected and 37 (47%) were left side affected. For the lower-extremity, 98 (95%) were evaluated, with 57 (58%) affected most on right side and 41 (42%) affected most on left side, with 44 (45%) of these being bilaterally involved.

MAS scores for elbow and knee flexors and extensors are shown in figure 2. For the elbow ABI patients had higher elbow flexor spasticity than SCI patients \( (p = .008) \) and higher elbow extensor spasticity compared to MS patients \( (p = .005) \). For the knee, ABI patients had lower flexor spasticity compared to SCI patients \( (p = .003) \), and CP patients had higher extensor spasticity compared to SCI \( (p = .006) \) and ABI \( (p = .004) \).

**BioTone™ measurements**

BioTone™ measures of passive and active range of motion and grip, elbow and knee strength for the four patient cohorts are summarized in table 2.

**Contracture and paresis**

For the elbow, there was a significant difference between ABI and SCI groups in paresis angle \( (p = .034) \) but no other differences were detected between cohorts. Paresis angle was also significantly higher for MAS category 3 compared to MAS category 0 \( (p = .024) \) but no other significant differences were detected. Contracture angles were not different by cohort nor were they different by MAS category.

For the knee, there were no significant differences in paresis angles for MAS categories or for cohorts, although there was a trend toward a significant difference between the SCI and ABI group \( (p = .069) \). Contracture angle was significantly higher for the MAS = 3 category compared to the MAS = 0 category \( (p = .011) \) but no other differences were detected.
Voluntary muscle strength

Although there was a significant difference between the strength level of the patient population compared to healthy adults ($p < .001$), no significant differences were detected between the patient groups for elbow muscle strength. Patients in MAS category 0 were significantly stronger than those in MAS category ‘3’ ($p < .001$), ‘2’ ($p = .014$), and ‘1+’ ($p = .009$), but MAS category 1 was not different from any group. For the knee, the control

---

**Table 1.** Site recruitment and participant demographic data.

| Gender | Age, years | Years since Dx | Upper extremity | Lower extremity |
|--------|------------|----------------|-----------------|-----------------|
|        | Male       | Female         | Mean (SD)       | Mean (SD)       | n   | n   |
| SCCR   |             |                |                 |                 |     |     |
| ABI ($n = 10$) | 6 | 4 | 38 (15) | 4.8 (3.6) | 10 | 9 |
| MS ($n = 10$) | 6 | 4 | 55 (15) | 18.1 (8.3) | 3 | 10 |
| CP ($n = 10$) | 5 | 5 | 36 (12) | 36.0 (12.3) | 7 | 10 |
| SCI ($n = 10$) | 10 | 0 | 38 (9) | 16.3 (13.0) | 2 | 10 |
| Total ($n = 40$) | 27 | 13 | 42 (15) | 19.9 (15.0) | 22 | 39 |
| NSRH   |             |                |                 |                 |     |     |
| ABI ($n = 15$) | 11 | 4 | 65 (11) | 6.0 (6.9) | 15 | 14 |
| MS ($n = 3$) | 0 | 3 | 57 (4) | 15.3 (13.1) | 2 | 3 |
| CP ($n = 0$) | 0 | 0 | — (—) | — (—) | 0 | 0 |
| SCI ($n = 6$) | 5 | 1 | 48 (14) | 1.2 (.8) | 4 | 5 |
| Total ($n = 24$) | 16 | 8 | 60 (13) | 6.2 (8.0) | 21 | 22 |
| SRHB   |             |                |                 |                 |     |     |
| ABI ($n = 29$) | 23 | 6 | 54 (16) | 3.5 (3.2) | 28 | 27 |
| MS ($n = 1$) | 0 | 1 | 42 (—) | 13.5 (—) | 1 | 1 |
| CP ($n = 2$) | 0 | 2 | 26 (4) | 26.0 (4.2) | 1 | 2 |
| SCI ($n = 7$) | 5 | 2 | 52 (17) | .8 (4) | 6 | 7 |
| Total ($n = 39$) | 28 | 11 | 52 (16) | 4.4 (6.1) | 36 | 37 |
| Total   |             |                |                 |                 |     |     |
| ABI ($n = 54$) | 40 | 14 | 54 (17) | 4.5 (4.7) | 53 | 50 |
| MS ($n = 14$) | 6 | 8 | 55 (13) | 17.2 (8.7) | 6 | 14 |
| CP ($n = 12$) | 5 | 7 | 34 (12) | 34.3 (11.9) | 8 | 12 |
| SCI ($n = 23$) | 20 | 3 | 45 (14) | 16.1 (10.3) | 12 | 22 |
| Total ($n = 103$) | 71 | 32 | 50 (17) | 10.3 (12.6) | 79 | 98 |

ABI = acquired brain injury including stroke; MS = multiple sclerosis; CP = cerebral palsy; SCI = spinal cord injury. SCCR = Stan Cassidy Centre for Rehabilitation; NSRH = Nova Scotia Rehabilitation Hospital; SRHB = Spaulding Rehabilitation Hospital.

**Figure 2.** Mean clinical spasticity scores (modified Ashworth scale, MAS) assessed by therapists for flexors (dark bars) and extensors (light bars) of elbows (left plot) and knees (right plot) for the four patient cohorts. The * symbol and dashed lines indicate statistical differences between pairs of cohort groups. Error bars are 95% confidence intervals.

**Clinical measures of spasticity**

A. Elbow

B. Knee

Voluntary muscle strength

Although there was a significant difference between the strength level of the patient population compared to healthy adults ($p < .001$), no significant differences were detected between the patient groups for elbow muscle strength. Patients in MAS category 0 were significantly stronger than those in MAS category ‘3’ ($p < .001$), ‘2’ ($p = .014$), and ‘1+’ ($p = .009$), but MAS category 1 was not different from any group. For the knee, the control
A McGibbon et al

sample was significantly stronger than all patient groups individually \((p < .001)\), and among patient groups SCI patients were significantly weaker \((p = .034)\) compared to ABI patients, but no other significant differences were found. There was no clear relationship between knee extensor strength and clinical MAS score.

As expected, there was a significant correlation between grip strength and elbow muscle strength across the patient population, with grip strength explaining 63% of the variance in elbow flexor strength and 62% of the variance in elbow extensor strength (figure 5(A)). Finally, figure 5(B) shows normalized elbow strength versus paresis angles for all stretches (pooled flexors and extensors).

**Inter-relationships between contracture, paresis and spasticity**

Table 3 show results of pairwise correlations between strength, paresis and contracture. Although correlation coefficients were small, there were some significant relationships detected. For both the elbow and knee there was a significant inverse correlation between voluntary isometric strength (using the LSMD) and passive-active limit angle. Contracture was not significantly related to strength or paresis for the elbow and knee.

Spasticity of the elbow had a significant correlation with strength and paresis, for both the clinical (MAS) measure of spasticity and an objective measure of spasticity, and was not related to contracture. For the knee, however, the clinical MAS score was significantly related to contracture.

**Table 2. BioTone™ outcomes measures for range of movement variables and maximum voluntary strength.**

| Cohort        | ABI        | MS         | CP         | SCI         | Total       |
|---------------|------------|------------|------------|-------------|-------------|
| Mean (SD)     | Mean (SD)  | Mean (SD)  | Mean (SD)  | Mean (SD)   | Mean (SD)   |
| Active and passive ROM Most affected elbow (only) |
| Active flexion and extension Flexion (°) | 103.3 (29.7) | 124.6 (24.9) | 125.6 (11.5) | 126.0 (13.0) | 109.8 (28.1) |
| Extension (°) | 19.3 (23.2) | 23.2 (34.4) | 18.8 (13.8) | 4.5 (3.8) | 16.0 (19.2) |
| ROM (°)       | 86.1 (37.0) | 101.3 (51.6) | 106.7 (15.9) | 121.5 (13.0) | 93.8 (36.4) |
| Passive flexion and extension Flexion (°) | 125.5 (23.6) | 137.7 (21.3) | 141.4 (14.5) | 135.1 (11.99) | 129.0 (22.0) |
| Extension (°) | 4.0 (9.3) | 3.0 (8.2) | 10.0 (11.9) | 1.5 (2.7) | 4.1 (8.9) |
| ROM (°)       | 121.5 (26.2) | 134.8 (26.2) | 131.3 (15.7) | 133.5 (12.1) | 124.9 (24.2) |
| Most affected knee Active extension Extension (°) | 10.8 (16.0) | 16.6 (21.0) | 10.8 (9.9) | 23.8 (26.6) | 13.9 (18.7) |
| Passive extension Extension (°) | 1.4 (5.3) | 2.4 (5.7) | 3.1 (8.8) | 1.7 (6.1) | 1.8 (6.0) |
| Least affected knee Active extension Extension (°) | 2.7 (3.8) | 10.2 (7.7) | 10.2 (8.2) | 11.4 (11.6) | 7.7 (8.4) |
| Passive extension Extension (°) | −0.9 (4.3) | 3.2 (3.3) | 4.9 (1.8) | 2.0 (3.2) | 2.3 (4.0) |
| Muscle and grip strength Most affected arm Grip (N) | 86.4 (66.0) | 143.4 (111.3) | 124.7 (66.1) | 152.9 (192.1) | 101.2 (93.3) |
| Flexor (N)     | 63.8 (33.5) | 85.7 (58.7) | 78.3 (38.0) | 83.0 (67.9) | 69.1 (54.7) |
| Extensor (N)   | 102.1 (64.5) | 122.0 (71.9) | 81.9 (31.0) | 92.6 (97.7) | 100.5 (67.3) |
| Most affected knee Extensor (N) | 255 (144) | 199 (144) | 156 (49.7) | 156 (117) | 217 (137) |
| Least affected knee Extensor (N) | 238 (140) | 253 (112) | 170 (40.6) | 157 (68.0) | 207 (110) |

ABI = acquired brain injury including stroke; MS = multiple sclerosis; CP = cerebral palsy; SCI = spinal cord injury. Hyper-extension < 0, full extension = 0, flexion > 0.
Abnormal muscle tone of upper and lower extremities requires long term management for many people with UMNS following neurological injury or disease. Important clinical indicators that comprise muscle tone examination are difficult to measure reliably in the clinic, and metadata from clinical trials are often too inconsistent and/or unreliable to translate to practice. To address this, we developed and validated a portable, fully integrated, toolkit consisting of hardware (sensors) and software (protocols and analytics) for performing routine assessment of muscle tone in a clinical setting. Below, we discuss the main findings of the study.

**Voluntary isometric strength**

Recent studies suggest that muscle strength is a better predictor of post-stroke function than spasticity or contracture (Ada et al 2006, Harris and Eng 2007) but can also be highly variable depending on testing angle (Koo et al 2003). Standardized measurements of muscle strength are therefore important.

The present study established criterion validity of both the LSMD-arm and LSMD-leg by comparing UMNS cohorts to the healthy age matched controls from the above studies. Controls were significantly stronger than all the patient groups (figure 4(top)) for elbow flexion and extension ($p < .001$) and knee extension ($p < .001$). Although it is difficult to directly compare data from other studies due to testing method (most report isokinetic torque) and reporting method (many report only strength deficits relative to unaffected side rather than absolute strength), the above findings agree with other comparative studies (with healthy control subjects) in patients with UMNS (Bohannon et al 1986, Colebatch et al 1986, de Groot et al 2012, Guclu-Gunduz et al 2012, Citaker et al 2013, Moreau and Gannotti 2015).

For the elbow, data in table 2 show that flexor strength was more impaired than extensor muscle strength for all patient populations, which is supported (Colebatch et al 1986) but also contradicted (Pasternak-Mladzka et al 2007) in the hemiparetic stroke population. Unfortunately, most published studies report only elbow flexor strength (or knee extensor strength) in UMNS populations; there is surprisingly little data on the relative strength of flexor and extensor groups of the arm or leg across this population.

Knee extensor strength is one of the most commonly assessed muscles in the UMNS population due to its direct involvement with mobility (Pohl et al 2002, Kim et al 2004, Wren and Engsberg 2009, Citaker et al 2013, Stevens et al 2013, DiPiro et al 2015, Gunter et al 2015). Data in figure 4(B)(top) shows that healthy controls have knee strength values over $\frac{1}{2}$ body weight (~.6 BW), whereas in the UMNS cohorts knee strength was approximately .35 BW for ABI, .3 BW for MS and about .25 BW for CP and SCI. Knee extensor strength of .3 BW (2.8 N kg$^{-2}$) is the reported threshold needed for independent activity of daily living (ADL) function (Hasegawa et al 2008). Although we did not measure gait function or use of walking aids in this study, it can be seen that quadriceps strength for SCI and CP cohorts were both below the ADL threshold. This is consistent with the fact that walking aids are commonly used by CP adolescents and young adults (Yeung et al 2012) and the SCI population (Jaeger et al 1989).

Table 3. Spearman $\rho$ correlations between measures of strength, paresis and contracture, and with clinical and objective measures of spasticity, for elbow and knee joints.

|          | Elbow joint | Knee joint |
|----------|-------------|------------|
|          | Paresis     | Contracture| Paresis     | Contracture|
| Strength | $-0.383$ (<.001)$^a$ | $-0.001$ (.987) | $-0.474$ (<.001)$^a$ | $0.185$ (.048) |
| Paresis  | $-0.092$ (.274)  | $-0.186$ (.383) |
| Spasticity | Strength | Paresis | Contracture | Strength | Paresis | Contracture |
| MAS      | $-0.363$ (<.001)$^a$ | $0.269$ (.001)$^a$ | $0.033$ (.696) | $-0.148$ (.116) | $-0.069$ (.449) | $0.262$ (.002)$^a$ |
| BioTone$^b$ | $-0.268$ (.003)$^b$ | $0.289$ (.001)$^b$ | $-0.001$ (.991) | $0.320$ (<.001)$^a$ | $0.078$ (.398) | $-0.040$ (.654) |

$^a$ Significant at $p < .017$.

$^b$ BioTone measure for elbow was Ds from stretch-reflex test, and for knee was RI from pendulum test.

**Discussion**

Although this relationship implies improving arm muscle strength would restore hand grip force. Data in figure 5(A) is discouraging in this regard, as the data suggests there is a stronger relationship between arm muscle and hand grip strength with diminishing strength; indeed, there were no participants with dimin-
ished or absent elbow muscle strength (or both flexors and extensors) that had preserved grip strength. Given that strength deficits with UMNS increase distally (Sukal-Moulton et al 2014) it makes sense that this would be the case. It is less clear, however, why there were also no cases of preserved elbow strength in the absence of grip strength. This would suggest a single mechanism may be responsible for impairing muscle force generating capacity of the entire limb.

Contracture and paresis
Muscle contractures are a common secondary condition with UMNS that prevents full functional range of movement and are a source of discomfort and pain (Skalsky and McDonald 2012). It is reported that half of stroke patients develop at least one contracture within 6 months of neurological injury (Kwah et al 2012). In spinal cord injury patients, the time between injury and admission to rehabilitation correlated significantly with increased incidence of contractures, especially for higher level injuries (Yarkony et al 1985). Although the precise mechanisms that cause contracture are unknown, contracture formation may begin in the acute and sub-acute phases post-injury, and may be related to the structural changes that occur with immobilized muscle and soft-tissue following paresis (for a detailed review see (Gracies 2005a)) or perhaps part of an on-going process of muscle shortening due to the hyper-excitability of the stretch reflex (O’Dwyer et al 1996, Ada et al 2006).

For the patient populations participating in this study, the ABI (stroke) cohort had the highest elbow contracture, having a mean of about 15°, as shown in figure 3(A)(top). For the knee joint, flexor contracture (as we only measured passive knee extension) was lowest for the ABI cohort and highest for the CP cohort. However, there was significant variability in these data and differences between cohorts for elbow and knee contracture were found to be non-significant. Unfortunately, the published studies on contracture have not compared different UMNS cohorts, and typically only elbow flexor contracture is reported (O’Dwyer et al 1996, Ada et al 2006) for the upper extremity. This again points to a need for standardized protocols and measurement tools.

Paresis, as defined by Gracies (2005a), is ‘decreased voluntary motor unit recruitment, i.e. inability or difficulty to voluntarily recruit skeletal motor units to generate torque or movement’. In this sense, isometric strength of muscles is a measure of paresis. In our study, however, we quantified paresis differently, although still consistent with the formal definition above. The envelope of AROM (as induced by the patient) must, by definition, be contained within the envelope of PROM (as induced by a therapist). The difference in these envelopes (i.e. their non-overlapping end or limit regions) represents the degree to which one can fully extend or fully flex their joint within its limits of motion, and therefore should represent the minimal length of the agonist muscle at which motor units can be recruited within their non-contracted range. The passive-active limit angle (see figure 1(A)) is therefore our measure of paresis, and was computed by subtracting the passive limit angles (max and min) from the active limit angles (max and min), providing a measure of flexor and extensor paresis that is independent of contracture.

Data in table 2 show consistently high paresis (~18°) in elbows of the ABI cohort, slightly less but highly variable for the MS cohort, and lowest for the SCI cohort. The only statistically significant difference found was between ABI and SCI groups. For the knee (figure 3(B)), although not statistically significant, the degree of paresis was highest in the SCI cohort and lowest for ABI and CP cohorts. Again, there are no studies that have compared different UMNS cohorts to which we can corroborate these findings, but our findings appear consistent with the clinical presentation of these patient groups.

As expected, we found an inverse correlation between paresis and muscle strength for the elbow and knee (both significant at $p < .001$); however, figure 5(B) illustrates that the relationship is not a linear one. For both the elbow and knee the patients with the highest paresis were the weakest, and the patients with the lowest paresis were the strongest, as would be expected. However, for the elbow, the majority of participants clustered into a region of moderate-low strength and moderate-high paresis. For the knee the clustering was more spread out along the vertical (strength) axis. Clearly the inverse correlation is being driven by the values along the axes; without these observations the correlation analysis would have revealed a non-significant relationship. One could argue therefore that the relationship between voluntary strength at mid-range (90°) and the inability to generate force at shortest muscle lengths could be caused by two different mechanisms; the former being the ability to recruit (via descending pathways) sufficient motor units (Ada et al 2003) and the latter being a shift in reciprocal inhibition (Levin et al 2000) that effectively disallows the shortened muscle to activate when the stretched antagonist is nearing its tonic reflex activation threshold. As such, one would expect to see those with highest paresis to have high spasticity of both the flexors and extensors. Figure 5(B) illustrates this to some extent for the elbow.

Inter-relationships between strength, paresis, contracture and spasticity
Motor disorder associated with UMNS is the result of the interaction between muscle paresis, soft tissue contracture, and hyper-excitability of the stretch-reflex or spasticity. The once prevailing view that spasticity is the primary source of functional motor disorder in UMNS has been questioned (Fellows et al 1994) and several studies show there is a closer link between strength deficit and motor performance, than between muscle tone.
and motor performance (Ada et al 2006, Harris and Eng 2007). Nevertheless, the inter-relationships between these different components of UMNS remain elusive.

Although there is some evidence that spasticity may lead to contracture (O’Dwyer et al 1996, Ada et al 2006), other studies (Pohl et al 2007) including our present study do not support that there is a clear association. Although clinical MAS score for the knee extensors correlated with contracture of knee flexors (table 3), there were no other significant correlations between contracture and objective or clinical measures of spasticity. This contradicts the study by Ada et al (2006), which suggested a causal relationship. It is import to note that they examined the time course from injury to 1 year post-stroke, while our study was a cross-sectional analysis where >90% of participants were >1 year post injury/onset. It may be that the relationship is emergent during the recovery process but diminishes over time, or it could be that the two phenomena occur simultaneously but are not directly related.

Contracture was also unrelated to paresis (as measured by the passive-active limit angle) or isometric voluntary strength for both the elbow and the knee. This is a curious finding because ultimately the two sum up to

Figure 3. Joint paresis as measured by passive-active limit angle (dark gray bars) and joint contracture as measured by passive limit angle (light gray bars) for (A) elbow flexors and extensors (pooled) and (B) knee extensors. The left plots show results across for cohorts and the right plots show results by therapist rated MAS score. The * symbol and dashed lines indicate statistical differences ($p < .05$) between pairs. Error bars are 95% confidence intervals.
reflect the envelope of functional (voluntary) range of opposing muscle lengths, but the data show they did not do so in a proportional way. The fact that just as many patients with contractures had full use of the available range (low paresis) as those who did not (high paresis), and similarly for those without contractures, suggests the two are unrelated.

Our results show that the paresis was inversely (and non-linearly) related to strength, and was positively correlated with clinical and objective measures of spasticity for the elbow (but not the knee, as shown in table 3). The inverse relationship between strength and muscle spasticity for the elbow is supported by other studies. Bohannon et al (1987) reported a similar correlation between elbow flexor strength deficit and Ashworth grading (except theirs is positive because they used strength deficits rather than absolute strength) of elbow flexors for patients with stroke, and we similarly found no relationship between elbow flexor strength and spasticity of the elbow extensors. They also reported elbow flexor strength deficit in the stroke group was 74% of normal (their non-involved side). In our study the stroke (ABI) patients elbow flexor strength was about 70% lower than normal (compared to controls).

Although this finding might imply that increasing strength (via increase in motor unit recruitment) could reduce spasticity, it is likely that the causal factors that establish the relationship are not reversible. Indeed, while several studies show modest gains in strength with various exercise interventions, there is usually little to no effect on tone or spasticity (Sharp and Brouwer 1997). A possible explanation for this is that spasticity is present when the level of true paresis (muscle lengths that cannot generate force) is beyond the contractured range (unusable muscle lengths) and into the usable (passively free) muscle length range. In normal functioning joints a force can be produced at the shortest anatomical length of the muscle; this implies the tonic activation threshold length (below which no force can be generated) is normally well outside the anatomical range (Jobin and Levin 2000). Patients with high paresis (in our study) could not generate forces at muscle lengths that were within their passively free range, implying that the activation threshold length of the muscle was within their biomechanical range. Therefore, interventions that improve muscle motor unit recruitment may be able to improve strength, but it is unclear if they have an impact on tonic activation thresholds.

But why would this result in spasticity? The shortest length of the agonist determines the longest length of the antagonist. According to the activation threshold theory put forth by Levin and colleagues, during stretch

![Figure 4. Normalized (to BW) isometric strength for (A) elbow flexors and extensors (pooled, left panels) and (B) knee extensors (right panels) for cohorts (top panels) and therapist rated MAS score (bottom panels). The * symbol and dashed lines indicate statistical differences (p < .05) between pairs. Error bars are 95% confidence intervals.](imageURL)
the activation threshold where muscle is always active moves into the biomechanical range (as with a shortened muscle, this threshold length is normally outside the biomechanical range). If the tonic stretch-reflex activation is near the anatomical length of the (stretching) muscle, it would make sense that the opposing (shortening) muscle would be inhibited at rest to avoid a tonic stretch-reflex; even during functional movements, the maximal and minimal joint angles essentially represent static positions (zero velocity). Levin et al (2000) found that regions where no elbow torque could be produced corresponded to the predicted ingress of the activation threshold length into the biomechanical range. This is also supported by a recent study by Bhadane et al (2015) that found that spasticity of elbow flexors (MAS and Tardieu) was related to the standing elbow extension resting angle, which was always within the passive range of the elbow.

Could contracture be the result of the non-use of the paretic end regions where no forces can be generated (Gracies 2005a)? We found no relationship between these measures (indeed, generated from the same instrument, the FOG system), which only suggests they are not cross-sectionally related; if there is a temporal relationship within subjects as suggested by Ada et al (2006) it would not likely have been detected by our study. It is also completely unknown how our participants used their limbs in daily life, i.e. whether or not they achieve some neural stimulus at end regions of motion routinely, or whether their limbs are routinely immobile. It is relatively well established (Harvey et al 2011), though non-equivocal (Katalinic et al 2011), that passive stretching can help prevent contracture, but there is little evidence that stretching improves spasticity (Bovend’Eerdt et al 2008), possibly because it does not address end-range paresis and/or stretch-reflex regulation.

**Conclusions**

The study shows that comprehensive measures of muscle tone in the clinic using standardized and objective tools can be accomplished with the BioTone™ system. Benefits to the clinical trial community, both in academia and industry, could be significant not only in terms of improving quality and validity of outcomes, but more sites could be recruited to participate in trials that otherwise would not have access to high-tech equipment for measuring comprehensive outcomes.

**Clinical messages**

- Strength loss is characterized by reduced ability to generate isometric force, but also an inability to generate force at short muscle lengths, which defines a zone of paresis where the agonist will not activate.
- Muscle weakness and paresis as defined above were correlated with clinical and objective measures of spasticity for the elbow, and provides general support for the theory of stretch-reflex threshold regulation being a potential mechanism of spasticity.
• Contracture did not relate to any of the measures except for the knee where it correlated with clinical MAS score, suggesting that although clinical measures of spasticity were in good agreement with objective measures of spasticity for the elbow, the MAS score for the knee may be confounded by contracture.

Acknowledgments

The authors are indebted to the contributing staff and graduate students of the UNB Institute of Biomedical Engineering and Stan Cassidy Centre for Rehabilitation, Nova Scotia Rehabilitation Hospital in Halifax, and Spaulding Rehabilitation Hospital in Boston.

Funding acknowledgment

This work was supported by the Atlantic Canada Opportunities Agency, Atlantic Innovation Fund, Project # 195180.

Conflict of interest

The Authors declare that there is no conflict of interest.

ORCID iDs

Chris A McGibbon https://orcid.org/0000-0001-7849-7895

References

Ada L, Canning C G and Low S J 2003 Stroke patients have selective muscle weakness in shortened range Brain J. Neurol. 126 724–31
Ada L, O’Dwyer N and O’Neill E 2006 Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study Disabil. Rehabil. 28 891–7
Ansari N N, Naghdi S, Moammeri H and Jalaie S 2006 Ashworth scales are unreliable for the assessment of muscle spasticity Physiother. Theory Pract. 22 119–25
Bhadane M Y, Gao F, Francisco G E, Zhou P and Li S 2015 Correlation of resting elbow angle with spasticity in chronic stroke survivors Front. Neurol. 6 183
Bhimani R and Anderson L 2014 Clinical understanding of spasticity: implications for practice Rehabil. Res. Pract. 2014 279175
Bohannon R W 1986 Decreased isometric knee flexion torque with hip extension in hemiparetic patients Phys. Ther. 66 521–3
Bohannon R W 1987 Relationship between static strength and various other measures in hemiparetic stroke patients Int. Rehabil. Med. 8 125–8
Bohannon R W, Harrison S and Kinsella-Shaw J 2009 Reliability and validity of pendulum test measures of spasticity obtained with the Polhemus tracking system from patients with chronic stroke J. Neuroeng. Rehabil. 6 30
Bohannon R W and Smith M B 1987 Interrater reliability of a modified Ashworth scale of muscle spasticity Arch. Phys. Med. Rehabil. 69 406–7
Calota A, Feldman A G and Levin M F 2008 Spasticity measurement based on tonic stretch reflex threshold in stroke using a portable device Clin. Neurophysiol. 119 2329–37
Chen J J, Wu Y N, Huang S C, Lee H M and Wang Y L 2005 The use of a portable muscle tone measurement device to measure the effects of botulinum toxin type a on elbow flexor spasticity Arch. Phys. Med. Rehabil. 86 1655–60
Citaker S, Guclu-Gunduz A, Yazici G, Bayraktar D, Nazliel B and Irkec C 2013 Relationship between lower extremity isometric muscle strength and standing balance in patients with multiple sclerosis NeuroRehabilitation 33 293–8
Colebatch J G, Gandevia S C and Spira P J 1986 Voluntary muscle strength in hemiparesis: distribution of weakness at the elbow J. Neurol. Neurosurg. Psychiatry 49 1019–24
de Groot S, Dallmeijer A J, Besems P J, Lamberts M L, van der Woude I H and Janssen T W 2012 Comparison of muscle strength, sprint power and aerobic capacity in adults with and without cerebral palsy J. Rehabil. Med. 44 932–8
DiPiero N D et al 2015 Lower extremity strength is correlated with walking function after incomplete SCI Top Spinal Cord Inj. Rehabil. 21 133–9
Ekstrand E, Lexell J and Brog ard C 2015 Isometric and isokinetic muscle strength in the upper extremity can be reliably measured in persons with chronic stroke J. Rehabil. Med. 47 706–13
Ekstrand E, Lexell J and Brogard C 2016 Grip strength is a representative measure of muscle weakness in the upper extremity after stroke Top Stroke Rehabil. 23 400–5
Fellows S J, Krau C and Thilmann A F 1994 Voluntary movement at the elbow in spastic hemiparesis Ann. Neurol. 36 397–407
Fleuren J F et al 2009 Stop using the Ashworth scale for the assessment of spasticity J. Neurol. Neurosurg. Psychiatry 81 46–52
Gracies J M 2003a Pathophysiology of spastic paresis. I: Paresis and soft tissue changes Muscle Nerve 31 535–51
Gracies J M 2003b Pathophysiology of spastic paresis. II: Emergence of muscle overactivity Muscle Nerve 31 552–71
Guclu-Gunduz A, Citaker S, Nazliel B and Irkec C 2012 Upper extremity function and its relation with hand sensation and upper extremity strength in patients with multiple sclerosis NeuroRehabilitation 30 369–74
Guner S, Haghari S, Inanici F, Alsancak S and Aytekin G 2015 Knee muscle strength in multiple sclerosis: relationship with gait characteristics J. Phys. Ther. Sci. 27 809–13
Harris J E and Eng J J 1977 Paretic upper-limb strength best explains arm activity in people with stroke Phys. Ther. 87 88–97
Harvey L A, Glinksy J A, Katalinic O M and Ben M 2011 Contracture management for people with spinal cord injuries NeuroRehabilitation 28 17–20
Hasegawa R, Islam M M, Lee S C, Koizumi D, Rogers M E and Takeshima N 2008 Threshold of lower body muscular strength necessary to perform ADL independently in community-dwelling older adults Clin. Rehabil. 22 902–10
Ivanhoe C B and Reistetter T A 2004 Spasticity: the misunderstood part of the upper motor neuron syndrome Am. J. Phys. Med. 83 53–9
Jaeger R J, Yarkony G M and Roth E J 1989 Rehabilitation technology for standing and walking after spinal cord injury Am. J. Phys. Med. Rehabil. 68 128–33
Jobin A and Levin M F 2000 Regulation of stretch reflex threshold in elbow flexors in children with cerebral palsy: a new measure of spasticity Dev. Med. Child Neurol. 42 531–40
Katalinic O M, Harvey L A and Herbert R D 2011 Effectiveness of stretch for the treatment and prevention of contractures in people with neurological conditions: a systematic review Phys. Ther. 91 11–24
Kim C M, Eng J J and Whittaker M W 2004 Level walking and ambulatory capacity in persons with incomplete spinal cord injury: relationship with muscle strength Spinal Cord 42 156–62
Koo T K, Mak A F, Hung L and Dewald J P 2003 Joint position dependence of weakness during maximum isometric voluntary contractions in subjects with hemiparesis Arch. Phys. Med. Rehabil. 84 1380–6
Kumar R T, Pandyan A D and Sharma A K 2006 Biomechanical measurement of post-stroke spasticity Age Ageing 35 371–5
Kwah L K, Harvey L A, Diong J H and Herbert R D 2012 Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study J. Physiother. 58 41–7
Landry J, Sexton A, Hughes G and McBignon C A 2013 Limb strength measurement device US Patent Specification 9,028,433
Levin M F, Selles R W, Verheul M H and Meijer O G 2000 Deficits in the coordination of agonist and antagonist muscles in stroke patients: implications for normal motor control Brain Res. 853 352–69
Logan L R 2011 Rehabilitation techniques to maximize spasticity management Top Stroke Rehabil. 18 203–11
Malhotra S, Pandyan A D, Day C R, Jones P W and Hermens H 2009 Spasticity, an impairment that is poorly defined and poorly measured Clin. Rehabil. 23 651–8
Malhotra S et al 2008 An investigation into the agreement between clinical, biomechanical and neurophysiological measures of spasticity Clin Rehabil. 22 1105–15
McBignon C, Sexton A, Jones M and O’Connell C 2016 Quantification of elbow muscle tone from an instrumented manual stretch-reflex test Phys. Med. Rehabil. Res. 1 1–11
McBignon C A, Sexton A, Jones M and O’Connell C 2013 Elbow spasticity during passive stretch-reflex: clinical evaluation using a wearable sensor system J. Neurom. Eng. 10 611
Moseau N G and Gannotti M E 2015 Addressing muscle performance impairments in cerebral palsy: Implications for upper extremity resistance training J. Hand Ther. 28 91–9
Naghide S, Ansari N N, Mansouri K, Asgari A, Olyaei G R and Kazemnejad A 2008 Neurophysiological examination of the modified modified Ashworth scale (MMAS) in patients with wrist flexor spasticity after stroke Electromyogr. Clin. Neurophysiol. 48 35–41
O’Dwyer N J, Ada L and Neilson P D 1996 Spasticity and muscle contracture following stroke Brain J. Neurol. 119 1373–49
Pandyan A D, Price C I, Rodgers H, Barnes M P and Johnson G R 2001 Biomechanical examination of a commonly used measure of spasticity Clin. Biomech. 16 859–65
Pandyan A D, Van Wijck F M, Stark S, Vuadens P, Johnson G R and Barnes M P 2006 The construct validity of a spasticity measurement device for clinical practice: an alternative to the Ashworth scales Disabil. Rehabil. 28 579–85
Pasternak-Mladzika J, Mladziki Z, Bedzinski R and Baran B 2007 Objective measurements of muscle force in a group of after-stroke patients with hemiparesis Acta Bioeng. Biomech. 9 19–23
Paulis W D, Horemans H L, Brouwer B S and Stam H J 2010 Excellent test-retest and inter-rater reliability for Tardieu Scale measurements with inertial sensors in elbow flexors of stroke patients Gait Posture 33 185–93
Pisano F, Miscio G, Del Conte C, Pianca D, Candello E and Colombo R 2000 Quantitative measures of spasticity in post-stroke patients Clin. Neurophysiol. 111 1015–22
Pohl M, Mehrholz J, Rockstroh G, Buckiern S and Koch R 2007 Contractures and involuntary muscle overactivity in severe brain injury Brain Inj. 21 421–32
Pohl P S et al 2002 Rate of isometric knee extension strength development and walking speed after stroke J. Rehabil. Res. Dev. 39 651–7
Sharp S A and Brouwer B J 1997 Isokinetic strength training of the hemiparetic knee: effects on function and spasticity Arch. Phys. Med. Rehabil. 78 1231–6
Skalsky A J and McDonald C M 2012 Prevention and management of limb contractures in neuromuscular diseases Phys. Med. Rehabil. Clin. North Am. 23 675–87
Stevens S L, Fuller D K and Morgan D W 2013 Leg strength, preferred walking speed, and daily step activity in adults with incomplete spinal cord injuries Top Spinal Cord Inj. Rehabil. 19 47–53
Sukal-Moulton T, Krosschel K J, Gaebler-Spira D J and Dewald J P 2014 Motor impairment factors related to brain injury timing in early hemiparesis. Part I: expression of upper-extremity weakness Neurorehabil. Neural Repair. 28 13–23
Whelan A, Sexton A, Jones M, O’Connell C and McBignon C A 2018 Predictive value of the pendulum test for assessing knee extensor spasticity J. Neurom. Eng. Rehabil. 15 68
Wisser J, Manack A and Brainin M 2013 Toward an epidemiology of poststroke spasticity Neurology 80 S13–9
Wren T A and Engberg J R 2009 Normalizing lower extremity strength data for children, adolescents, and young adults with cerebral palsy J. Appl. Biomech. 25 193–202
Yarkony G M, Bass L M, Keenan V III and Meyer P R Jr 1985 Contractures complicating spinal cord injury: incidence and comparison between spinal cord centre and general hospital acute care Paraplegia 23 265–71
Yelnik A P, Simon O, Parratte B and Gracies J M 2010 How to clinically assess and treat muscle overactivity in spastic paraparesis J. Rehabil. Med. 42 801–7
Yeung E H, Chow D H and Su Y H 2012 Kinematic and electromyographic studies on unaided, unilateral and bilateral crutch walking in adolescents with spastic diplegia Prosthet. Orthot. Int. 36 63–70