Robotic Identification of Kinesthetic Deficits After Stroke

Jennifer A. Semrau, PhD; Troy M. Herter, PhD; Stephen H. Scott, PhD; Sean P. Dukelow, MD, PhD

Background and Purpose—Kinesthesia, the sense of body motion, is essential to proper control and execution of movement. Despite its importance for activities of daily living, no current clinical measures can objectively measure kinesthetic deficits. The goal of this study was to use robotic technology to quantify prevalence and severity of kinesthetic deficits of the upper limb poststroke.

Methods—Seventy-four neurologically intact subjects and 113 subjects with stroke (62 left-affected, 51 right-affected) performed a robot-based kinesthetic matching task with vision occluded. The robot moved the most affected arm at a preset speed, direction, and magnitude. Subjects were instructed to mirror-match the movement with their opposite arm (active arm).

Results—A large number of subjects with stroke were significantly impaired on measures of kinesthesia. We observed impairments in ability to match movement direction (69% and 49% impaired for left- and right-affected subjects, respectively) and movement magnitude (42% and 31%). We observed impairments to match movement speed (32% and 27%) and increased response latencies (48% and 20%). Movement direction errors and response latencies were related to clinical measures of function, motor recovery, and dexterity.

Conclusions—Using a robotic approach, we found that 61% of acute stroke survivors (n=69) had kinesthetic deficits. Additionally, these deficits were highly related to existing clinical measures, suggesting the importance of kinesthesia in day-to-day function. Our methods allow for more sensitive, accurate, and objective identification of kinesthetic deficits after stroke. With this information, we can better inform clinical treatment strategies to improve poststroke rehabilitative care and outcomes. (Stroke. 2013;44:3414-3421.)

Key Words: kinesthesia ■ proprioception ■ stroke

Proprioception has been defined as the sense of limb geometry (position sense) and limb motion (kinesthesia).1 Proprioceptive deficits affect more than half of stroke survivors2–4 and can lead to significant problems with balance,5 coordination,6 and performance of activities of daily living.5,6 Unfortunately, clinicians are faced with significant challenges to measure proprioception accurately, because clinical assessments of proprioception have been identified as insensitive, unreliable, and subjective.8 We commonly see patients after stroke who are unable to perform activities of daily living, such as brushing their hair, or reaching for a cup; yet current clinical assessments make it difficult to discern whether the underlying problem is a result of damage to the motor system, sensory system, or both. The current situation in stroke rehabilitation is problematic, because poststroke proprioceptive deficits are recognized as important and exceedingly common, but there is limited ability to identify their nature or magnitude accurately.

The use of robotic technology to advance stroke recovery and rehabilitation is a rapidly developing field. Work from many groups has shown much promise for stroke rehabilitation through robotics.9–13 As well, a growing area of research is the use of robotic tools to quantify sensorimotor dysfunction poststroke.14–16 Robotics promise increased precision and accuracy of measurement, in addition to better reliability compared with standardized observer-based ordinal scales. Work from our group has detailed the success of using robotic technology to objectively identify and assess position sense deficits. We have found that position sense deficits occur in ≈50% of stroke patients.4,7 However, the incidence and severity of kinesthetic deficits is largely unknown after stroke. Although many rehabilitation clinicians suggest that intact kinesthesia is important for recovery, there are few standardized ways to assess it. By developing techniques to quantify and assess kinesthesia, we will be better able to develop and monitor rehabilitative strategies directed at individuals who display predominantly kinesthetic and proprioceptive deficits. We hypothesize that stroke may result in significant kinesthetic deficits that can be reliably quantified using robotic technology.

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From the Hotchkiss Brain Institute (J.A.S., T.M.H., S.P.D.), and Department of Clinical Neurosciences (J.A.S., T.M.H., S.P.D.), University of Calgary, Alberta, Canada; Centre for Neuroscience Studies (T.M.H., S.H.S.), Department of Anatomy and Cell Biology (S.H.S.), and School of Medicine (S.H.S.), Queen’s University, Kingston, Ontario, Canada; and Department of Exercise Science, University of South Carolina, Columbia (T.M.H.).

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Correspondence to Sean P. Dukelow, MD, PhD, 1403 29th St NW, Foothills Medical Centre, South Tower, Room 905, Calgary, Alberta T2N 2T9, Canada. E-mail spdukelo@ucalgary.ca

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Methods

Subjects

Subjects with stroke were recruited from the acute stroke unit at Foothills Hospital and the stroke rehabilitation units at Foothills Hospital and Dr Vernon Fanning Center in Calgary, Alberta, Canada. Control subjects were recruited from the community. Subjects with stroke were included if they had first onset, clinically diagnosed unilateral stroke and could understand the task instructions. Forty-three subjects were excluded because of visuospatial neglect, significant active medical issues (eg, angina, uncontrolled hypertension, respiratory distress), bilateral stroke (confirmed via scan), other neurological diagnoses, or significant issues caused by previous orthopedic injuries to either upper limb. This study was approved by the University of Calgary Ethics Board, and all subjects provided informed consent.

Clinical Assessments

Clinical assessments for subjects were conducted by either the study physician or a study therapist with expertise in stroke rehabilitation. Subjects with stroke were assessed using the Edinburgh Handedness Inventory,17 Chedoke–McMaster Stroke Assessment–impairment inventory for the arm and hand (CMSA), the Purdue Peg Board (PPB), the Thumb Localizing Test (TLT, a clinical assessment of position sense),18 the Behavioral Inattention Test (BIT) of visuospatial neglect, and the Functional Independence Measure (FIM). Subjects with hemineglect (BIT score <130) were excluded. For all clinical assessments, we observed no significant differences between left-affected (LA) and right-affected (RA) subjects (unpaired t-test; P>0.05).

As ≈15% of stroke survivors with unilateral brain lesions exhibit ipsilesional deficits poststroke,19 in the remainder of the article we will refer to subjects with stroke by their most affected side of their body.

Kinesthesia Task

The kinesiological instrument for normal and altered reaching movements (KINARM, BKIN Technologies, Kingston) exoskeleton robotic device has been previously described7,15 (Figure 1 in the online-only Data Supplement). Subjects sat in the wheelchair base of the robot with their arms supported against gravity by the exoskeleton. Subjects performed a kinesthetic matching task with vision of the upper limbs occluded. Before beginning each trial, the robot moved 1 arm to 1 of 3 possible locations in the workspace. Simultaneously, subjects saw a red circle on the opposite side of the workspace (located at 1 of 3 possible locations, mirrored across the midline) and a white circle representing the position of the index finger of their opposite arm. Subjects were asked to move the white circle into the red circle, which effectively brought the 2 limbs to a mirrored start position. Before beginning each trial, the robot moved the subjects’ arm (passive arm) to 1 of the other 2 targets at a preset speed (Figure 1). The straight-line movement between targets was 20 cm in length, with a bell-shape velocity profile with a peak speed of 0.28 m/s. As soon as subjects felt movement of their passive arm, they were instructed to mirror-match this movement with the opposite arm (active arm). For subjects with stroke, the robot only ever moved the most affected upper limb, and subjects actively mirror-matched with their opposite arm. For control subjects, both arms were tested. All subjects performed 36 total movements, for a total of 6 movements in each of 6 movement directions (Figure 1). Subjects were also required to complete the movement within 10 s after the robot initiated movement. If subjects did not fulfill this time requirement the movement was categorized as a non-movement.

Data Analysis

Movement in the x-direction of the active arm was mirror-transposed to compare the direction of movement of the 2 limbs. Minor compliance in the robots created small variations in peak speed (±0.028 m/s) and distance (±0.016 cm) of the passively moved arm, and these variations were accounted for in the analysis. The mean and variance of 4 parameters were used to characterize the spatial (X,Y) and temporal (hand speed) performance of the subject. These included:

1. Response Latency (RL; Figure 1C). The difference in movement onset time between the active and passive arms. Movement initiation was defined as the point when subjects reached 10% of the maximum movement speed.

2. Peak Speed Ratio (PSR; Figure 1C). The ratio between the maximum hand speed of the active and passive arms. A ratio of 1 indicates perfect speed matching, and ratios <1 or >1 would indicate that the active arm moved slower or faster, respectively, than the passive, robotically moved arm.

3. Initial Direction Error (IDE; Figure 1D). This parameter quantified the sense of direction by measuring absolute angular deviation at peak hand speed between the active and passive arms.

4. Path Length Ratio (PLR; Figure 1D). This quantified a subject’s ability to match the distance of hand movement. Movement end was defined as the point when subjects slowed to 10% of maximum speed after achieving maximum speed. PLR was calculated by dividing the total movement length of the subject’s active arm by the length moved by the passive arm. A ratio of 1 indicated perfect matching, and ratios <1 or >1 indicated that movement of the active arm was smaller or larger in magnitude, respectively, than the passive arm moved by the robot.

Average responses for RL, PSR, IDE, and PLR were calculated for each subject by taking the mean response of all 36 trials performed. To determine kinesthetic deficits in the stroke groups, we derived normative control ranges for RL, PSR, IDE, and PLR from the distribution of control data as the 95% confidence interval (95% CI, 0%–95%) for 1-sided metrics (IDE, RL) and 2-sided metrics (2.5%–97.5%; PLR, PSR).

Variability

For the 4 abovementioned measures, we computed variability as the standard deviation across all movements within a single subjects’ performance on that particular parameter (RL variability [RLv], PSR variability [PSRv], IDE variability [IDEv], and PLR variability [PLRv]).

Statistical Analyses

To determine statistical significance for each of the abovementioned measures, we calculated normative ranges derived from the 95% CI for 1-sided measures (IDE, RL, all variability measures) and 2-sided measures (PLR, PSR) of our control group (Figure 2C, grey box). Establishing this control range (95% CI) allows us to determine whether individuals in our stroke group display abnormal behavior relative to the behavior of control subjects. Those individuals lying outside of the 95% CI are significantly different from controls with P<0.05. Linear regressions were performed to compare robotic measures with clinical assessments, and Bonferroni corrections adjusted for multiple comparisons where needed.

Results

Subjects

We measured kinesthesia in 74 controls and 113 subjects with subacute stroke. Subject demographics and clinical measurements are described in Table 1. Despite all subjects with stroke having a unilateral lesion, some demonstrated mild bilateral impairments, with the ipsilesional arm being affected in 9 predominantly LA and 3 predominantly RA stroke subjects. The majority of these individuals had CMSA arm scores of 6, with 2 having CMSA arm scores of 5 for the ipsilesional arm. In general, patients in the LA and RA groups exhibited relatively similar demographic and clinical profiles.

Many subjects with stroke demonstrated significantly impaired kinesthesia. Several stroke subjects failed to even...
acknowledge that the robot had moved their limb (a non-movement as described in Methods section). Although 95% of control subjects made $\leq 1$ non-movement error, 42% of LA (n=26; 95\% CI; $P < 0.05$) and 29% of RA (n=15; 95\% CI; $P < 0.05$) subjects with stroke had $>1$ non-movement error. In fact, 15\% of LA (n=9; 95\% CI; $P < 0.05$) and 14\% of RA (n=7; 95\% CI; $P < 0.05$) failed to move in $>25\%$ of trials. Non-movement trials were discarded from further analysis.

Response Latency

Compared with controls, many subjects with stroke had difficulty quickly and consistently initiating movements in response to the movement of the robot. In our exemplar control subject (Figure 1B), we see that this subject regularly initiated movement of the opposite arm quickly after the robot began to move (mean: RL, 314 ms; RLv, 148 ms). Exemplar data from a stroke subject (Figure 2A) show significant difficulty with initiation of matching movements and high variability in RL (mean: RL, 1288 ms; RLv, 613 ms). Forty-seven percent of LA (n=29; median, 817 ms) and 20\% of RA (n=10; median, 500 ms) subjects with stroke fell outside the normative control range for RL (95\% range, $<893$ ms; Figure 2C). Variability of RL (RLv; Figure IIA in the online-only Data Supplement) also showed that 42\% of LA (n=26; median, 355 ms) and 22\% of RA (n=11; median, 237 ms) strokes fell significantly outside of the normative control range (95\% range, $<414$ ms).

Peak Speed Ratio

Many subjects with stroke had difficulty modulating their active arm speed to match the speed of the passive arm. The exemplar control (Figure 1B) demonstrated good speed matching with a PSR close to 1 (mean PSR, 0.90) and low variability (mean PSRv, 0.13). In Figure 2A, a LA exemplar subject with stroke displayed low PSR (mean, 0.66), with high variability (PSRv; mean, 0.36). Similarly, a RA exemplar (Figure 2B) subject with stroke displayed high PSR (mean, 1.69), with high variability (PSRv; mean, 0.42). Thirty-two percent of LA (n=20; median, 1.02) and 27\% of RA (n=14; median, 1.11) subjects with stroke had difficulty matching speed, with values significantly outside the normative control range for PSR (95\% range, 0.80–1.40; Figure 2D). Additionally, many subjects with stroke demonstrated high variability in matching the
speed of the passive arm (PSRv), with 44% of LA (n=27) and 39% of RA (n=20) falling significantly outside the normative control range (95% range, <0.37; Figure IIB in the online-only Data Supplement).

Initial Direction Error
Compared with controls, subjects with stroke commonly failed to match the direction accurately or consistently of the passive movement generated by the robot. Exemplar control data are shown in Figure 1A, where the subject accurately mirror-matched the direction the robot had moved their left arm (mean IDE, 15.9º; mean IDEv, 14.9º). In contrast, exemplar stroke subjects displayed high IDE (Figure 3A; mean, 54.9º; Figure 3B; mean, 55.7º) and IDEv (Figure 3A; mean, 55.7º; Figure 3B; mean, 45.6º), suggesting impaired sense of direction.

We found that the majority of subjects with stroke (69% LA [n=43]; median, 28.0º; 49% RA [n=25]; median, 22.1º) made significantly larger IDE errors than 95% of the controls (95% range, <22.4º; median, 14.8º). In addition to making larger IDE errors, subjects with stroke were also more variable about the IDE errors they made (IDEv). Fifty percent of LA strokes (n=31; median, 23.2º) and 26% of RA strokes (n=13; median, 16.4º) fell significantly outside the control range for IDEv (95% range, <23.2º; median, 12.0º; Figure IIC in the online-only Data Supplement).

Path Length Ratio
We measured PLR as a function of subjects’ ability to match the distance the robot moved the passive arm. The exemplar control (Figure 1A) accurately and consistently matched the length of the robot movement (PLR mean, 1.05; PLRv mean, 0.12). In contrast, the LA exemplar stroke subject (Figure 3A) had difficulty matching PLR with movements that were too long (PLR mean, 1.64) and highly variable (PLRv mean, 0.50) compared with the passive movement of the robot. We established a normative control range for PLR (95% range, 0.86–1.23; median, 1.06), where values below this range indicated a shorter movement distance and values above indicated a longer movement distance. Many subjects with stroke displayed abnormal PLR matching, where 42% of LA (n=26; median, 1.13) and 31% of RA (n=16; median, 1.10) subjects with stroke fell significantly outside of the normative range.
PLRv (Figure 3B; Figure IID in the online-only Data Supplement) showed that, in addition to poor PLR matching, both LA (56%; n=35; median, 0.34) and RA (37%; n=19; median, 0.26) subjects with stroke were more variable about the distance they moved to match movements (control: 95% range, <0.30; median, 0.18).

Parameter Summary
Figure 4 shows a summary of the number of parameters that failed as a function of subject group. Across the 8 parameters described previously (IDE, IDEv, PLR, PLRv, RL, RLv, PSR, PSRv), only 5% of control subjects were outside the normative reference range on >2 parameters. In contrast, 71% of LA (n=44) and 49% of RA (n=25) subjects with stroke failed ≥3 parameters.

Clinical and Anatomic Significance
We found that several of our kinesthetic parameters were significantly related to clinical measures. For subjects with stroke, nearly all parameters were significantly related to FIM and FIM motor subscore measures (Table 2; P<0.0063). Furthermore, IDE and IDEv were significant predictors of poor performance on almost all clinical measures evaluated (FIM, FIM motor subscore, PPB, CMSA arm, CMSA hand [IDE only], and TLT [P<0.0063]).

Additionally, we tested whether there was a difference in the mean scores on all clinical measures for subjects that failed the kinesthesia task versus those who did not. We found that subjects that failed kinesthesia performed significantly worse on clinical tests compared with subjects that passed kinesthesia (1-way ANOVA; FIM total: pass kinesthesia [mean±SD], 115.25±12.55; fail kinesthesia, 96.19±20.70; P<0.0083; F(1,110)=30.18; FIM motor: pass kinesthesia, 84.05±9.93; fail kinesthesia, 66.03±19.43; P<0.0083; F(1,110)=32.36; PPB: pass kinesthesia, 7.68±4.44; fail kinesthesia, 5.68±3.82; P<0.0083; F(1,110)=29.99; CMSA arm: pass kinesthesia, 6.20±1.23; fail kinesthesia, 4.41±2.19; P<0.0083; F(1,110)=41.23; CMSA hand: pass kinesthesia, 5.89±1.33; fail kinesthesia, 4.29±1.93; P<0.0083; F(1,110)=22.81; TLT: pass kinesthesia, 0.36±0.65; fail kinesthesia, 0.80±0.93; P<0.0083; F(1,110)=7.25; Bonferroni correction, α=0.05; n=6).

Anatomically, we observed that the majority of our stroke subjects experienced cortical, subcortical, or combined cortical and subcortical strokes (Table I in the online-only Data Supplement). Additionally, the majority of subjects in our LA and RA subject groups had middle cerebral artery strokes (LA, n=41; RA, n=35; Table II in the online-only Data Supplement). We observed significantly impaired kinesthesia in 76% of right middle cerebral artery strokes and 49% of left middle cerebral artery strokes.

Discussion
We found that many subjects with stroke had significantly impaired kinesthesia. Across the 8 parameters examined, 20% to 69% of stroke subjects were impaired on any individual parameter and 61% (n=69) were impaired on >3 parameters.
Many of the parameters measured demonstrated significant correlations with clinical measures.

**Robotic Quantification of Kinesthesia**

Implementing robotic technology for proprioceptive and motor assessments poststroke could benefit rehabilitation in several ways. Objective, quantitative, and sensitive measures should allow for a more accurate identification of impairments. In turn, this could allow for better tailoring of interventions to the individual and, ultimately, improved outcomes. Furthermore, better assessment tools could potentially result in more accurate prognostication around recovery.

It is important to highlight that we evaluated active movements of the less affected arm in all our stroke subjects. Using this methodology, subjects relied on sensation derived from the stroke-affected arm to signal and drive movement of the opposite arm. This technique has been modified from a widely accepted technique for examining position sense, known as position matching. We are currently unaware of other published quantitative measures to assess kinesthesia after stroke. The technique itself is not without limitations. In a previous study, we examined visually guided reaching in individuals with stroke. In that study, ≈20% of stroke subjects displayed deficits in ipsilesional reaching for a number of robotic parameters, with ≈30% having deficits in IDE. Additionally, in that study ≈20% of patients had ipsilesional motor deficits as measured by CMSA, whereas our sample had a smaller percentage (11%) of stroke subjects with mild motor impairments (CMSA, 5 or 6) of the ipsilesional arm. These individuals may have had difficulty with the present task because of motor impairment. However, when we removed these subjects from our data analyses and recalculated percent impairment in each parameter (Figures 2C, 2D, 3C, and 3D), we saw little change.

![Exemplar Left Affected Stroke - Hand Position](image1)

![Exemplar Right Affected Stroke - Hand Position](image2)

![Mean Initial Direction Error](image3)

![Path Length Matching](image4)
Clinical Scores

Table 2. Relationship of Kinesthetic Parameters to Clinical Scores

<table>
<thead>
<tr>
<th>Clinical Score</th>
<th>Parameters</th>
<th>IDE</th>
<th>IDEv</th>
<th>PLR</th>
<th>PLRv</th>
<th>RL</th>
<th>RLv</th>
<th>PSR</th>
<th>PSRv</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIM (total)</td>
<td></td>
<td>0.229*</td>
<td>0.171*</td>
<td>0.008</td>
<td>0.220*</td>
<td>0.107*</td>
<td>0.133*</td>
<td>0.021</td>
<td>0.109*</td>
</tr>
<tr>
<td>FIM (motor)</td>
<td></td>
<td>0.190*</td>
<td>0.139*</td>
<td>0.012</td>
<td>0.217*</td>
<td>0.105*</td>
<td>0.126*</td>
<td>0.010</td>
<td>0.127*</td>
</tr>
<tr>
<td>PPB</td>
<td></td>
<td>0.144*</td>
<td>0.112*</td>
<td>0.007</td>
<td>0.184*</td>
<td>0.052</td>
<td>0.053</td>
<td>0.004</td>
<td>0.091*</td>
</tr>
<tr>
<td>CMSA arm</td>
<td></td>
<td>0.195*</td>
<td>0.097*</td>
<td>0.017</td>
<td>0.170*</td>
<td>0.015</td>
<td>0.017</td>
<td>&lt;0.001</td>
<td>0.126*</td>
</tr>
<tr>
<td>CMSA hand</td>
<td></td>
<td>0.163*</td>
<td>0.080</td>
<td>0.026</td>
<td>0.185*</td>
<td>0.010</td>
<td>0.010</td>
<td>&lt;0.001</td>
<td>0.134*</td>
</tr>
<tr>
<td>TLT</td>
<td></td>
<td>0.130*</td>
<td>0.112*</td>
<td>0.085*</td>
<td>0.015</td>
<td>0.069*</td>
<td>0.032</td>
<td>0.061</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Linear regressions of clinical scores to kinesthetic robotic measures. Values indicate F value. CMSA indicates Chedoke–McMaster Stroke Assessment; FIM, Functional Independence Measure; IDE, initial direction error; IDEv, IDE variability; PLR, path length ratio; PLRv, PLR variability; PPB, Purdue Peg Board; PSR, peak speed ratio; PSRv, PSR variability; R, right; RL, response latency; RLv, RL variability; and TLT, Thumb Localizing Test. *P<0.0063 (Bonferroni correction, α=0.05; n=8).

Differences in LA and RA Strokes

Overall, we did not observe any significant differences in the severity of stroke between the 2 groups as measured by FIM, CMSA, and PPB. However, robotic measures of kinesthesia demonstrated a number of differences between LA and RA subjects with stroke. Overall, 17% more LA subjects exhibited impaired kinesthesia compared with RA subjects. This finding is potentially consistent with previous literature suggesting right hemispheric dominance for the processing of kinesthetic information.

Clinical Use of Measuring Kinesthetic Deficits

We observed that 61% of stroke survivors we tested had significant deficits in kinesthesia. This high percentage of subjects with kinesthetic deficits stresses the necessity for addressing proprioceptive deficits in a clinical setting. Our clinical experience has been that it is possible for proprioceptive deficits to be mistaken for motor deficits. A better understanding of the relative contribution of sensory and motor deficits to functional impairment after stroke is critical to developing a comprehensive treatment plan aimed at maximizing stroke recovery and functional outcomes.

As improved technology for assessment and robotics become more common in stroke rehabilitation centers, the possibility of implementing an assessment of kinesthesia, such as the one described here, becomes more achievable. However, the challenge remains to develop a treatment that specifically addresses kinesthetic deficits. Recent reviews of sensory retraining in stroke have not identified any treatment specifically targeted at kinesthesia. This is not surprising because, to date, no tools to identify and monitor patients accurately with kinesthetic deficits have existed. In a treatment setting, once kinesthetic deficits have been identified it is possible that existing strategies that are thought to aid sensory function, such as thermal or electric stimulation, could be attempted to determine if they benefit recovery. Additionally, we know that motor performance after stroke improves substantially with the implementation of repetitive motor training paradigms; so, repetitive training on kinesthetically focused tasks might be a potential treatment strategy. It is likely that this model be used to develop similar kinesthetic training paradigms to improve kinesthetic function after stroke.
Conclusions
We have developed a novel robotic assessment tool to quantify kinesthesia after stroke. Our robotic assessment found that many stroke subjects are significantly impaired in kinesthesia. Improved assessment and identification of sensory deficits in stroke may be fundamental to improving poststroke rehabilitation and recovery.

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Disclosures
Dr Scott is cofounder and chief scientific officer of BKIN Technologies, which commercializes the KINARM robotic technology.

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COVER TITLE: Robotics for kinaesthesia post-stroke

AUTHORS: 1,2Jennifer A. Semrau, PhD, 1,2,3,6Troy M. Herter, PhD, 3,4,5Stephen H. Scott, PhD, 1,2Sean P. Dukelow, MD, PhD
1Hotchkiss Brain Institute, 2Department of Clinical Neurosciences, University of Calgary, Calgary, Alberta, Canada; 3Centre for Neuroscience Studies, 4Department of Anatomy and Cell Biology, 5School of Medicine, Queen’s University, Kingston, Ontario, Canada; 6Department of Exercise Science, University of South Carolina, Columbia, South Carolina, USA.

ADDRESS: 1403 29th St NW
Foothills Medical Centre
South Tower – Room 905
Calgary, AB, T2N 2T9, Canada

CORRESPONDENCE:
Email: spdukelo@ucalgary.ca
Telephone: (403) 944-5930
Fax: (403) 944-0977

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FIGS/TABLES:

Supplemental Figure I: Picture of the KINARM robotic exoskeleton

Supplemental Figure II: Group data demonstrating variability of behaviour of subjects with stroke across all four kinaesthetic parameters.

Supplemental Table I: Number of stroke patients who fail the Kinaesthesia Task and Parameters by Lesion Location

Supplemental Table II: Number of subjects who fail the Kinaesthesia Task and Individual Parameters based on Vascular Territory of Stroke
Supplemental Figure I: Picture of the KINARM robotic exoskeleton
Supplemental Figure II: Cumulative sum histograms depicting variability for each of the 4 kinaesthetic parameters (Fig. 3A and B, Fig. 4A and B). Grey areas on each plot indicate the normative range (95% CI). We observed that stroke subjects displayed significantly increased variability across all 4 parameters as compared to controls.
Table SI. Number of stroke patients who fail the Kinaesthesia Task and Parameters by Lesion Location

<table>
<thead>
<tr>
<th>Kinaesthetic Parameter</th>
<th>IDE</th>
<th>IDEv</th>
<th>PLR</th>
<th>PLRv</th>
<th>RL</th>
<th>RLv</th>
<th>PSR</th>
<th>PSRv</th>
<th>Kinaesthesia Task</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cortical</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LA (17)</td>
<td>71%</td>
<td>41%</td>
<td>29%</td>
<td>37%</td>
<td>35%</td>
<td>32%</td>
<td>35%</td>
<td>32%</td>
<td>65% (11)</td>
</tr>
<tr>
<td>RA (18)</td>
<td>39%</td>
<td>11%</td>
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Abbreviations: LA, Left Affected; RA, Right Affected; IDE, Initial Direction Error; IDEv, Initial Direction Error Variability; PLR, Path Length Ratio; PLRv, Path Length Ratio Variability; RL, Response Latency; RLv, Response Latency Variability; PSR, Peak Speed Ratio; PSRv, Peak Speed Ratio Variability. *Four subjects did not fall into the above lesion location categories and were categorized as "Other". For two, initial acute imaging (performed within the first 24 hours of stroke) was inconclusive and while the treating stroke neurologist confirmed the clinical diagnosis of stroke, they did not obtain a follow-up scan. Further, one subject experienced a posterior spinal artery stroke at spinal level C1 and one was diagnosed with a sinus thrombosis.
Table SII. Number of subjects who fail the Kinaesthesia Task and Individual Parameters based on Vascular Territory of Stroke

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<tr>
<th>Kinaesthetic Parameter</th>
<th>IDE</th>
<th>IDEv</th>
<th>PLR</th>
<th>PLRv</th>
<th>RL</th>
<th>RLv</th>
<th>PSR</th>
<th>PSRv</th>
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Abbreviations: ACA, Anterior Cerebral Artery; MCA, Middle Cerebral Artery; PCA, Posterior Cerebral Artery; ICA, Internal Carotid Artery; BA, Basilar Artery; PICA, Posterior Inferior Cerebellar Artery; PA, Pontine Artery; VA, Vertebral Artery; LA, Left Affected; RA, Right Affected; IDE, Initial Direction Error; IDEv, Initial Direction Error Variability; PLR, Path Length Ratio; PLRv, Path Length Ratio Variability; RL, Response Latency; RLv, Response Latency Variability; PSR, Peak Speed Ratio; PSRv, Peak Speed Ratio Variability. *Four subjects did not fall into the above lesion location categories and were categorized as "Other". For two, initial acute imaging (performed within the first 24 hours of stroke) was inconclusive and while the treating stroke neurologist confirmed the clinical diagnosis of stroke, they did not obtain a follow-up scan. Further, one subject experienced a posterior spinal artery stroke at spinal level C1 and one was diagnosed with a sinus thrombosis.