Control of reach extent with the paretic and nonparetic arms after unilateral sensorimotor stroke: kinematic differences based on side of brain damage

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Abstract Scaling of reach kinematics to targets that vary in distance is indicative of the use of planning and feedback-based adjustments. The control of reach extent, however, has not been reported for the paretic arm after stroke. The purpose of this study was to determine whether individuals post-stroke utilized planning (scaling acceleration magnitude) and feedback-based adjustments (scaling acceleration duration) to reach to targets that varied in distance. Individuals with mild-to-moderate motor impairment after stroke and nondisabled adults reached with both arms to targets presented at three distances (8, 16, 24 cm). Kinematic data were used to determine scaling of peak acceleration magnitude and duration to target distance and compared between arms (control, nonparetic, paretic). Despite differences in the magnitude of movement variables, individuals post-stroke utilized both planning and feedback-based adjustments to meet the demands of the task with the nonparetic and paretic arms in a similar manner as controls. However, there was variability in the use of planning with the paretic arm, some individuals utilized planning while others did not. After right brain damage, differences in reach control related to the specialized role this hemisphere plays in endpoint control were found in both arms; no hemisphere-specific changes were found after left brain damage (LBD). The appearance of hemispheric-specific effects after right but not LBD were not due to age, degree of motor impairment, or time post-stroke, but, instead, may be related to relative differences in visual-motor processing ability, lesion characteristics, or interhemispheric inhibition changes between groups.

Keywords Stroke · Upper extremity · Reaching · Motor planning

Introduction After stroke, individuals can be left with residual motor deficits that impact performance of functional activities and overall quality of life (Lai et al. 2002; Mayo et al. 2002). A frequently reported contribution to these functional limitations is an inability to incorporate the paretic arm and hand into daily activities (Mayo et al. 2002; Nichols-Larsen et al. 2005; Lai et al. 2006). Deficits in the ability to execute targeted arm movements after stroke are well documented and include impairments in endpoint accuracy (Lang et al. 2005), movement speed (Lang et al. 2005; Wagner et al. 2007b), joint coordination (Levin 1996; Levin et al. 2002), and patterns of muscle activation (Wagner et al. 2007a). However, there has been limited research on the utilization of anticipatory planning for goal-directed actions with the paretic arm after stroke.

Skilled performance of targeted reach actions includes the use of anticipatory planning prior to movement execution in nondisabled individuals. Evidence of planning suggests the mover is able to anticipate task requirements and choose an appropriate motor command for task completion.
Anticipatory scaling of peak velocity and peak acceleration for the control of reach extent is a well-described characteristic of planned reach actions in nondisabled adults (Gottlieb et al. 1989; Gordon et al. 1994a, c; Pfann et al. 1998; Messier and Kalaska 1999; Sainburg and Schaefer 2004; Stewart et al. 2013). Scaling of peak velocity and peak acceleration to target distance is indicative of how an individual meets the demands of the task, e.g., selecting a higher speed to reach to farther targets. It is not known if individuals post-stroke exhibit this same anticipatory behavior for reach actions with the paretic limb.

Scaling of peak velocity for reaches to targets that vary in distance can be achieved in two ways, scaling of acceleration magnitude or scaling of acceleration duration. Scaling of the magnitude of the initial peak of acceleration is thought to be indicative of anticipatory planning as the peak occurs early after movement onset before the availability of feedback (Brown and Cooke 1981; Gordon and Ghez 1987a; Sainburg and Schaefer 2004). After the time of peak acceleration, feedback is available to assist the process of making adjustments to the reach (Gordon and Ghez 1987b; Mutha and Sainburg 2007). Therefore, scaling of acceleration duration is thought to represent the use of feedback to control the reach behavior. Several studies have found that the scaling of peak velocity and peak acceleration to target distance is preserved for single-joint actions with the ipsilesional arm after stroke (Velicki et al. 2000; Schaefer et al. 2007; Haaland et al. 2009). There is limited-to-no evidence, however, on the scaling of peak velocity and peak acceleration for 3-dimensional reaches with the contralesional, paretic arm (van Vliet and Sheridan 2009).

Scaling of peak acceleration with the ipsilesional arm after stroke differs based on side of brain damage due to the influence of hemispheric specialization for the control of reaching. The left hemisphere is hypothesized to play a specialized role in planning, while the right hemisphere is hypothesized to play a specialized role in achievement of final endpoint position via the use of feedback (Sainburg and Schaefer 2004). Schaefer et al. (2007) found that individuals with left brain damage (LBD) demonstrated a decreased reliance on planning (scaling of peak acceleration magnitude) and an increased reliance on feedback-based adjustments (scaling of acceleration duration) to control elbow extension extent with the ipsilesional arm. Individuals with right brain damage (RBD) showed the opposite effect, an increased reliance on planning and a decreased reliance on adjustments that corresponded to increased endpoint error. It is not known, however, if these differences in control based on side of brain damage extend to unconstrained reaches with the contralesional arm.

The purpose of this study was to determine if individuals with mild-to-moderate motor impairment after stroke utilized anticipatory planning and feedback-based adjustments to reach to targets that varied in distance as evidenced by the scaling of acceleration magnitude and acceleration duration, respectively. We hypothesized that while the magnitude of peak acceleration and acceleration duration would be different from controls, the scaling of these kinematic variables, and therefore the use of planning and feedback-based adjustments, would be preserved in both the ipsilesional and contralesional arms. We also hypothesized that scaling of kinematics with both the ipsilesional and contralesional arms would differ based on the side of brain damage due to the specialized role each hemisphere is thought to play in reach control.

**Methods**

**Participants**

Fourteen individuals with mild-to-moderate motor impairment due to stroke and six age-matched, nondisabled adults participated in this study (Table 1). All participants were right-hand dominant (Oldfield 1971) (determined by pre-stroke handedness). Potential participants were excluded if they presented with current pain that interfered with movement in either arm, surgical intervention in either arm within the last 6 months, previous history of nonstroke neurological diagnoses in participants with stroke and any neurological diagnoses in nondisabled participants, or a score less than 25 on the Mini-Mental State Exam (Folstein et al. 1975). Individuals post-stroke were excluded if they presented with stroke within the last 3 months, an upper extremity Fugl-Meyer (UE FM) motor score (Fugl-Meyer et al. 1975) less than 28, botulinum toxin injection in the UE in the last 3 months, or hemispatial neglect as measured by a score less than 52 on the BIT star cancelation test (Hartman-Maeir and Katz 1995). All participants provided written informed consent prior to participation through a protocol approved by the Health Sciences Institutional Review Board of the University of Southern California.

Participants with stroke presented with a current or previous clinical picture consistent with unilateral anterior circulation involvement (Bamford et al. 1991): six with right hemisphere damage (RBD group) and eight with left hemisphere damage (LBD group). Lesion side and location were confirmed for 12 of the 14 participants either through magnetic resonance imaging or examination of medical records (Table 1). All participants had a stroke lesion in a single hemisphere along the distribution of the middle cerebral artery. Lesions tended to be subcortical with two
participants in each group having a lesion that affected temporal, frontal or parietal cortex.

Experimental task

Targets were presented in an immersive virtual display (Innovative Sport Training, Inc., Chicago, IL) (Fig. 1a). The environment consisted of a simple black background and colored spheres to indicate finger position and target location. A single electromagnetic marker placed on the index finger of the reaching hand acted as the primary interface with the virtual environment (VE). Finger position was represented in the VE as a 2-cm white sphere, or cursor, that moved in real-time as the participant moved the finger. This same electromagnetic marker was used for collection of position data while reaching. Stereoscopic glasses sampled at 60 Hz per eye were worn to allow 3-dimensional (3D) visualization of targets.

The workspace consisted of six targets (3.8 cm red spheres) presented in two directions (+45°, −45°) and three distances (8, 16, 24 cm) (Fig. 1b). At the start of each trial, the participant placed the Cursor onto a Home position represented as a 2.5-cm blue sphere that aligned with a physical start switch. After a variable foreperiod (1.3, 1.6, 1.9 s), the Home position and the Cursor position disappeared and a single target appeared at which time the participant performed an unconstrained, 3D reach movement. The target was visible while reaching, but the arm and the finger Cursor were not visible, thereby eliminating online visual feedback of movement. Visual post-response feedback was provided after each trial showing proximity of final finger position to the target. If the Cursor overlapped with the target (error tolerance of 2.9 cm), the target turned green on feedback, indicating to the participant that they successfully hit the target on that trial. If the Cursor did not overlap with the target, the target remained red during feedback.

Experimental procedure

Participants performed all trials of the experimental task with the ipsilesional arm first followed by the contralesional

Table 1 Participant demographics

<table>
<thead>
<tr>
<th>Subject ID</th>
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<th>ARA (%)</th>
<th>SIS hand (%)</th>
<th>MVPT (%)</th>
<th>Apraxia score</th>
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</table>

UEFM Upper Extremity Fugl-Meyer (max motor score = 66), ARA Action Research Arm Test (max score = 56), SIS Stroke Impact Scale (max score = 100 %), MVPT Motor-Free Visual Perceptual Test age-normed rank score (max score = 100 %), Apraxia score hand and finger gesture imitation assessment with the nonparetic arm (max score = 40), R right, L left, BG basal ganglia, IC internal capsule

* p < 0.05 between stroke groups
For ease of discussion and presentation of results, we will refer to these as the nonparetic and paretic arms, respectively. The order of arm used first in controls was counterbalanced. Participants were instructed to “Reach to the target as fast as possible when ready.” Speed of movement was prioritized over accuracy, and participants were reminded to move quickly throughout data collection.

A single electromagnetic marker was positioned on the nail bed of the index finger, and a wrist and finger splint were applied to provide support and assist with maintenance of a pointing posture. The room was darkened during data collection to block vision of the arm and hand throughout task performance, eliminating visual feedback while reaching. After a short exposure period and 24 practice trials to orient to the VE and task, the participant performed a total of 168 reaching trials (7 blocks of 24 trials). Within each block, targets were presented in a pseudorandom order such that no consecutive trials were to the same target and each target was presented four times. Rest breaks were provided between blocks, and fatigue and pain were monitored throughout. After block 7, extra trials were collected if any errors occurred on individual trials. The first 2 blocks (48 trials) were dropped from data analysis to eliminate any effects of learning related to the VE; the remaining 120 trials (20 trials to each target) were used for analyses.

After a 30–60-min break, the same practice and data collection sequence were completed with the paretic arm (opposite arm in controls). Participants with stroke returned on a separate day for the collection of clinical measures including the UE FM (Fugl-Meyer et al. 1975) to determine degree of motor impairment, the Action Research Arm Test (ARA) (Lyle 1981; Yozbatiran et al. 2008) to quantify motor function, the hand subsection of the Stroke Impact Scale (SIS) (Duncan et al. 1999) to measure health-related quality of life, the Motor-Free Visual Perception Test (MVPT) (Calarusso and Hammill 1972) to measure visual perception, and a gesture imitation assessment of apraxia performed with the nonparetic arm (Goldenberg 1996, 1999).

Dependent measures

The 3D position of the index finger was collected from the electromagnetic marker at a sampling rate of 120 Hz throughout each reach trial and analyzed using a custom script in Matlab (Mathworks, Inc., Natick, MA). Position data were filtered with a low-pass second-order Butterworth with a 10 Hz cut-off and differentiated to determine velocity and acceleration (Winter 2005). Movement onset was determined by searching backward in time from the peak of velocity until velocity dropped below 10 cm/s and either

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**Fig. 1** Experimental set-up. A Side view schematic of participant sitting at virtual display unit. Stereoscopic glasses were worn to allow 3-dimensional view of virtual environment. Virtual objects were sent to the projector and reflected off the mirror into the workspace below the glass. Participants began each trial with the right hand on a physical start switch but ended the reach in free space (above the table). B Top down view of targets. Six targets were presented in 2 directions (+45°, −45°) and 3 distances (8, 16, 24 cm). The start switch (open square) aligned with the sternum.
changed directions or the change in velocity was <1 cm/s for two consecutive samples, whichever was identified first. To eliminate any obvious corrections at the end of the movement, movement offset was determined by searching forward in time from the peak of velocity until velocity dropped below a minimum value (10 cm/s if peak velocity ≤50 cm/s; 20 cm/s if peak velocity >50 cm/s) and either changed directions or the change in velocity was <0.3 cm/s, whichever was identified first. All trials were visually inspected to confirm the accuracy of the movement onset and offset determination and manually corrected as necessary to eliminate any errors related to the automated algorithm.

For each reach trial, several variables were extracted for subsequent analysis of movement scaling. Movement distance was defined as the 3D linear distance between the position at movement onset and the position at movement offset. Peak velocity and peak acceleration were determined by searching forward in time from movement onset to the first peak followed by two consecutive samples that decreased. Time of peak velocity was the time that corresponded to the initial peak of velocity. Since we were interested in the planning of the reach action, we extracted the first peak of velocity and acceleration even if there were later additional peaks that were larger. Movement time was the time between movement onset and movement offset. Endpoint error was the 3D linear distance between the position at movement offset and target position.

Means of kinematic variables to each of the three target distances were determined individually for each participant. Due to the effect of movement direction on the magnitude of kinematic variables (Gordon et al. 1994b; Stewart et al. 2013), data for each direction were analyzed separately. Target direction was converted to indicate either an ipsilateral reach (right arm reaching to +45° targets, left arm reaching to −45° targets) or contralateral reach (right arm reaching to −45°, left arm reaching to +45° targets) for group analyses.

Statistical analysis

To determine the presence of movement scaling, a mixed model analysis of variance (ANOVA) was carried out for each kinematic variable (movement distance, peak velocity, peak acceleration, time to peak velocity) similar to previous work in this area (Sainburg and Schaefer 2004; Schaefer et al. 2007; Haaland et al. 2009). Target directions (ipsilateral/contralateral) were analyzed separately for all comparisons. First, to determine the overall effect of stroke on movement scaling, all participants with stroke were collapsed into a single group irrespective of side of brain damage (between group factor of arm: paretic, nonparetic, control; within group factor of target distance: 8, 16, 24 cm). To determine the presence of planning for each participant, a linear regression analysis between peak acceleration and target distance was completed separately for each individual. A positive, significant correlation between peak acceleration and target distance indicated that planning was used by that individual participant. A total of 60 trials were included in the regression for each target direction; therefore, an r value ≥0.255 equated to a significant relationship.

Next, to determine if movement scaling differed based on side of brain damage, a mixed model ANOVA was performed on the right and left arms separately. For each arm, all three groups (Control, RBD, LBD) were included in the analysis (e.g., right arm of controls, right nonparetic arm of the RBD group, right paretic arm of the LBD group). The analysis of changes in reach control due to side of brain damage focused on expected kinematic differences based on previous literature. Specifically, reach planning was investigated in the LBD group while acceleration duration scaling and endpoint error were investigated in the RBD group. Due to the known differences in reach kinematics between the dominant and nondominant arms in controls (Sainburg and Schaefer 2004; Schaefer et al. 2007), paretic arms (right dominant in LBD group, left nondominant in RBD group) were not directly compared between stroke groups.

The least significant difference was used for post hoc comparison of significant between arm or group differences. Significant interactions were followed with three arm by target distance ANOVAs (e.g., paretic vs. nonparetic, paretic vs. control, nonparetic vs. control). If an interaction between arm and target distance was still present, this analysis was followed with a repeated measure ANOVA to determine whether each group scaled the kinematic variable to target distance and a t test at each target distance to determine differences between groups. Significance level was set at p < 0.05 for all statistical tests. In instances where within group variance is high as is common after stroke, power to find group differences can be limited. Therefore, Cohen’s d, a measure of effect size, was calculated for comparisons that were different but did not reach the statistical threshold; d was calculated based on the mean across target distances for each direction. Effect size was considered small if d = 0.2, medium if d = 0.5, and large if d = 0.8 (Portney and Watkins 2009). SPSS 16.0 (SPSS, Inc., Chicago, IL) statistical software was used for all analyses.

Results

Reach performance

Figure 2 shows individual trial reach hand paths to each target for a control participant reaching with the right arm
(Fig. 2a), a participant with LBD reaching with the paretic, right arm (Fig. 2b), and a participant with RBD reaching with the paretic, left arm (Fig. 2c). All participants scaled movement distance to target distance in both directions; reaches to 24-cm targets were longer than reaches to 8 and 16-cm targets. Both participants with stroke tended to undershoot the target more frequently, especially when reaching to the farthest targets. However, as a group, participants with stroke scaled movement distance to target distance (Fig. 3a) in a similar manner as controls (main effect of distance: $p < 0.001$; main effect of group: $p > 0.05$). Participants with stroke did show larger endpoint error (Fig. 3b) and longer movement times (Fig. 3c) for reaches with the paretic arm. Endpoint error and movement time

Fig. 2 Reach hand paths for an individual control participant reaching with the right arm (a), a stroke participant with LBD (S18) reaching with the paretic, right arm (b), and a stroke participant with RBD (S12) reaching with the paretic, left arm (c) to the 8, 16, and 24 cm targets. Each line represents the hand path for a single reach trial; open circle represents target location. Mean velocity and acceleration profiles are shown for reaches to ipsilateral targets for the same control participant (d) and stroke participants (e, f). Each line represents an ensemble average of all trials to a specific target distance (8, 16, 24 cm)
for reaches with the nonparetic arm did not differ from the control group in either direction ($p > 0.05$).

Scaling of peak velocity and peak acceleration to target distance

Ensemble average velocity trajectories for individual participants reaching to ipsilateral targets are shown in Fig. 2. The control participant (Fig. 2d) scaled peak velocity to target distance such that reaches to the 8 cm targets had a lower average peak velocity than reaches to the 24 cm targets. For the participants with stroke (Fig. 2e, f), the magnitude of peak velocity tended to be lower and movement time longer compared with the control participant; however, scaling of peak velocity was still evident. Group peak velocity across distances is shown for each arm in Fig. 4a. Scaling of peak velocity to target distance was present in the control, nonparetic, and paretic arms for reaches in both directions (main effect of distance: $p < 0.001$).

The control participant in Fig. 2d scaled peak acceleration to target distance although this scaling was less distinct than peak velocity. Additionally, the duration of the acceleration phase scaled to target distance (i.e., time of 0 crossing from acceleration to deceleration). Despite the lower overall magnitude of the initial peak of acceleration,
the participants with stroke demonstrated some scaling of acceleration magnitude to target distance (Fig. 2e, f). Scaling of acceleration duration was also present (more evident in S12) as were additional peaks of acceleration (S18). Group data for peak acceleration is shown in Fig. 4b. Scaling of acceleration magnitude was present on average in the control, nonparetic, and paretic arms in both directions (main effect of distance: \( p < 0.01 \)). Participants also varied acceleration duration (time to peak velocity) to targets of different distances irrespective of group (Fig. 4c; main effect of distance: \( p < 0.001 \)). Therefore, on average, participants with stroke used a combined control pattern that included scaling of acceleration magnitude (anticipatory planning) in addition to acceleration duration (feedback-based control) to reach to targets of increasing distance, a similar pattern to that of control participants.

To determine the presence of planning for each individual participant, we calculated the correlation between peak acceleration and target distance. While on average all groups showed utilization of planning to control reach extent (mean \( r \) fell above the dotted line in Fig. 5), there was a fair amount of variability between participants, especially in the stroke group. A few control participants (2 in each direction) and participants with stroke reaching the nonparetic arm (3 in each direction) did not show evidence of planning to control reach distance (\( r \) value below the dotted line in Fig. 5). However, in the stroke group, approximately half the participants showed evidence of planning, while half did not. The correlation coefficient between peak acceleration and target distance did not correlate with scores on any clinical measures including UE FM, ARA, SIS hand domain, and the MVPT.

For both target directions, the magnitude of peak velocity and peak acceleration were lower for the paretic arm compared with the control group and the nonparetic arm (Fig. 4). There was no significant difference in the magnitude of peak velocity or peak acceleration between the nonparetic and control arms for reaches in either direction (main effect of arm: \( p > 0.05 \) for all comparisons).

**Effect of side of brain damage**

Participants with stroke were separated into two groups based on side of lesion (RBD, LBD) to determine the effect of side of brain damage on reach behavior. Data for reaches to both ipsilateral and contralateral targets are reported; however, only reaches to ipsilateral targets are shown in Fig. 6. Scaling of movement distance did not differ from controls for reaches with the nonparetic or paretic arms in either the LBD or RBD groups (\( p > 0.05 \) for all comparisons); therefore, any differences in kinematics were not due to differences in movement distance.

To determine whether left hemisphere damage led to deficits in planning reach extent, the scaling of peak velocity and peak acceleration magnitude was examined in the LBD group. Both the nonparetic and paretic arms demonstrated scaling of peak velocity and peak acceleration to target distance (main effect of distance: \( p < 0.005 \) for all comparisons) (Fig. 6a, b). Additionally, in the correlation analysis, the number of participants in the LBD group who did not show utilization of planning to control reach distance (\( r \) value below the dotted line in Fig. 5) did not differ from the control group or the RBD group. For reaches with the nonparetic arm, only one participant in the LBD group did not utilize planning when reaching to ipsilateral targets. For reaches with the paretic arm, 44% of comparisons (8 participants \( \times \) 2 directions) in the LBD group did not show utilization of planning while 58% of comparisons (6 participants \( \times \) 2 directions) in the RBD group did not show evidence of planning. Therefore, there were no changes in reach behavior related to hemispheric specialization in the nonparetic or paretic arms after LBD.

To determine whether right hemisphere damage led to deficits in endpoint control, scaling of acceleration duration and endpoint error were examined in the RBD group. Scaling of acceleration duration was present in both arms in the RBD group (main effect of distance: \( p < 0.001 \)) and did not differ from controls (\( p > 0.05 \) for all comparisons). There was no increase in endpoint error for reaches with the right nonparetic arm (Fig. 6c). However, individuals slowed their movements down compared with the right arm of controls as shown by lower peak velocity (Fig. 6a; ipsilateral: \( p = 0.057, d = 1.12 \); contralateral: \( p = 0.084 \),
d = 1.15), lower peak acceleration (Fig. 6b; ipsilateral: p = 0.104, d = 0.91; contralateral: p = 0.423), and longer movement times (Fig. 6d; ipsilateral: p = 0.363; contralateral: p = 0.166, d = 1.00). Endpoint error was significantly increased when reaching with the left, paretic arm, however (p < 0.03 both directions). Therefore, there were changes in both the nonparetic and paretic arms after RBD consistent with the effects of hemispheric specialization for the control of reaching.

Differences between the RBD and LBD for the control of reaching could not be explained by age, time post-stroke, UE FM motor score, ARA score, or apraxia score (Table 1). The groups did differ on two clinical measures, the SIS Hand domain and MVPt scores. The LBD group ranked the ability to use their paretic hand in functional activities significantly lower than the RBD group on the SIS (p = 0.045). Visual perception was also different between groups; the age-normalized MVPt score was lower in the RBD group compared with LBD group (p = 0.081; d = 1.04).

Discussion

This is the first study to demonstrate preserved scaling of initial peak acceleration to targets that vary in distance for 3D, unconstrained reaches with the paretic arm in individuals with mild-to-moderate motor impairment after stroke. On average, individuals chose a higher speed for reaches to farther targets and a lower speed for reaches to closer targets consistent with previous studies with the nonparetic arm that involved constrained, isolated elbow movements (Schaefer et al. 2007; Haaland et al. 2009). Such scaling of initial peak acceleration magnitude is thought to be indicative of anticipatory planning as the peak occurs early after movement onset before the availability of feedback (Brown and Cooke 1981; Gordon and Ghez 1987a; Sainburg and Schaefer 2004). Scaling of acceleration duration (time to peak velocity) was also present after stroke suggesting feedback-based control was also used to capture the target. Since vision of the arm was not provided during movement, this feedback consisted of internally derived proprioceptive information (Gordon and Ghez 1987b). Therefore, despite differences in the magnitude of some kinematic variables, individuals demonstrated utilization of both anticipatory planning and feedback-based adjustments to meet the demands of the task with the paretic arm in a similar manner as controls.

While on average the stroke group used planning to control reach extent with the paretic arm, the use of planning varied between individuals. It is not entirely clear why some individuals used planning to control reach extent
while others did not. The use of planning did not correlate with any of the clinical measures of motor impairment, motor function, or visual perception taken in the current study, and this group of participants did not present with apraxia. Variability between individuals in the use of planning to control reach distance may have been related to factors not measured in the current study including variability in force generation capability, history of paretic arm use, and lesion location and volume. Future work on the use of planning to control 3D reaches to targets that vary in distance should consider inclusion of additional metrics to explore these possible mechanisms.

Deficits in the control of reaching related to hemispheric specialization were not present in the LBD group. Previous work has shown deficits in the scaling of peak acceleration, and therefore, a decreased reliance on planning to control reach extent, in the ipsilesional arm after LBD (Schaefer et al. 2007). However, the presence of these deficits varies with degree of paretic arm motor impairment (Haaland et al. 2009). In Haaland et al. (2009), hemisphere-specific deficits were present in individuals with more severe motor impairment but not in individuals with mild-to-no motor impairment. The current study did not find any changes in the scaling of initial peak acceleration to target distance for reaches with the nonparetic or paretic arms in the LBD group. The cohort of participants in this study presented with relatively mild motor impairment (mean UE FM motor score >51). Therefore, the lack of deficit in the nonparetic arm found here is consistent with the “nonparetic” group in Haaland et al. (2009). A recent study found a similar gradient in hemisphere-specific deficits for reaches with the paretic arm using a paradigm that varied target direction (Mani et al. 2013). In that study, individuals with moderate motor impairment after LBD showed planning-related deficits, while individuals with mild motor impairment did not. The LBD group in our study had UE FM motor scores that were more closely related to the moderately impaired group in Mani et al. (2013). It is not clear why we found no deficit in planning with the paretic arm while Mani et al. (2013) did, however, differences in task paradigm (varying reach target distance vs. varying target direction), planning requirement (planning reach distance vs. planning intersegmental coordination), reach condition (3D unconstrained vs. two-dimensional planar), and lesion location or volume may explain these disparate findings.

Deficits in the control of reaching related to hemispheric specialization were present in the RBD group. Right hemisphere stroke has been shown to lead to deficits in endpoint accuracy in both the nonparetic and paretic arms during planar reaching (Schaefer et al. 2007; Mani et al. 2013). While there was no increase in error for reaches with the nonparetic arm after RBD in the current study, participants appeared to adopt a compensatory strategy of slowing down movement speed to achieve a higher level of accuracy consistent with the well-known speed-accuracy trade-off (Schmidt and Lee 2005). While Schaefer et al. (2007) found increased error for reaches with the nonparetic arm after RBD, the magnitude of peak velocity was controlled in that study. In the current study, participants were encouraged to move quickly but no feedback was given regarding movement speed and peak velocity was not controlled. Therefore, after RBD, participants appeared to have traded movement speed for endpoint accuracy. This pattern of decreasing movement speed to improve accuracy was insufficient for reaches with the paretic arm. Endpoint error with the paretic, left arm was significantly greater than for the matched arm in controls. Persistent difficulty with endpoint accuracy in the paretic arm after RBD even in individuals with mild impairment is consistent with a recent study that varied target direction (Mani et al. 2013).

It is not entirely clear why changes in reach control related to hemispheric specialization were seen after RBD but not LBD in this group of individuals with mild-to-moderate motor impairment. The two groups did not significantly differ in motor impairment, age, or time post-stroke. It is possible that differences in lesion location could explain the finding of hemisphere-specific effects in the RBD group but not the LBD group. Recent work has found that planning-related deficits for the control and learning of a motor task after LBD are related to lesions in the left parietal lobe but not lesions in the left frontal cortex (Mutha et al. 2011a, b). In contrast, deficits in the ability to make feedback-based corrections to ongoing movement after RBD are found after lesion to the right frontal cortex but not the right parietal cortex (Mutha et al. 2011a). Therefore, the presence of hemisphere-specific effects after RBD but not LBD in the current study could be explained by lesion location if most participants had lesions that impacted frontal cortical regions. While we cannot fully determine the effect of lesion on reach behavior, the location of lesions in participants with available data do not suggest lesion location alone explains the current findings (i.e., lesions were not predominantly in frontal cortex, see Table 1). This does not, however, rule out the possibility that lesion location and lesion volume differed between groups which led to the differences in the presentation of hemisphere-specific deficits. Lesion location and lesion volume were not matched or controlled in our study and warrant inclusion in future studies that investigate the effect of side of brain on reach control with the paretic arm.

Visual perception, as measured by the MVPT, was more impaired in the RBD group than the LBD group consistent with previous work reporting greater visual perceptual changes after right hemisphere stroke (York and Cermak 1995). The experimental task in the current study was visually driven. Therefore, behavioral differences in reach
control after RBD in the current study may have been due to visual processing changes. It is also possible that lesions in the right and left hemispheres had different effects on visual-motor processing that impacted task performance. The MVPT is a screening tool that measures several domains of visual perception, and therefore, it is not possible to specify what component of perception was altered. Future studies should include additional measures that target specific visual-motor processes with more precision.

It is also possible that the behavioral differences after RBD but not LBD found in the current study were due to changes in interhemispheric inhibition. After stroke, the nonlesioned hemisphere shows an increase in inhibition onto the lesioned hemisphere during movement (Murase et al. 2004; Duque et al. 2005; Grefkes et al. 2008; Carter et al. 2010). This change in interhemispheric inhibition after stroke, however, may differ based on side of brain damage in right-hand dominant individuals. During UE movement in nondisabled adults, the hemisphere contralateral to the moving hand has an inhibitory influence on the ipsilateral hemisphere (Herbert et al. 1992; Gerloff et al. 1998; Di Lazzaro et al. 1999; Daskalakis et al. 2002). The relative change in inhibition, however, differs based on the hand being moved in right-hand dominant adults (Ziemann and Hallett 2001; Duque et al. 2007). Movement of the right hand leads to a relatively greater increase in interhemispheric inhibition (left hemisphere to right hemisphere) compared with movement of the left hand (right hemisphere to left hemisphere). As such, changes in inhibition may not be symmetrical after stroke. Since there is greater interhemispheric inhibition from the left to the right hemisphere in the healthy brain, the increase in inhibition from the nonlesioned (left) to the lesioned hemisphere (right) after RBD may be relatively greater than after LBD. There is some evidence that side of brain damage has an effect on changes in interhemispheric inhibition after stroke (Lewis and Perreault 2007); however, further research that combines brain measures of inhibition with behavioral measures of reach control is needed.

The results of this study can only be applied to individuals with mild-to-moderate motor impairment in the chronic phase of stroke recovery. During the acute/subacute stage of stroke or in individuals with more severe motor impairment, the influence of hemispheric specialization on reach performance may not be evident. The sample size in the current study was small which may have limited statistical power in finding differences based on side of brain damage. While significant main effects were not found in the overall ANOVA analyses on the effects of side of brain damage, the medium to large effect sizes seen suggest the current results would persist with a larger sample size.

Differences in the control of reaching based on side of brain damage may have clinical implications that warrant further investigation. Side of paresis has been shown to affect recovery of UE function. Stroke leading to hemiparesis of the nondominant arm has been reported to relate to increased magnitude of UE impairment (Harris and Eng 2006), decreased responsiveness to a bilateral UE intervention program (McCombe Waller and Whitall 2005), and differences in the use of both the paretic and nonparetic arms during the performance of functional tasks (Rinehart et al. 2009) compared with hemiparesis of the dominant arm. However, it is not known what if any role hemispheric specialization plays in these differences between nondominant and dominant arms. The results of the current study suggest that future research should include kinematic-based assessments that target expected differences based on side of brain damage, especially in individuals with mild-to-moderate motor impairment, and explore the clinical implications of changes in reach control due to hemispheric specialization.

In conclusion, individuals with mild-to-moderate motor impairment after stroke utilized both planning and feedback-based adjustments to meet the demands of the reaching task with the nonparetic and paretic arms in a similar manner as controls. Overall, individuals scaled acceleration magnitude and acceleration duration to control reach extent, although there was some variability between individuals on the use of planning when reaching with the paretic arm. Differences based on side of brain damage were found and were not related to clinical measures of motor impairment and function. Therefore, changes in reach control based on side of brain damage may reflect underlying neural differences in response to stroke injury that warrant future investigation.

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References


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