FUNCTIONAL DEFICITS IN CHRONIC STROKE DEPEND ON EXTENT OF CONTRALESIONAL IMPAIRMENT AND ON THE SIDE OF BRAIN DAMAGE

A Thesis in
Kinesiology

by
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Previous research has detailed the hemisphere dependence and specific behavioral nature of non-paretic arm motor deficits in patients with unilateral stroke. We now examine whether these deficits depend on hemisphere of damage and severity of contralesional paresis by quantifying the effect of unilateral stroke on clinical tests of motor function. We recruited 48 left hemisphere damaged (LHD) participants, 62 right hemisphere damaged (RHD) participants, and 54 age matched control participants. Measures of motor function included: 1) Jebsen-Taylor Hand Function Test (JTHFT), 2) Grooved Pegboard Test, and 3) Grip strength. We measured the extent of contralesional impairment with the upper extremity component of the Fugl-Meyer (UEFM) assessment of motor impairment. Non-paretic arm motor deficits depended strongly on severity of contralesional arm impairment on all measures, but hemisphere specific deficits were only evident on some measures. LHD participants with severe paresis (UEFM ≤ 28) took, on average, 112% longer to complete the JTHFT than control participants using the left non-dominant arm ($p < 0.0001$), while the severe RHD group took, on average, 61% longer than control participants using the right arm ($p < 0.0001$). Thus, stroke survivors with the most severe paretic arm impairment, who must rely on their ipsilesional arm for daily activities, have the greatest motor deficit in the non-paretic arm. We recommend remediation of this arm to improve functional independence in this cohort of stroke patients.
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Introduction

Stroke is a major health problem in the United States that leaves many survivors with chronic motor impairment, including hemiparesis in the limbs that are on the opposite side of the body to the damaged brain hemisphere. A substantial body of research has now established that the non-paretic arm also can have motor deficits that limit performance of activities of daily living and thus functional independence (Wetter, Poole & Haaland, 2005; Swinnen et al., 2002; Schaefer, Haaland & Sainburg, 2009a; 2007; Quaney et al., 2010; Hermsdorfer et al., 1999a; 1999b; Hanlon, Buffington & McKeown, 2005; Haaland et al., 2009; Debaere et al., 2001; Chestnut & Haaland, 2008; Arya, Pandian & Kumar, 2017; Bustren, Sunnerhagen & Murphy, 2017; Semrau et al., 2017). These deficits are important clinically, if they produce functional performance deficits in chronic stroke patients with severe paresis, who are unable to carry out manipulations with the paretic arm. In this study, we assess non-paretic arm function through three standardized tests of motor performance: grip strength, Grooved Pegboard Test, and the Jebsen-Taylor Hand Function Test (JTHFT). This battery of upper limb motor performance assesses strength, distal dexterity, and a range of whole arm tasks that have been shown to reflect performance of activities of daily living. By comparing the performance of right-handed (premorbid status) stroke survivors to that of the same arm (right or left) of age-matched control participants, we ask whether the ipsilesional arm of stroke survivors displays deficits in these areas of motor performance. In addition, by
clustering stroke patients by degree of impairment in the contralesional arm, we ask whether ipsilesional motor deficits vary with contralesional impairment level. We characterize contralesional impairment level using the upper extremity portion of the Fugl-Meyer Assessment (UEFM) (Fugl-Meyer et al., 1975). Woytowicz et al. (2017) recently reported an extensive cluster analysis of 247 stroke patients, and identified a severely paretic group in the UEFM score range of 0-28, a moderately paretic group in the UEFM score range of 29-42, and a mildly paretic group in the UEFM score range 43-66. With the exception of mass finger flexion, 95-100% of the patients in the severe group were unable to perform any of the wrist and finger items of the UEFM. Thus, this group of stroke patients are unable to use the paretic arm for manipulation purposes in activities of daily living.

While motor deficits in the non-paretic arm of patients with unilateral stroke have been documented as early as 1967 (Wyke) more recent research has shown that these deficits are often functionally limiting and that they can persist throughout the chronic phase of stroke (Wetter, Poole & Haaland, 2005; Schaefer, Haaland & Sainburg, 2009a; Schaefer, Haaland & Sainburg, 2007; Haaland et al., 2009; Chestnut & Haaland, 2008; Arya, Pandian & Kumar, 2017; Bustren, Sunnerhagen & Murphy, 2017; Semrau et al., 2017; Mutha, Haaland & Sainburg, 2013; Schaefer et al., 2012; Mutha, Haaland & Sainburg, 2011a; 2011b; Mutha, Sainburg & Haaland, 2010; Poole, Sadek & Haaland, 2009; Schaefer, Haaland & Sainburg, 2009; Haaland, 2006; Haaland et al., 2004; Prestopnik et al., 2003a; Prestopnik et al., 2003b; Haaland, Harrington & Knight, 2000; Pohl & Winstein, 1999; Winstein, Merians & Sullivan, 1999). In fact, studies of non-
paretic arm function in chronic stroke patients have reported performance deficiencies on a number of clinical tests, including the Purdue Pegboard Test (Rapin, Tourk & Costa, 1966), the Jebsen-Taylor Hand Function Test (Schaefer, Haaland & Sainburg, 2009), and a variety of tests that directly assess or simulate activities of daily living (Wetter, Poole & Haaland, 2005; Desrosiers et al., 1996; Haaland et al., 2012). Furthermore, significant deficits in movement coordination and accuracy have been shown through studies that use motion analysis (Schaefer, Haaland & Sainburg, 2009a; Haaland et al., 2009; Mutha, Haaland & Sainburg, 2013; Schaefer et al., 2012; Mutha, Haaland & Sainburg, 2011a; 2011b; Schaefer, Haaland & Sainburg, 2009; Haaland et al., 2004; Prestopnik et al., 2003b; Haaland & Harrington, 1996; Haaland & Harrington, 1989a;1989b; Weinstein et al., 1997; Weinstein & Pohl, 1995; Mani et al., 2014; Mutha, Haaland & Sainburg, 2012; 2010; Esparza, et al., 2003; Pohl, Weinstein & Onla-Or, 1997). Some of these studies have indicated that the nature of ipsilesional motor deficits varies with the hemisphere of damage (Haaland et al., 2009; Mutha, Haaland & Sainburg, 2013; Schaefer et al., 2012; Mutha, Haaland & Sainburg, 2011a; 2011b; Pohl & Weinstein, 1999; Weinstein & Pohl, 1995; Mani et al., 2014; Mutha, Haaland & Sainburg, 2012; 2010; Mutha et al., 2014. These studies have proposed that stroke-related non-paretic arm motor deficits result, at least in part, from a loss of the contributions of the ipsilateral hemisphere to motor control, and that right- and left-hemisphere damage lead to deficits in different aspects of motor control (Mutha, Haaland & Sainburg, 2013; Schaefer et al., 2012; Weinstein & Pohl, 1995; Sainburg, 2014; Tretriluxana et al., 2009). However, while these studies have examined ipsilesional motor deficits using detailed kinematic analysis of arm and hand
kinematics, it remains to be determined whether functional deficits, measured by standardized clinical tests, vary with extent of contralesional impairment and with side of brain damage following stroke.

In this study we examine 110 stroke survivors, distributed across a large range of paretic arm impairment levels, with both right and left hemisphere damage. All participants completed a battery of four standardized motor evaluations. We grouped our subjects by paretic arm severity, defined by the Fugl-Meyer Assessment scores, as recommended by Woytowicz et al. (2017), into three severity groups, Mild, Moderate, and Severe. We hypothesize that 1) ipsilesional motor deficits are produced by the loss of contributions of the ipsilateral hemisphere to control of the non-paretic arm, and 2) that greater non-paretic arm impairment should reflect a reduction in the contribution of ipsilesional hemisphere control to both arms. We thus predict that ipsilesional deficits will vary with extent of contralesional impairment. We also hypothesize that some tasks should recruit greater contributions from one hemisphere because of the nature of the task. Therefore, we predict that the effects of hemisphere should vary with task.

**Methods**

**Participants**

Sixty-two right hemisphere damaged (RHD) and 48-left hemisphere damaged (LHD) chronic unilateral stroke survivors, and 54 age- and education- matched control participants were tested at the Penn State Milton S. Hershey Medical Center. Table 1
includes participant characteristics and demographic information). Participants were screened and excluded based on history of (1) hospitalization for substance abuse and/or psychiatric diagnosis such as psychosis; (2) non-stroke neurological diseases for the stroke survivors and all neurological diagnoses for the control subjects; (3) brain stem or bilateral lesions; and (4) peripheral movement restrictions, such as neuropathy or orthopedic disorders. Stroke participants were right handed prior to stroke, and all control participants were right-handed according to the 10-item version of the Edinburgh inventory used to assess handedness (Oldfield, 1971). The control group was randomly assigned to use either their left or right arm. Stroke participants always used their less-impaired, ipsilesional arm. The Pennsylvania State College of Medicine Institutional Review Board approved the study protocol, and consent was obtained from all participants.
a Statistics give mean ± standard deviation in years;

b Years post stroke

Table 1: Summary of participant information

<table>
<thead>
<tr>
<th>Group</th>
<th>Contra</th>
<th>Severity</th>
<th>N</th>
<th>Male</th>
<th>Female</th>
<th>Age^a</th>
<th>EDU^a</th>
<th>Chronicity^a,b</th>
</tr>
</thead>
<tbody>
<tr>
<td>LHC</td>
<td>Control</td>
<td></td>
<td>27</td>
<td>15</td>
<td>12</td>
<td>57.4 ± 11.3</td>
<td>15.3 ± 2.7</td>
<td>NA</td>
</tr>
<tr>
<td>LHD</td>
<td>Mild</td>
<td></td>
<td>32</td>
<td>23</td>
<td>9</td>
<td>61.9 ± 10.5</td>
<td>14.5 ± 2.5</td>
<td>2.0 ± 1.5</td>
</tr>
<tr>
<td>LHD</td>
<td>Moderate</td>
<td></td>
<td>6</td>
<td>4</td>
<td>2</td>
<td>52.4 ± 9.8</td>
<td>13.5 ± 2.0</td>
<td>5.4 ± 4.1</td>
</tr>
<tr>
<td>LHD</td>
<td>Severe</td>
<td></td>
<td>10</td>
<td>8</td>
<td>2</td>
<td>59.8 ± 12.2</td>
<td>14.6 ± 2.8</td>
<td>2.2 ± 1.6</td>
</tr>
<tr>
<td>RHC</td>
<td>Control</td>
<td></td>
<td>27</td>
<td>17</td>
<td>10</td>
<td>58.2 ± 10.9</td>
<td>14.9 ± 2.9</td>
<td>NA</td>
</tr>
<tr>
<td>RHD</td>
<td>Mild</td>
<td></td>
<td>26</td>
<td>20</td>
<td>6</td>
<td>59.5 ± 10.9</td>
<td>14.2 ± 2.7</td>
<td>3.1 ± 4.6</td>
</tr>
<tr>
<td>RHD</td>
<td>Moderate</td>
<td></td>
<td>8</td>
<td>4</td>
<td>4</td>
<td>64.6 ± 11.7</td>
<td>12.0 ± 0.5</td>
<td>3.5 ± 3.2</td>
</tr>
<tr>
<td>RHD</td>
<td>Severe</td>
<td></td>
<td>28</td>
<td>23</td>
<td>5</td>
<td>57.2 ± 11.4</td>
<td>13.5 ± 2.7</td>
<td>3.2 ± 2.9</td>
</tr>
<tr>
<td>LHD</td>
<td>(All)</td>
<td></td>
<td>48</td>
<td>35</td>
<td>13</td>
<td>60.3 ± 11.0</td>
<td>14.4 ± 2.5</td>
<td>2.5 ± 2.3</td>
</tr>
<tr>
<td>RHD</td>
<td>(All)</td>
<td></td>
<td>62</td>
<td>47</td>
<td>15</td>
<td>59.1 ± 11.3</td>
<td>13.6 ± 2.6</td>
<td>3.2 ± 3.7</td>
</tr>
</tbody>
</table>

Notes: L Left hand; R Right hand; HC Healthy control; HD Hemisphere damage; EDU education

Handedness

Edinburgh Handedness Inventory

We determined handedness using the Edinburgh Handedness Inventory (Oldfield, 1971). We recruited only right-handed participants because left-handers do not represent a behaviorally- (Oldfield, 1971) nor a neurologically- (Kim et al., 1993) homogenous population, while right-handers are a fairly homogenous population, with respect to both behavioral and neurophysiological measures (Perelle & Ehrman, 1994). In addition, our inability to recruit sufficient numbers of left-handed stroke participants who fit our
inclusion criteria prevents us from using handedness as an experimental factor.

“Handedness” assessment is based on pre-stroke status.

**Paretic Arm Evaluation**

**Upper Extremity Fugl-Meyer Assessment**

Stroke participants were given the Upper Extremity Fugl-Meyer Assessment (UEFM) to quantify impairment in the contralesional upper limb. Lin et al. (2009) reported psychometric characteristics of the Fugl-Meyer with three other tests (Action Research Arm Test (ARAT); Upper Extremity subscale of the Stroke Rehabilitation Assessment of Movement, and the Wolf Motor Function Test). Results indicated high correlations with the other tests at different points after stroke (Spearman Correlation .82-.96), substantial responsiveness (effect size .37-.52), and high inter-rater reliability (ICC=.92-.98). Based on the UEFM score, we stratified participants into 3 impairment groups: Mild, Moderate, and Severe. Woytowicz et al. (2017) reported a cluster analysis of 247 chronic stroke patients, identifying three impairment groups: Mild (UEFM >43, Moderate (UEFM = 29-42) and Severe (UEFM = 0-28). In the severe group, 95-100% were unable to perform wrist movements, mass finger extension, and the prehension items of the UEFM. It should be noted that this is a higher upper-end UEFM score for the severe group than was previously reported by Woodbury et al. (2007), but that study did not include chronic patients and had a smaller severe cohort.
Non-Paretic Arm Evaluations

Jebsen-Taylor Hand Function Test

The Jebsen-Taylor Hand Function Test (JTHFT) is a clinical assessment of unilateral arm function (Beebe & Lang, 2009) that includes a range of tasks that simulate the coordination requirements of functional daily activities (Jebsen et al., 1969; 1971). Beebe and Lange (2009) evaluated six upper limb functional tests in stroke patients, demonstrating that all six were highly correlated. They concluded that rehabilitation scientists and therapists should use whichever test is most appropriate to the particular environment, but that using more than one would be redundant. Of these tests (Jebsen-Taylor Hand Function Test, grip strength, Pinch Strength, ARAT, 9-Hole peg test, and the Stroke Impact Scale-Hand), the JTHFT showed the greatest responsiveness and highest correlations with the other tests. The Jebsen-Taylor Hand Function Test has whole arm tasks that mimic activities of daily living, and thus is considered to evaluate unilateral functional performance. It includes 7 timed tasks that require dexterity and arm coordination: stacking checkers, feeding, manipulating and lifting soup-type cans of two different weights, writing, and page turning (Jebsen et al., 1969). The movements required for the tasks are common to those used in activities of daily living.
**Grooved Pegboard Test**

The Grooved Pegboard Test is a timed dexterity test consisting of placing 25 small pegs in holes of different orientations as quickly as possible (Lafayette instrument). This assessment combines distal dexterity with perceptual orientation to place a peg with a raised spine into holes with grooves to accommodate those spines, but with a range of differing orientations. Pegs must be rotated to match the hole before they can be inserted. Standardized T-scores for the Grooved Pegboard Test were obtained using the Halstead-Reitan Battery (2004), which accounts for age, gender, education, and ethnic differences. Higher scores indicate faster performance.

**Grip strength**

Grip strength was measured using a hand dynamometer (Lafayette instrument), and maximum force was averaged across 3 trials, separated by short rest periods. The handgrip was adjusted to comfortably fit the participant’s hand. The participant stood with the elbow straight, fully extended at the side of the body while squeezing the dynamometer for approximately 5 seconds. Standardized T-scores were obtained using the Halstead-Reitan Battery (2004), which accounts for age, gender, education, and ethnic differences. We report in Kg, as it is the standard for dynamometer testing; however, Kg is not a measure of force, and can be converted to Newtons by multiplying by 9.81 m/s$^2$. 
Ideomotor Limb Apraxia

Ideomotor limb apraxia was measured using a standardized test for ideomotor limb apraxia in which participants were videotaped for later scoring (Haaland & Flaherty, 1984). Using only their ipsilesional arm, stroke participants were asked to imitate five nonrepresentative (e.g. hand behind head), five representative (e.g salute), and five pretend object use (e.g. brush teeth) movements. Each of the 15 items was scored as 1 (correct movement) or 0 (incorrect movement), resulting in a possible score of 0-15 points, with scores of less than 11/15 correct, indicating apraxia (see Haaland et al., 2000). Scoring was based on 1. Hand position error, 2. Arm position error, 3. Hand orientation, 4. Target location (gesture on wrong body part), 5. Body-part-as-object, and 6. Qualitative (uncoordinated movement). Errors in any of the six categories resulted in a zero for that item.

Statistics

We subjected our dependent measures to a two-way ANOVA with severity group (Control, Mild, Moderate, Severe) and hand group (Left, Right) as the factors. It is important to note that in stroke survivors, hand indicated the hemisphere that was damaged: LHD participants always used their left, ipsilesional arm, while RHD participants used their right arm for all ipsilesional measures. When warranted, post-hoc analysis was conducted using Students’ t-test, Bonferoni corrected as appropriate.
Results

Upper-Extremity Fugl-Meyer (UEFM)

Figure 1 shows the mean ± SE for Upper-Extremity Fugl-Meyer (UEFM) scores within each severity group \( F(2,104) = 507.51, p < 0.001 \). We found no main effects of hemisphere group \( F(1,104) = 1.49, p = .22 \), nor interactions, indicating that our LHD and RHD groups were fairly well matched for contralesional impairment level, within each impairment level.

Figure 1: shows the mean Upper Extremity Fugl-Meyer (UEFM) scores for our stroke survivors, separated into contralesional impairment group. Right side lines indicate the UEFM ranges used to classify participants into Mild, Moderate, and Severe groups. Means and standard errors of each group are shown.
The Jebsen-Taylor Hand Function Test (JTHFT) has been shown to be a reliable test of unilateral motor function that is predictive of functional independence and includes simulated activities of daily living (Vliet Vlieland et al., 1996). The individual tasks include ‘page turning’ (flipping five 3X5 index cards on a table), simulated feeding (carrying and placing a bean with a spoon), writing (copying a sentence from an index card), picking up, transporting, and placing heavy and light soup-cans in a row on a surface, and stacking four checkers. Figure 2 shows the total time to complete all items on the JTHFT, separated by contralesional impairment group (Control, Mild, Moderate, Severe), and hand group, which reflected the ipsilesional hand for the stroke groups. It should be noted that the ordinate axis in Figure 2 represents the cumulative time to complete all the tasks in the JTHFT. A low value reflects effective performance, while high values indicate poor performance. Consistent with the patterns shown by Figure 2, the two-way ANOVA indicated a main effect of contralesional impairment \([F(3,156) = 30.6, p < 0.0001]\), a main effect of hand \([F(1,156) = 67.19, p < 0.0001]\), and an interaction between these factors \([F(3,156) = 6.86, p = 0.0002]\). The main effect of contralesional impairment severity is reflected in Figure 2 by the increasing time to complete the JTHFT, at each successive level of severity, from control participants (no impairment-column 1) to severe impairment (column 4). The main effect of hemisphere (ipsilesional hand) is reflected by the longer time to complete the JTHFT for the left hand of control participants (column 1), and for participants within each level of severity.
(columns 2-4), indicating a greater ipsilesional deficit in LHD vs. RHD participants, regardless of level of impairment. For example, the severe LHD participant group took, on average, 112% longer to complete the JTHFT than age matched control participants using the left non-dominant arm ($p < 0.0001$), while the severe RHD group took, on average, 61% longer than age matched control participants using the right arm ($p < 0.0001$). In both cases, this represents a substantial deficit in performance, but for LHD participants the deficit is substantially larger. This larger impact of contralesional severity on LHD than RHD participants was reflected by the interaction in our ANOVA reported above. Post-hoc analysis confirmed that each group (LHD: Mild, Moderate, Severe and RHD: Mild, Moderate, Severe) showed performance that was significantly longer than their age and arm matched control group.

Figure 2
Figure 2: JTHFT total time (in seconds) for control groups and stroke groups with mild, moderate, and severe contralesional paresis

In order to better understand which aspects of performance were modulated by the hemisphere that is damaged and by the extent of contralesional impairment, we subjected the time to complete each task of the JTHFT, to ANOVA considering both factors. Our findings indicate that all of these individual tasks, writing $[F(3,156) = 25.21, p < 0.0001]$, lifting small objects $[F(3,156) = 10.54, p < 0.0001]$, placing checkers $[F(3,156) = 13.06, p < 0.0001]$, simulated feeding $[F(3,156) = 16.78, p < 0.0001]$, lifting light objects $[F(3,156) = 14.05, p < 0.0001]$, and lifting heavy objects $[F(3,156) = 13.8, p < 0.0001]$, showed a main effect of contralesional impairment level. Thus, the time to complete each of the individual tasks of the JTHFT with the ipsilesional arm varied with severity level. In terms of a main effect of hand (hemisphere of damage in stroke survivors), only the simulated feeding $[F(1,156) = 13.2, p = 0.0004]$ and writing (copying a sentence) components $[F(1,155) = 147.88, p < 0.0001]$ showed significant main effects. There was also an interaction between contralesional impairment severity and hand $[F(3,156) = 13.12, p < 0.0001]$ on the writing component. Thus, those tasks that tend to be most preferred by the dominant arm showed an effect of hemisphere of damage.

Ideomotor Limb Apraxia

One factor that might contribute to ipsilesional deficits is ideomotor limb apraxia, which is well known to affect skilled, purposeful movements like those assessed by the JTHFT, particularly following a left hemisphere stroke (Mutha, 2010; Berti, Garbarini &
Neppi-Modona, 2015). Twenty stroke participants (12 LHD, 8 RHD) were identified as apraxic by making errors on 4 or more of the 15 items on the standardized ideomotor limb apraxia battery, as recommended by Haaland & Flaherty (1984). To examine the effect of apraxia, we removed participants with apraxia from our ANOVA. Without the inclusion of apraxic participants, we found a main effect of severity ([\(F(3,136) = 20.3, p < 0.0001\]) and hand ([\(F(3,136) = 41.62, p < 0.0001\]), but there was no longer an interaction between the two factors on the total time to complete the JTHFT (Figure 3). This indicates a strong effect of contralesional severity and hemisphere of damage on ipsilesional arm motor performance, regardless of apraxia. It is plausible that the presence of apraxia might modulate the interaction between LHD and RHD with contralesional severity level. However, this interpretation must be taken with caution, because removing 20 participants from our analysis also decreases the statistical power of our ANOVA.
Figure 3: JTHFT total time (in seconds) for control groups and stroke groups with Mild, Moderate, and Severe contralesional paresis, including all participants (gray line) and excluding apraxic participants (black line).

Grip Strength & Grooved Pegboard Test

The two additional measures that we assessed were grip strength and a measure of distal coordination and manipulation (Grooved Pegboard Test). Mean ± SE for grip
strength is shown in Figure 4a, across our 4 severity groups (Control, Mild, Moderate, Severe). Grip strength was assessed using the Lafayette hand dynamometer, as detailed in the methods. Consistent with Figure 4a, our two-way ANOVA revealed a main effect of contralesional impairment level, but no effect of hand, nor interactions between factors \([F(3,156) = 6.02, p < 0.001]\) on grip strength. Thus, regardless of the hemisphere that was lesioned, greater contralesional impairment was associated with a larger grip strength deficit.

For the Grooved Pegboard Test, the ANOVA revealed a main effect of contralesional impairment \([F(3,156) = 7.17, p = 0.0002]\) (Figure 4b), but no effect of hand, nor interactions between these factors. Post-hoc analysis showed participants with severe contralesional hemiparesis \((M = 34.58, SE = 1.89)\) took longer to complete the Grooved Pegboard Test compared to the Control group \((M = 45.07, SE = 1.23)\) and the Mild impairment group \((M = 41.14, SE = 1.41)\), but their performance was not significantly different than the Moderate impairment group \((M = 38.64, SE = 2.55)\). Mean scores have been standardized using the Halstead-Reitan Battery (2004) to account for age, gender, education, and ethnic differences. Therefore, a lower mean score indicates worse performance.
Figure 4a, 4b: Scores have been standardized using the Halstead-Reitan Battery (2004) to account for age, gender, education, and ethnic differences. X-axis represents contralesional impairment severity measured by the Fugl-Meyer Assessment. The Y-axis represents: (4a) grip strength measured using a hand dynamometer (kg); (4b) the T-score representing the total time to complete the Grooved Pegboard Test (Lafayette instrument company). A higher T-score indicates faster performance.
Overall, these patterns demonstrated that all of our measures of ipsilesional arm motor performance varied with the severity of the participants’ contralesional impairment. In addition, our functional performance measure, the JTHFT, which included simulated activities of daily living, varied with arm, reflecting an effect of the hemisphere that was damaged. The presence of apraxia appeared to modulate an interaction between arm (hemisphere of damage) and contralesional severity. These findings suggest that some aspects of performance are affected by ipsilateral MCA damage, regardless of hemisphere, while others depend on the hemisphere that is damaged. However, in all cases, the degree of contralesional impairment influences the extent of ipsilesional motor deficit. This is
important given that the extent of ipsilesional and contralesional impairment have a direct impact on the functional independence of chronic stroke survivors.

Discussion

In the current study, we used three clinical evaluations of unimanual upper extremity performance to assess the ipsilesional arm of 110 stroke participants stratified across the full range of contralesional impairment levels (UEFM Assessment) and grouped by left and right hemisphere damage, as well as 54 age-matched control participants. Our results revealed three critical findings that have substantial implications for clinical rehabilitation. First, all of our groups showed significant ipsilesional arm motor performance deficits, when compared the same arm performance of an age and demographically matched control group. While ipsilesional motor deficits have been reported in stroke survivors as early as 1967 (Wyke), this is the first large-scale study to report functional deficits, identified by standardized clinical assessments of arm function, and stratified by contralesional impairment level and hemisphere of damage. Second, all three of our measures of ipsilesional arm motor performance (grip strength, JTHFT, and Grooved Pegboard Test) varied substantially with the severity of contralesional arm impairment. The greater the impairment of the contralesional arm, the greater the extent of ipsilesional arm motor deficits. This is particularly important because those stroke survivors with severe impairment, who are unable to use the contralesional arm for grasp and manipulation, have the most substantial deficits in movement with the ipsilesional arm. The ipsilesional arm, for these patients, is the only means of carrying out manipulations for activities of daily living.
Third, the extent of impairment in the performance of functional activities, as measured by the JTHFT, depends on the hemisphere that has been lesioned. Left-hemisphere-damaged stroke survivors show greater ipsilesional deficits than right hemisphere damaged stroke survivors. Item analysis of the JTHFT revealed that this hemisphere effect is a function of two subtests: writing and simulated feeding. Previous work from our laboratory with a much smaller sample of stroke survivors (Schaefer, Haaland, & Sainburg, 2009) has shown that the score on the JTHFT can be similar between RHD and LHD stroke survivors, while the fundamental motor control deficits that give rise to these similar scores appear to be different. However, our current findings with a much larger group of stroke survivors reveals that JTHFT scores are significantly different between groups due to differences in component tasks that are most often performed by the dominant arm, writing and feeding. This may be because these subtasks are more extensively dependent on left-hemisphere processes in right-handers.

**Motor Lateralization Predicts the Presence of Ipsilesional Deficits in Stroke**

Early research emphasized the role of the contralateral hemisphere in controlling limb movements (Kuypers, 1982; Sperry, 1961). However, more recent studies have demonstrated that the ipsilateral hemisphere also plays a significant role in motor control. As a result, ipsilesional motor deficits have been reported in both animal models of unilateral brain damage (Gonzalez et al., 2004; Grabowski et al., 1993) and in human stroke studies (Desrosiers et al., 1996; Fisk & Goodale, 1988; Schaefer et al., 2007; Sunderland, 2000; Weinstein & Pohl, 1995; Wyke, 1967; Yarosh, Hoffman & Strick, 2004; Semrau et al.,
Winstein and Pohl (1995) showed that the nature of ipsilesional deficits varied with the side of the lesion. Right hemisphere damage produced slowing of the deceleration phase of rapid aimed movements, while left hemisphere damage produced slowing of the initial acceleration phase of motion (Winstein and Pohl, 1995). This hemisphere dependence of ipsilesional motor deficits was also reported by Haaland and coworkers in a series of studies that employed rapid reciprocal tapping between two targets (Haaland et al., 1977; Haaland & Delaney, 1981; Haaland & Harrington, 1996; 1989a; 1989b;). These studies indicated that stroke survivors with right hemisphere lesions showed deficits in final position accuracy, whereas left hemisphere lesions produced deficits in the initial phase of motion, including substantial slowing of the movements. This line of research led to the hypothesis that left hemisphere motor control processes are specialized for predictive aspects of control that determined the early phase of rapid movements, while right hemisphere processes are specialized for control in the later phase of movement that is responsible for movement corrections and bringing the hand to a stable final position.

We have proposed that ipsilesional deficits can be explained by a model of hybrid control, in which each brain hemisphere contributes unique motor control processes to each arm. Based on studies in neurologically intact individuals, we proposed a model of motor lateralization which attributes specialization of each cerebral hemisphere to distinct but complementary functions: the dominant system for predictive control of limb and task dynamics and the non-dominant system for impedance control mechanisms, that provide robustness in the face of unexpected environmental forces (Yadav & Sainburg, 2014) and which are used to bring the hand to stable positions at the end of a reaching motion (Sainburg, 2002). We termed this hypothesis “dynamic dominance” because of evidence that
dominant-arm control entails more efficient coordination of muscle actions with the complex biomechanical interactions that arise between the moving segments of the limb (Ghez & Sainburg, 1995; Gribble & Ostry, 1999; Sainburg, Ghez & Kalakanis, 1999 Sainburg et al., 1995). We have also demonstrated substantial non-dominant-limb advantages in positional accuracy (Bagesteiro & Sainburg, 2002; Sainburg, 2002; Sainburg & Kalakanis, 2000), load compensation (Bagesteiro & Sainburg 2005; 2003), and a tendency to rely on impedance control when adapting to novel dynamic conditions (Duff & Sainburg, 2007; Schabowsky et al., 2007), and stabilizing objects during bimanual manipulations (Woytowicz et al., 2018). Taken together, these findings suggest that each hemisphere/limb system is differentially tuned to minimize different costs: The dominant system might optimize dynamic parameters, such as energy expenditure and trajectory shape, while the non-dominant system might optimize stability through limb impedance. In fact, we have operationalized this dual-hemisphere control hypothesis in two simulation studies (Yadav & Sainburg 2011; 2014). In those studies, an optimal and an impedance controller were implemented in series. The time at which the controller switched to impedance mechanisms was left open and the simulations were optimally fit to empirical data. The results indicated that non-dominant arm movements switched to impedance control in the early phase of motion, prior to peak velocity, while dominant arm movements did not switch until late in the deceleration phase. These studies provided an operational mechanism by which the two hemispheric controllers might be integrated, and are consistent with the findings that left hemisphere damage produces deficits in the early phase of motion, while right hemisphere damage produced deficits in the later phase of motion.
Sainburg, Haaland and co-workers examined the quality of motor performance following right and left hemisphere damage, in chronic stroke survivors with unilateral middle cerebral artery stroke, largely limited to cortical structures. Schaefer et al. (2007; 2009a; 2009b) examined single joint and multijoint reaching movements, and demonstrated that RHD interferes with the ability to stabilize the final position accurately and to make corrections in response to visual perturbations. In contrast, LHD interferes with the ability to stabilize the trajectory direction and position in the early phase of motion (Schaefer et al., 2009a, 2009b). Mutha et al. (2011b; 2012; 2014), examined the effects of LHD and RHD on visuomotor adaptation paradigms, and found that LHD prevents the ability to adapt the initial direction of movement, but does not affect the ability to correct back to the target later in movement, while RHD interferes with movement corrections, but spares directional adaptation. In a series of focal lesion studies, Mutha et al. (2011; 2014) demonstrated that left posterior parietal lesions produce deficits in direction adaptation, while right dorsolateral frontal lesions produce deficits in movement corrections. Taken together, these studies indicate differential roles of the right and left hemispheres in specifying early movement parameters, such as movement direction, and in making movement corrections and accurately stabilizing hand position at the end of motion. Thus, ipsilesional movement deficits can be understood as the loss of the contribution of the ipsilesional hemisphere to specific aspects of ipsilateral arm control.
**Functional Ipsilesional Deficits**

Interestingly, Schaefer (2009a) examined how functional performance measured by the JTHFT differed between RHD and LHD groups, who also performed reaching movements that were analyzed kinematically. They demonstrated that while RHD showed deficits in final position stabilization, and LHD showed deficits in initial direction accuracy, JTHFT scores were similar between groups. However, in the two groups, the functional deficits measured by JTHFT scores, correlated with different aspects of reaching kinematics, suggesting that the same level of functional performance deficit can be produced by impairment of different motor control mechanisms for left- and right- hemisphere damaged stroke survivors. Poole, Sadek and Halaand (2009) compared performance on a one hand shoe-tying task between with 20 RHD and 28 LHD unilateral stroke participants (using only their ipsilesional hand) and 62 healthy control participants (using either right or left hand, but their was no significant difference between hands). Participants were given verbal instructions and demonstration on how to complete the task. After 10 consecutive timed trials, a score out of 10 was given based on the number of correct trials (knot tied snug) and the total time to complete all 10 trials. Spatial deficits, including visual neglect, organizing complex spatial behaviors, and visuoperceptual deficits, have been shown to be more common in RHD vs. LHD (Benton & Tranel, 1993), whereas sequencing deficits, like those assessed in apraxia, has been shown to be more common following LHD (Harrington & Halaand, 1992). Therefore, to address these possible cognitive influences, Poole and colleagues (2009) measured spatial abilities with the Block Design subtest from the Wechsler Adult Intelligence Scale–Revised, and sequencing deficits with a test of apraxia.
They found left and right hemisphere damage had similar performance outcomes on the shoe-tying task, but accuracy and speed were predicted by different mechanisms in each hemisphere. Shoe-tying accuracy was predicted by both limb praxis and outcomes of a spatial skill test (Block Design) in the LHD group, whereas accuracy was predicted by only outcomes of a spatial skill test RHD group.

A number of other studies that have shown that right and left hemisphere damage produce different underlying motor control deficits that are often manifested by similar performance outcomes on clinical or functional measures (Haaland et al., 2004; Desrosiers et al., 1996; Fisk & Goodale, 1988; Yelnik et al., 1996; Wetter, Poole & Haaland, 2005). In the current study, we found no differences between RHD and LHD groups on the Grooved Pegboard Test and 5 out of 7 items of the JTHFT. It is plausible that the underlying mechanisms responsible for the deficits differ between hemispheres. Because we did not assess kinematics in the current study, it is not possible to dissociate whether the lack of differences between hemisphere groups for some of our measures reflected similar scores due to different underlying motor control deficits, or similar deficits. In fact, as in the current study, grip strength is a measure that has consistently failed to show differences between hemisphere groups in stroke survivors (Desrosiers et al., 1996; Bohannon & Andrews, 1995; Haaland & Delaney, 1981). However, the literature remains equivocal regarding whether movement kinematics of the ipsilesional arm differs between right and left hemisphere damaged stroke survivors. Some studies have reported similarities between groups in movement speed (Schaefer, Haaland & Sainburg, 2007; 2009b; Kim et al., 2003b), movement smoothness (van Dokkum et al., 2018; Noskin, Krakauer & Lazar, 2008), initial direction of wrist movements (Yarosh, Hoffman & Strick, 2004), and a recent robot-based
motor evaluation that included many kinematic parameters of a center-out-reaching task (Semrau et al., 2017). Given that many studies have detailed hemisphere specific motor deficits in the non-paretic, ipsilesional arm of stroke stroke survivors (Haaland et al., 1977; Haaland & Delaney, 1981; Haaland & Harrington, 1996; 1989a; 1989b; Weinstein and Pohl, 1995; Schaefer et al., 2007; 2009a; 2009b; Mutha et al., 2011; 2014), the discrepancies between studies regarding whether motor deficits in the ipsilesional arm are hemisphere-specific, are likely a function of variations in the specific measures reported and variations in distributions of severity levels in the two stroke groups across different studies.

The Role of Apraxia in Ipsilesional Deficits

In the current study, we found that the amplitude of functional deficits, measured by JTHFT, was significantly different between our hemisphere groups (RHD and LHD). Item analysis of the individual components of the JTHFT indicated that only the writing and simulated feeding components varied with the hemisphere of damage. While the other component tasks on the JTHFT are tasks that are often performed with either hand, feeding and writing are tasks that are almost exclusively performed with the dominant hand. It is plausible that the dependence on hemisphere of specific components of the JTHFT might result from apraxia, which is more common in LHD stroke survivors, and which is known to affect performance in the ipsilesional arm. In fact, there is evidence indicating that ipsilesional motor coordination deficits processes are potentiated by ideomotor limb apraxia (Mutha, Sainburg & Haaland, 2010) and predict speed deficits on functional tasks (Poole, Sadek & Haaland, 2009). Goldmann Gross and Grossman (2009) showed praxic disorders
impacted both ipsilesional and contralesional motor function. While there are different types of apraxia, ideomotor limb apraxia was the only type assessed in this study. Most relevant to the current study, Wetter et al. (2005) demonstrated stroke patients with apraxia scored worse on the JTHFT. However, we tested our participants for apraxia using the same methods as Wetter et al. (2005). Our findings indicated that although apraxia potentiated the effects of hemisphere on JTHFT performance, removal of apraxic participants from our analysis did not remove the effect of hemisphere on the writing and feeding components of the JTHFT. Our current findings suggest that apraxia might play a role in the differences in ipsilesional performance between hemisphere groups, but that it does not primarily account for this difference. We conclude that hemisphere specific motor deficits might contribute to similar levels of functional deficits for most component tasks of the JTHFT, but that a greater dependence of feeding and writing on left-hemisphere mechanisms led to greater functional deficits, specifically in these components in the LHD group, compared with the RHD group.

**Contralesional Impairment Severity**

The severity of contralesional impairment reflects the extent to which motor-related circuits have been lesioned in the damaged hemisphere. Because we have previously provided evidence that both hemispheres contribute to control of each arm, we hypothesized that unilateral lesions will affect performance in the ipsilesional arm. Supporting this hypothesis, the current study indicates that the extent of ipsilesional deficits varies with contralesional impairment severity. Our functional measure, the Jebsen-Taylor Hand
Function Test, showed a main effect of contralesional impairment severity such that the more extensive the contralesional arm impairment, the ipsilesional arm shows more extensive performance deficiencies. It should be emphasized that the current study included substantial numbers of participants across levels of contralesional severity (Control, Mild, Moderate, Severe).

Our findings are consistent with a number of previous studies that have shown that ipsilesional motor performance impairments vary with contralesional arm impairment level. In a recent study, Bustrén and colleagues (2017) showed stroke participants with moderate contralesional paresis had more prominent movement deficits than participants with mild contralesional paresis on an ipsilesional drinking task. Therefore, even when stratifying within a small range of impairment, ipsilesional movement deficits varied with contralesional impairment severity. That study, however, did not include participants with severe contralesional deficits, which we found to be the group with the most extensive ipsilesional deficits. However, Noskin et al. (2008) reported contralesional severity measured by maximum grip strength did not vary with ipsilesional motor deficits, measured by the 9-hole peg test (9HPT). It should be noted that the participants with the most severe deficits could not perform grip strength, and therefore were not included in their correlation analysis. However, at one-year post stroke, the study showed a strong correlation between contralesional 9HPT, a test of distal manipulation, and ipsilesional 9HPT, although this analysis was limited to only 10 participants, and the ability to perform the pegboard task with the contralesional arm suggests that the participants were in the mild range of impairment. In our current study, we found that both the JTHFT, and the Grooved Pegboard Test varied substantially with contralesional arm impairment level (UEFM). In support of
the idea that contralesional impairment level is an important factor in the expression of ipsilesional motor deficits, Haaland et al. (2009) demonstrated that stroke participants with paresis, but not those without paresis, showed deficits in performance of a single joint reaching task. We now suggest that our current results indicating a strong dependence of ipsilesional motor deficits on contralesional severity level may help explain the discrepancies in identifying ipsilesional deficits in the literature. Studies that do not stratify contralesional impairment level, or that only assess participants in the mild range of impairment spectrum may not identify deficits in ipsilesional motor performance.

In the current study, our different measures of ipsilesional motor performance showed different dependencies on severity of contralesional impairment and on hemisphere of damage, suggesting that the nature of the test is also an important factor in the expression of ipsilesional motor deficits. Our functional measure (JTHFT), the Grooved Pegboard Test, and grip strength all showed an effect of contralesional impairment severity, but with different patterns. The JTHFT showed differences between the mild impairment group and the healthy control group, whereas grip strength and Grooved Pegboard Test did not. On these measures, performance of participants with mild contralesional impairment were similar to that of the healthy control group. Consistent with this findings, Noskin et al. (2008) found as the ipsilesional 9HPT improved over the course of one year, ipsilesional grip strength did not, emphasizing that these two measures of ipsilesional deficits do not represent the same aspects of performance. Metrot et al. (2013) reported that clinical scores on the 9HPT were comparable to that of healthy controls, yet identified ipsilesional deficits in smoothness during reaching movements when measured with kinematics. Therefore,
comparing between different measures with varying sensitivity to ipsilesional motor deficits can contribute to resolving the discrepancies in literature.

**Interaction Between Hemisphere Specificity and Contralesional Severity**

We have shown the nature of the task contributes to hemispheric ipsilesional deficits, and contralesional impairment severity varies with ipsilesional deficits, along with an interaction between the two factors. Because of this interaction it is critical to maintain these factors separately in analysis. This is particularly concerning when considering the possibility of studies unknowingly comparing mild left hemisphere damage participants with severe right hemisphere damage participants. These groups appeared to have similar performance on our measures, masking the significant differences in hemisphere-specific and severity-specific motor deficits when hemisphere was not factored into analysis. Schaefer (2009a) included RHD subjects with more severe deficits compared to milder deficits in LHD participants, which yielded no differences between groups in performance on the JTHFT. In contrast, on our functional measure, mild LHD participants still took longer than severe RHD participants to complete the JTHFT, but to a much lesser extent. Therefore, when comparing hemisphere differences, it is important to also consider contralesional impairment severity in order to accurately assess deficits.

In summary, this study identified certain aspects of ipsilesional movement deficits that are independent of the hemisphere that is damaged, but rather depend on the extent of contralesional impairment. Other functional measures showed hemispheric differences that are dependent on the type of task. These findings help to provide insight into possible
explanations for the inconsistencies in ipsilesional deficit research. Certain aspects of our functional measure showed hemisphere differences, while other measures did not. In addition, performance may be impaired in both hemispheres, but be due to unique deficits, illustrated by our model of motor lateralization. Studies that don’t show ipsilesional performance varying by lesioned hemisphere, but do show ipsilesional deficits in both groups compared to healthy control participants, can be explained by the measure used to assess function and contralesional impairment severity. Some simple measures, including grip strength, did not show differences between the mild group and the healthy control group. Without our other measures and inclusion of participants with more severe contralesional deficits, we could have misleadingly concluded that there were no ipsilesional deficits. Studies that do not show ipsilesional deficits in either LHD or RHD groups can also be explained by both the nature of the task, and extent of contralesional impairment severity of participants included in the study. Finally, some studies show more severe left hemisphere deficits (Haaland et al., 1994; Sunderland et al., 1999; Pohl et al., 2000; Laufer et al., 2001) while others show more severe right hemisphere deficits (Baskett et al., 1996; Baker et al., 1988; Coslett et al., 1987). We conclude this is due to hemisphere lateralization in which some tasks are more dependent on the right hemisphere, like stabilization, while others, like trajectory, are more dependent on the left hemisphere (Schaefer et al., 2007; 2009a; 2009b).
Clinical Implications

Deficits in coordination of the non-paretic arm can produce substantial limitations in the speed and efficacy of functional activities in participants with severe contralesional deficits. In fact, our findings indicate that unilateral tasks with the non-paretic arm take, on average, twice as long to complete as the comparable arm of age-matched control participants, regardless of hemisphere damaged, indicating labored and inefficient movement that can interfere with a patient’s participation in activities. However, these deficits are much more pronounced following left hemisphere damage. Unfortunately, clinical rehabilitation has yet to recognize the need to address non-paretic arm motor deficits, largely because scientific evidence has not yet been translated into clinical practice, nor has the best practice for this translation been specified through innovative intervention studies. This is particularly important for the left hemisphere damaged patients with severe paresis because they cannot use the paretic arm for manipulations in functional activities and must rely on their non-dominant arm as their primary controller (Rinehart et al., 2009). Vega-González and Granat (2005) found stroke participants used their non-paretic, ipsilesional arm 3 to 6 times more than their affected arm for ADLs. Despite this increased arm use, our participants, who were all in the chronic phase of stroke ($M = 2.88$ years, $SE = 3.14$), still persisted with significant ipsilesional deficits. To provide a reference, imagine using only your non-dominant arm to carry out all your activities of daily living, such as preparing food, dressing including buttoning, putting on socks, and shoes, etc. This would be somewhat frustrating. Now imagine that your non-dominant arm has become more than twice as slow and less coordinated than it was, which is the case for our severely impaired
LHD participants. On the other hand, RHD participants are able to use the previously
dominant arm, but with a 66% decrement in function. Either way, these impairments are
substantially functionally limiting.

Based on these findings, we recommend that stroke patients with severe impairment
in the paretic arm should be referred for evaluation of non-paretic arm performance. If
substantial deficits in performance exist, we advise treatment focused on improving non-
paretic arm performance. Pandian and colleagues (2014) found balance and functional
abilities (measured by the Barthel Index) can be improved with ipsilesional training. In
Pandian’s study, stroke participants who received motor therapy on the ipsilesional side,
along with conventional therapy, had improved outcome measures compared to participants
receiving conventional therapy alone. Though this study did not attempt to specifically
rehabilitate the upper extremity, results indicate that ipsilesional deficits can be improved.
Therefore, future research should determine whether ipsilesional arm training could be
beneficial to participants with moderate to severe contralesional paresis.

Limitations

To avoid selection bias we included all willing stroke participants that met inclusion
criteria during our enrollment period. While this yielded a large cohort of participants, and
the ability to separate LHD and RHD groups by impairment severity within each
hemisphere, the groups were not equal in numbers. However, statistical power analysis
indicated the number of subjects per group was sufficient for analysis. This study did not
include kinematic analyses, which limited our ability to assess the underlying mechanisms
responsible for the ipsilesional deficits. Instead, we focused on functional deficits identified through clinical measures. However, we have previously detailed kinematics of ipsilesional deficits, including those in relation to the JTHFT, our functional measure (Schaefer et al., 2009a).

This study only included chronic stroke participants, but progression of ipsilesional deficits from the acute phase could give insight into the best mechanisms for stroke recovery and optimal rehabilitation timeframe. In fact, Semrau and colleagues (2017) showed that a 1/3 of a population of subacute stroke patients showed substantial deficits in the ipsilesional arm, when measured through a robotic-implemented paradigm, but that these deficits did not vary substantially with contralesional impairment or side of lesion.

We suggest that future research should include therapy interventions that include the ipsilesional arm to determine the best treatment, and phase of stroke that would be most beneficial. Though substantial changes in neural activation have been demonstrated in the acute phase of stroke (Nudo et al., 2006; Ward et al., 2003), some patients with early ipsilesional deficits progress rapidly while others remain significantly impaired (van Dokkum et al., 2018). In the chronic phase, however, there is little functional improvement in the contralesional arm in patients with severe deficits, and it remains unknown whether intense remediation of the ipsilesional arm could lead to significant functional improvements in these patients.
Conclusion

Our results indicate that regardless of contralesional severity level, all participants of our stroke groups showed significant deficits in performance with their ipsilesional arm when compared to age match controls using the same arm. In addition, all of our tests indicated that ipsilesional arm performance deficits increase with contralesional severity. However, only the tasks that normally show substantial disparities between the dominant and nondominant hands on the JTHFT consistently varied with hemisphere of damage. Grip strength and Grooved Pegboard Test performance did not vary with hemisphere of damage. Most importantly, movement function, measured by JTHFT, for patients with severe paresis was 60% to 125% worse than that of age-matched controls. This is particularly important because this group of stroke survivors with severe impairment cannot use the paretic arm for manipulations in functional activities. Based on these findings, we recommend that patients with severe impairment in the paretic arm should be referred for evaluation of non-paretic arm performance. If substantial deficits in performance exist, we recommend treatment focused on improving non-paretic arm performance.
References


