DOI: 10.1093/brain/awh133 *Brain* (2004), **127,** 1145–1158

Hemispheric asymmetries for kinematic and positional aspects of reaching

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Summary

Kinematic analyses of reaching have suggested that the left hemisphere is dominant for controlling the open loop component of the movement, which is more dependent on motor programmes; and the right hemisphere is dominant for controlling the closed loop component, which is more dependent on sensory feedback. This open and closed loop hypothesis of hemispheric asymmetry would also predict that advance planning should be dependent on the left hemisphere, and on-line response modification, which defines closed loop processes, should be dependent on the right hemisphere. Using kinematic analyses of reaching in patients with left or right hemisphere damage (LHD or RHD), we examined the ability: (i) to plan reaching movements in advance by examining changes in reaction time (RT) when response amplitude and visual feedback were cued prior to the response; and (ii) to modify the response during implementation when target location changed at the RT. Performance was compared between the stroke groups, using the ipsilesional arm, and age-matched control groups using their right (RNC) or left (LNC) arm. Aiming movements to a target that moved once or twice, with the second step occurring at the RT, were performed with or without visual feedback of hand position. There were no deficits in advance planning in either stroke group, as evidenced by comparable group changes in RT with changes in amplitude and visual feedback. Response modification deficits were seen for the LHD group in secondary velocity only. In addition, LHD produced slower initial peak velocity with prolongation of the deceleration phase and faster secondary peak velocities, and the RHD group produced deficits in final error only. These differences are more consistent with the dynamic dominance hypothesis, which links left hemisphere specialization to movement trajectory control and right hemisphere specialization to position control, rather than to global deficits in open and closed loop processing.

Keywords: dominance; cognition disorders; motor skills; cerebral cortex; cerebral infarction

Abbreviations: LHD = left hemisphere damage group; LNC = left normal control (control group performing with the left hand); MT = movement time; LPAR = left parietal group; RHD = right hemisphere damage group; RNC = right normal control (control group performing with the right hand); RPAR = right parietal group; RT = reaction time

Received April 23, 2003. Revised October 13, 2003. Second revision December 29, 2003. Accepted December 31, 2003. Advance Access publication March 19, 2004

Introduction

A large number of studies examining brain-damaged patients have shown that motor deficits in the limb ipsilesional to unilateral hemispheric damage are more common after left than right hemisphere damage (Haaland and Harrington, 1996). Limb apraxia is the best clinical example of this asymmetry (Geschwind, 1965; Haaland *et al*., 2000). Neuroimaging experiments (Kim *et al*., 1993; Schluter *et al*., 2001) and a study using transcranial magnetic stimulation (Schluter *et al*., 1998) have also shown that the left hemisphere is specialized for controlling a variety of

motor skills. Some studies have also identified a role for the right hemisphere (Haaland and Flaherty, 1984; Iacoboni *et al*., 1999; Roy *et al*., 2000), and the relative importance of each hemisphere is likely to vary depending upon the requirements of the movement being examined (Harrington and Haaland, 1991, 1997; Harrington *et al*., 2000; Haaland *et al*., 2004), which was exemplified recently by a reach and grasp study that showed greater implementation deficits after left hemisphere damage (LHD) when greater planning was required (Hermsdorfer *et al*., 1999*a*).

For goal-directed reaching, one appealing hypothesis is that the left hemisphere is more specialized for ballistic movements that are more dependent on planning and motor programme development and less dependent on direct sensory feedback (Haaland and Harrington, 1989*a*, 1994; Winstein and Pohl, 1995). This hypothesis links the right hemisphere to the control of non-ballistic movements that are more dependent on sensory feedback and less dependent on motor programmes (Haaland and Harrington, 1989*b*; Winstein and Pohl, 1995; Hermsdorfer *et al*., 1999*a*, *b*). However, this distinction has not been universally supported (Hermsdorfer *et al*., 1996; Harrington and Haaland, 1997), and it is quite common for predicted effects to be demonstrated in either the LHD or right hemisphere damage (RHD) group, but not both, suggesting the need for further study.

Historically, the separation of open and closed loop movements is based upon a two-component model of aiming, which originated with Woodworth (Woodworth, 1899; Keele, 1986; Elliott *et al*., 2001). Using kinematic analyses, the initial component can be separated into an acceleration and deceleration phase, which brings the hand to the vicinity of the target and was associated with open loop processing. The secondary component allows the hand to hit the target and is more dependent on sensory feedback and closed loop processing. This distinction is not as clear as once thought. Some have suggested that even the acceleration phase of the movement is influenced by changes in sensory input that are used to modify or update the internal model or motor programme (for a review see Desmurget and Grafton, 2000), independent of visual or proprioceptive feedback of arm position (Goodale *et al*., 1986; Bard *et al*., 1999). Other work using behavioural or electromyographic data in normals and in proprioceptively deafferented patients has shown that only the deceleration phase (Forget and Lamarre, 1987; Heath *et al*., 1998; Shapiro *et al*., 2002) or only the interval following peak acceleration (Gordon and Ghez, 1987) are dependent on sensory feedback and closed loop control. Taken together, these findings suggest that the separation of different components of the reaching movement on the basis of open and closed loop processing is relative rather than absolute and, while the acceleration phase of the initial component appears to be more reflective of open loop processing, and the deceleration phase and secondary movement are more reflective of closed loop and feedback processing, these distinctions are controversial especially for slower movements. Therefore, reaction time (RT) may be a better way of assessing the impact of motor plans on reaching.

RT has the advantage that it is independent of simultaneous closed loop processing. Advance planning in addition to other processes, such as attention, are reflected by the RT, which precedes movement initiation (Sternberg *et al*., 1978). Systematic variations in RT partially reflect the effects of anticipating the response rather than response initiation alone. Studies of motor sequencing have shown that RT increases as the number of different movements in a sequence increase (Harrington and Haaland, 1987), reflecting advance planning of the sequence, which is associated with open loop processing. This same paradigm has been used to show that LHD does not produce deficits in advance planning for such sequences (Harrington and Haaland, 1991).

RT findings in arm reaching studies are variable, sometimes increasing after LHD only (Haaland and Harrington, 1989*a*) or after RHD only (Hermsdorfer *et al*., 1999*b*). However, these increases have not been associated with task characteristics that increase advance planning in normal individuals, such as movement amplitude, which if cued prior to the response affects advance planning because, when response amplitude is shorter, there is less time and less need for ongoing planning or response modification during the movement (Rosenbaum *et al*., 1987).

Another way of assessing closed loop processing besides kinematic analysis is to perturb the location of the target unpredictably after the response has been initiated. Some studies using small target perturbations without visual feedback of arm position have emphasized rapid modification of the response that has been attributed to feedforward processing through internal feedback loops (Goodale *et al*., 1986) and appears to be dependent on the contralateral parietal cortex (Desmurget *et al*., 1999). A recent review argues that feedforward processing is similar to feedback or closed loop processing due to its dependence on feedback loops (Desmurget and Grafton, 2000). However, another study in which visual feedback was available during the movements showed that early parts of the movement (peak velocity) were associated with the speed and accuracy demands of the first target, while later parts of the movement (i.e. deceleration and secondary phase) were associated with the demands of the second target (Heath *et al*., 1998). This pattern of findings suggested that changes in target location did not produce response modification until after the acceleration phase. Therefore, we predicted that if the right hemisphere was dominant for response modification, the RHD group would show deficits during response implementation, which could be seen in all components based upon the Desmurget and Grafton conceptualization but only in the later phases of the movement based upon the Heath conceptualization.

The goal of the current study was to determine if LHD produced deficits in open loop processing and RHD produced deficits in closed loop processing by examining the relationship between kinematic measures and measures of advance

Table 1 *Demographic and descriptive variables**

Variable	LNC (n = 17)	LHD (n = 23)	RNC (n = 14)	RHD (n = 15)
Age (years)	60.7 (10.3)	59.0 (10.2)	64.6 (8.1)	66.4 (12.8)
Gender (% female)	29%	35%	57%	20%
Education (years)	15.8 (3.1)	14.6 (2.5)	14.5 (2.9)	15.0 (2.8)
Years post-stroke		7.2 (4.8)		6.3 (4.8)
Lesion volume (cm^3)		48.0 (40.8)		34.1 (29.3)
Hemiplegia (n, %)*,+		3, 13%		1, 7%
Visual neglect (n, %)*,+		0, 0%		1, 7%
Visual field cut (n, %)*,+		1, 4%		3, 20%
Limb apraxia	13.8 (1.4)	12.9 (2.8)	14.1 (1.0)	14.1 (0.8)
Speech†	20.0 (0.0)	14.5 (7.4)§	20.0 (0.0)	19.8 (0.8)
Comprehension†	79.5 (1.9)	65.1 (21.1)§	79.7 (1.1)	78.5 (3.9)
Repetition†	97.2 (3.5)	69.4 (37.1)§	98.6 (1.2)	96.2 (4.4)
Grip right‡	34.9 (12.7)	32.4 (19.2)	42.3 (13.5)	43.9 (15.3)
Grip left‡	39.7 (10.2)	46.6 (13.6)	41.2 (10.3)	40.8 (19.6)
Tap right‡	41.7 (5.9)	26.5 (14.2)§	46.7 (7.9)	43.3 (8.3)
Tap left‡	41.4 (4.8)	38.1 (10.9)	42.2 (11.4)	32.0 (14.3)¶

*Tabled values are means with SDs in parentheses, except for gender, hemiplegia, visual neglect and visual field cut. In these cases, the number of cases and percentage of patients are specified. Apraxia is designated as mean number correct out of 15 items (Haaland and Flaherty, 1984; Haaland *et al*., 2000). Groups did not differ in age, education, years post-stroke or lesion volume using t tests. Stroke groups did not vary in incidence of hemiplegia, visual neglect or visual field cuts using χ^2. +Hemiplegia defined by contralateral grip strength = 0; visual field cuts based on confrontation; visual neglect defined by a higher percentage of errors on line cancellation relative to the worst control subject in LNC or RNC group. †These scores are from the Western Aphasia Battery (Kertesz, 1982) and assess spontaneous speech, auditory comprehension and repetition, with maximum scores of 20, 80 and 100, respectively. ‡Grip strength and finger tapping are expressed as a standardized t score. ¶t tests comparing RNC and RHD revealed significant group differences for left finger tapping [$t(27) = 2.12$, $P = 0.04$]. §t tests comparing LNC and LHD revealed significant group differences in right finger tapping [$t(38) = 4.14$, $P < 0.001$], speech [$t(38) = 3.1$, $P < 0.01$], comprehension [$t(38) = 2.8$, $P < 0.01$] and repetition [$t(38) = 3.1$, $P < 0.01$].

planning and response modification. We compared the ipsilesional performance of stroke patients with RHD or LHD with control groups using their right or left hand [right normal control (RNC) or left normal control (LNC)] as they performed movements that varied in their response modification requirements by unpredictably moving the target once (80, 120, 200 or 300 mm) or twice (from 80 to 120 mm or from 200 to 300 mm) at the RT. We assessed advance planning by measuring changes in the RT as a function of response amplitude and visual feedback, which were cued prior to the response. If the open loop hypothesis of left hemisphere functioning is correct for reaching, LHD should be associated with decreased advance planning, and kinematic changes, if present, should be present in the acceleration phase only. If the closed loop hypothesis is correct, RHD should be associated with impaired response modification, as evidenced by greater implementation deficits in perturbed (two-step) than unperturbed (one-step) movements, especially in the deceleration or the secondary component of the aiming movement, or both.

Methods

Participants

Thirty-eight right-handed stroke patients and 31 right-handed healthy control subjects were examined after obtaining approval from the Human Research and Review Committee of The University of New Mexico School of Medicine and informed consent from each participant, according to the Declaration of Helsinki. Age and education were similar across groups (see Table 1). Twenty-three of the stroke patients had LHD and 15 had RHD, at least 3 months before participation in this study. All stroke patients performed with the hand ipsilesional to the damaged hemisphere. Normal control participants were randomly assigned to perform the task with their right or left hand. Seventeen performed with their left hand (LNC) and 14 performed with their right hand (RNC). Participants were excluded for history of substance abuse, psychiatric diagnosis, neurological disorders other than stroke, or peripheral problems that restricted movement. No significant differences were found between the RHD and LHD groups for the number of years post-stroke or lesion volume.

Table 1 summarizes the characteristics of the four groups. Language skills (Kertesz, 1982) using the Western Aphasia Battery, were impaired in the group with LHD. While index finger tapping speed (Reitan and Davison, 1974) was slower in the contralesional finger in both stroke groups, grip strength (assessed with a hand dynamometer) was not impaired in either hand for either stroke group. The incidence of hemiplegia, visual field cuts, visual neglect (cancellation test) and limb apraxia using a validated apraxia battery (Haaland and Flaherty, 1984; Haaland *et al*., 2000) did not differ between the two stroke groups.

Lesion reconstruction

MRIs were done in the stroke patients on a Siemens or a Picker 1.5 T scanner. CT scans were done in two patients who could not undergo an MRI for medical reasons. Slice thickness for MRIs and CT scans was 5 mm, and all scans were performed at least 3 months after stroke. Infarcts were traced by a neurologist (R.K.) onto axial templates derived from the DeArmond Atlas (DeArmond *et al*.,

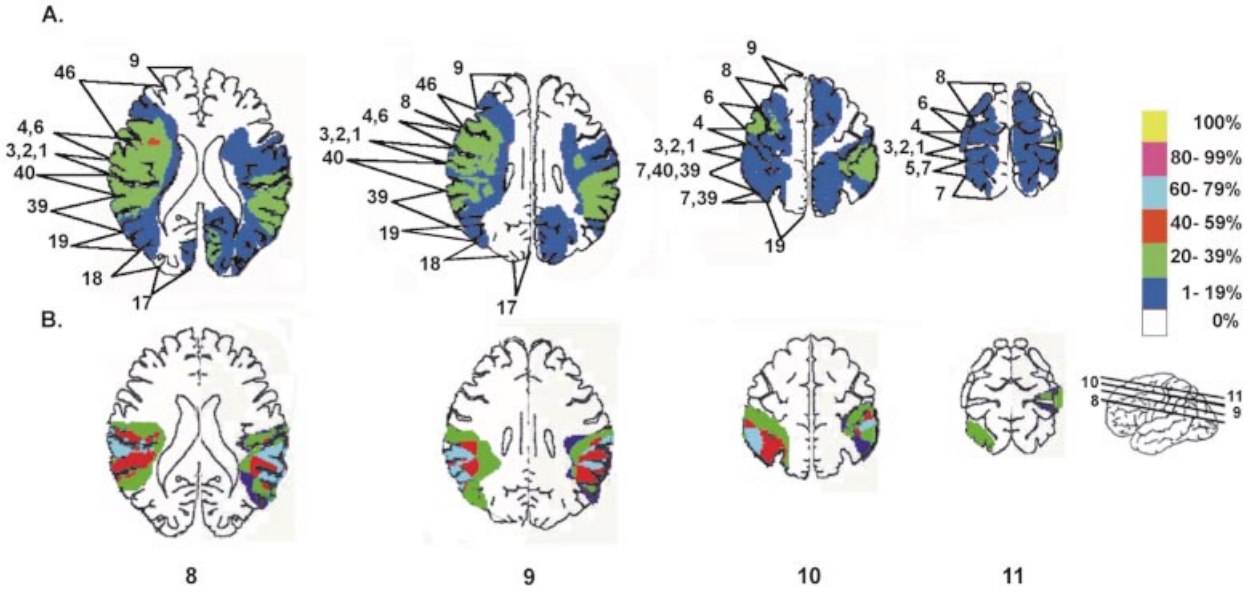

Fig. 1 Lesion locations based upon tracing lesions from MRI or CT scans and superimposing lesions on axial slices from the DeArmond Atlas (DeArmond *et al*., 1989) separately for the LHD patients (displayed on the left) and the RHD patients (displayed on the right). (**A**) The overlaps for the entire LHD and RHD groups. (**B**) The overlaps for a subset of patients with left parietal (LPAR) or right parietal (RPAR) damage. The key for the degree of overlap is on the right.

1989), and then retraced into a computer program (Frey *et al*., 1987), which allowed us to overlap the areas of damage (Fig. 1) and to calculate lesion volume. The lesions of the entire RHD and LHD groups are displayed in Fig. 1A, and all statistical analyses were done in this group.

Apparatus

A 21 inch monitor was 813 mm away from the participant's eyes, and their chin rested on a chin rest. They grasped a vertical dowel that was attached to a stylus on a digitizing tablet. The stylus was mounted in a low friction, ball bearing-mounted handle that glided along a single axis. A cursor appeared on the monitor representing the position of the participant's arm, and this cursor had a one-to-one relationship to the movement of the participant's hand in time and distance. The spatial and temporal accuracy of the digitizing tablet was 0.1 mm and 0.1 ms, respectively. The digitizing table was sampled at a rate of 110 coordinates per second. The spatial resolution of the monitor was 0.56 mm.

Procedures

At the beginning of a trial, a start circle appeared on the monitor at the subject's midline. The participant was instructed to move the cursor into the start circle to begin a trial. When the participant entered the start circle, a 50 ms tone sounded. After a variable delay of 500–1000 ms, the start circle was removed and a target circle appeared. The target circle and the start circle were 5 mm in diameter in all trials. The subject grasped a vertical handle with their forearm parallel to the digitizing tablet. When the target appeared, they were asked to move from midline laterally as quickly and accurately as possible to hit the target. All movements were made in the same hemispace as the performing hand. The ipsilesional hand and hemispace were always used in the stroke groups (e.g. right arm and right hemispace in the right stroke group).

See Table 2 for study design. Movement amplitude and target perturbations (one-step, two-step) were randomized, but amplitude and visual feedback conditions were cued prior to response. The target circle either appeared at a variable distance and stopped (one-step), or appeared at a specific distance and jumped to a position 50% further than the original distance (two-step) at the RT. Two visual feedback conditions were used. The target was always present, and visual feedback of arm position was either removed at response onset or always present. For each of the two visual feedback conditions, six different trials were used (specified by target perturbation and distance, respectively): one-step 80, 120, 200 and 300 mm; and two-step 80 to 120 mm and 200 to 300 mm, for a total of 12 different conditions. Participants were given one block of practice trials in which one trial of each of the conditions was presented with, then without, visual feedback of arm position. The experimental trials were presented in five blocks. The feedback and no feedback trials were blocked, such that within each block of 36 trials, all the trials for one feedback condition were presented before the trials for the other feedback condition. The order of the feedback conditions was counterbalanced across subjects so that half of the participants received the feedback condition first and half received the no feedback condition first, in all five blocks. Target amplitude and step were presented randomly within each block. Three trials of each step and feedback condition were presented within each block, for a total of 36 trials in a block, 180 trials across all blocks and 15 trials for each condition. The inter-trial interval was 2 s.

A trial ended when (i) the participant reached the target circle and remained in the circle for 500 ms; (ii) 3000 ms had elapsed from the start of the trial; or (iii) the participant remained still for 1000 ms. Any trials that had an RT <150 ms or >750 ms were aborted and repeated later in the same block. In these cases, participants were given feedback about their response ('too fast' or 'too slow', respectively). Trials were excluded as false starts if the initial movement time (MT) was <100 ms or if the peak velocity of the secondary component was larger than the peak velocity of the initial

Table 2 *Study design*

Feedback				No feedback			
One-step condition							
(1) 80 mm	(2) *120 mm	(3) 200 mm	(4) *300 mm	(5) 80 mm	(6) *120 mm	(7) 200 mm	(8) *300 mm
Two-step condition							
	(9) *120 mm		(10) *300 mm		(11) *120 mm		(12) *300 mm

Conditions that were analysed in ANOVAs; all conditions had 15 trials; final amplitudes are displayed for the two-step condition (80 to 120 mm and 200 to 300 mm); target amplitude and perturbation were randomized and visual feedback was blocked.

component. The number of trials eliminated was small and did not differ among groups ($P > 0.10$). A mean of one trial was excluded per block of 15 trials averaging across all four groups, and all conditions with a range of 0.56 to 1.14 trials excluded. No more than 1.6 trials were excluded from any single condition.

Measures

For each participant, means were computed for each of the 12 conditions for each of the kinematic measures. In most cases, measures of speed (peak velocity for the initial component, secondary peak velocity), MT and error (absolute error and variable error for the deceleration phase and secondary component) or distance moved (acceleration phase only) were taken. See Fig. 2 for position and velocity profiles in a normal control subject and specification of most of the dependent measures. See Figs 3 and 4 for position and velocity profiles for a patient with LHD and a patient with RHD. The velocity profiles were derived from the position profiles by calculating the slope in a running average across five position coordinates. The acceleration component began at response onset and ended at peak velocity. The deceleration component began at peak velocity and ended when (i) velocity dropped to 20 mm/s or less; (ii) velocity did not change more than 1.5 mm over a 100 ms interval at the beginning of the interval; or (iii) velocity decreased to at least 50% of peak velocity and then increased, indicative of a second acceleration phase and a new movement. The secondary movement began at the end of the initial movement and ended when velocity was 20 mm/s or less. Dependent measures from the acceleration, deceleration and secondary components of the movements are listed in Table 3. Absolute error is the distance from the centre of the target at the end of a particular phase, and variable error is the standard deviation of absolute error. Percent deceleration is the percentage of the initial MT that is in the deceleration phase, and percentage secondary is the percentage of trials that include a secondary component. All times are in ms, distances and errors are in mm, and velocities are in mm/s.

Normal performance is first described using separate analyses of variance (ANOVAs) for each dependent measure with amplitude, visual feedback and target perturbation as factors. The 80 and 200 mm one-step conditions were not analysed. ANOVAs were calculated comparing each stroke group and its control group (RHD and RNC, LHD and LNC) with emphasis on any group differences or interactions with group.

Group differences in heterogeneity of variance are always a concern when studying brain-damaged patients, and these differences could affect the pattern of our findings. Variability was not consistently greater in one particular group, but appeared to vary in an unpredictable manner across different dependent measures. Nevertheless, this factor could potentially affect the pattern of our results, a common limitation of studies with brain-damaged patients.

Results

Normal aiming

The results of the statistical analyses are summarized in Table 3, which also provides the means and SEMs for each of the measures as a function of target perturbation, amplitude and visual feedback for the pooled normal control group. Amplitude effects were seen for RT in that RT was greater for the 120 mm movement, which probably reflects the greater planning necessary when movement duration is less because there is less time for ongoing planning during movement execution (Harrington and Haaland, 1987; Rosenbaum *et al*., 1987). MT, distance, error, error variability, velocity and proportion of trials with secondary components were greater for the 300 mm than the 120 mm movement. However, there was no change in the shape of the velocity profile as amplitude increased, indicating that the duration of the acceleration and deceleration components increased proportionately. This pattern of findings is consistent with previous work suggesting that the same base velocity profile underlies simple aiming movements across various amplitudes, thus simplifying planning requirements (MacKenzie *et al*., 1987). It also suggests that in the normal control group, there was no significant modification of the motor programme as amplitude changed during the initial component after the response was initiated. However, the increase in trials with secondary movements as amplitude increased indicates that modification occurred in that component.

The initial components were faster and either covered more distance or were more accurate when visual feedback of arm position was available, especially when the movement was large (feedback × amplitude). The proportion of trials with a secondary component increased and the secondary component was more accurate, but not faster, with visual feedback. These findings are consistent with the conclusion that all components of the movement were influenced by visual feedback (Elliott *et al*., 1995; Elliott and Carson, 2000). However, because visual feedback availability was cued prior to the response, the independent effect of open or closed loop processing cannot be specified. Similar to the effect of amplitude, the shape of the velocity profile did not change as a function of visual feedback. This finding is

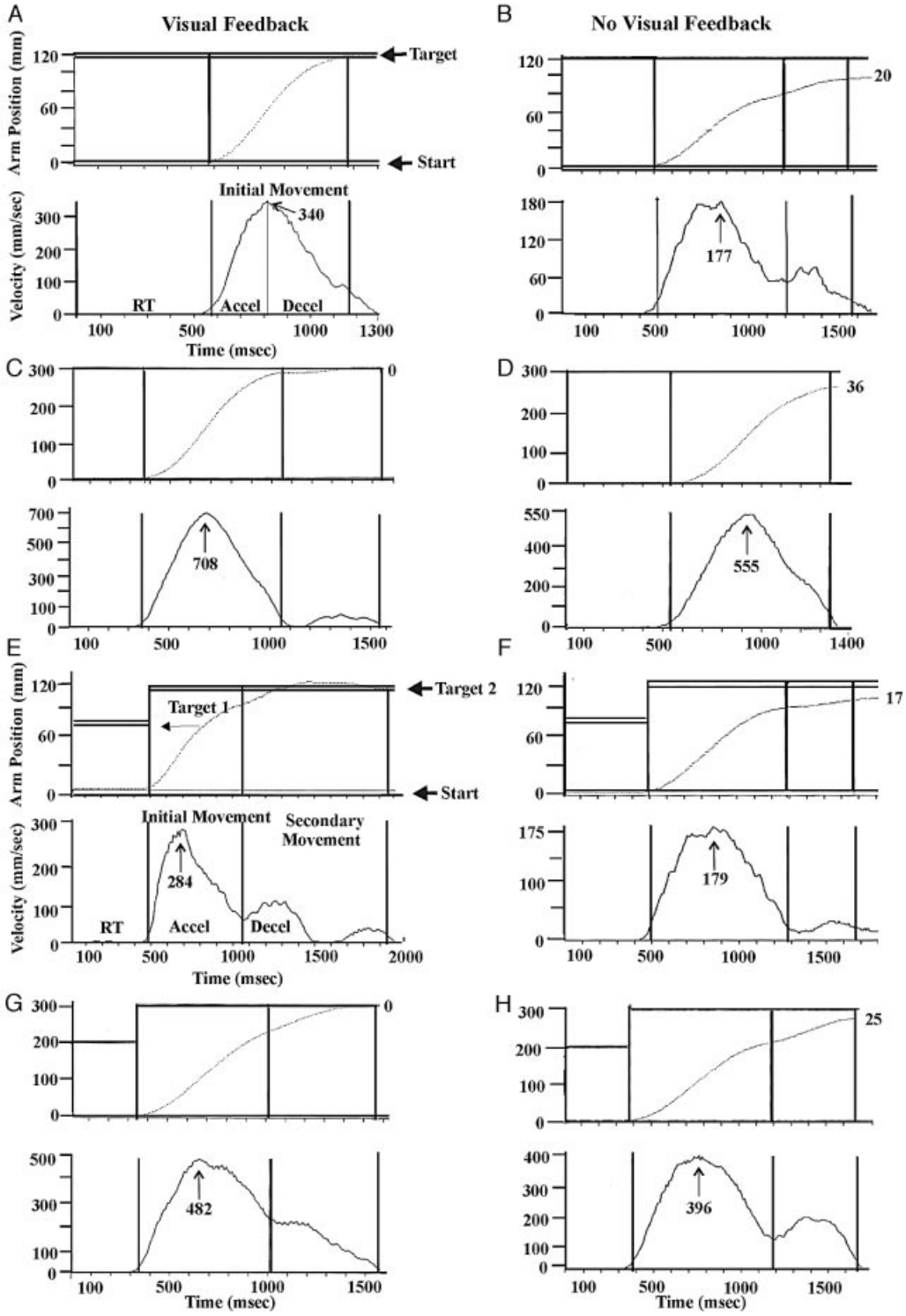

Fig. 2 Position and velocity profiles are displayed for a normal control subject, with time displayed on the *x*-axis and either distance or velocity on the *y*-axis. Trials with and without visual feedback of hand position are displayed on the left and right, respectively. All conditions are illustrated: one-step 120 mm (**A** and **B**); one-step 300 mm (**C** and **D**); two-step 120 mm (**E** and **F**); and two-step 300 mm (**G** and **H**). Examples of most of the dependent measures can be seen in **A** and **E**. Peak velocity of the initial component is designated in all velocity figures with an arrow, and final error is designated to the far left in all position figures.

inconsistent with studies which have shown that deceleration time increases when visual feedback is available due to the increased time necessary to compare limb and target position visually and make online corrections (Marteniuk *et al*., 1987; Carson *et al*., 1993). However, in contrast to these previous studies, we separately measured the deceleration phase and

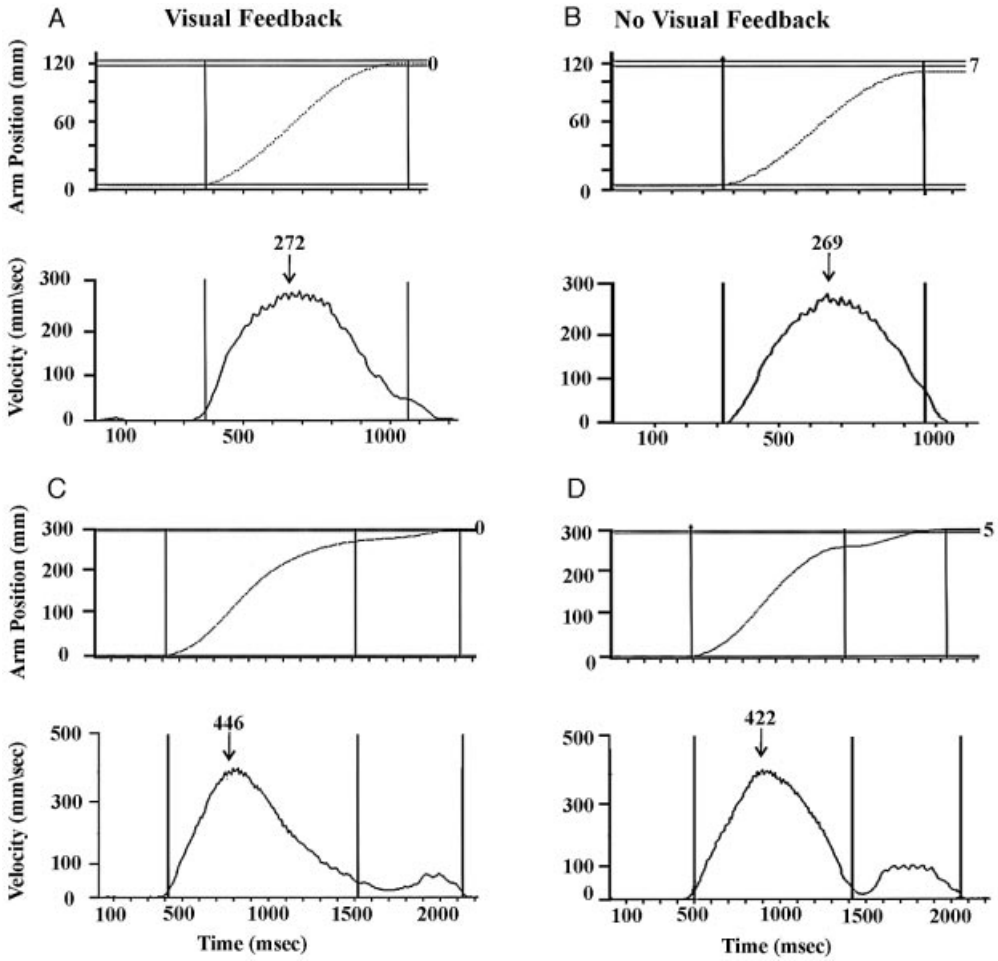

Fig. 3 Position and velocity profiles for the stationary conditions are displayed for an LHD patient: (**A** and **B**) 120 mm condition with and without visual feedback, and (**C** and **D**) 300 mm condition with and without visual feedback. See Fig. 2 for other details.

Table 3 *Normal control performance as a function of step, feedback and amplitude*

	Measures	Feedback				No feedback			
		One-step		Two-step		One-step		Two-step	
		120 mm	300 mm	120 mm	300 mm	120 mm	300 mm	120 mm	300 mm
	RT*,+,†,‡	424 (10)	415 (10)	449 (10)	417 (9)	452 (10)	431 (11)	469 (9)	433 (9)
	% Deceleration*	44 (1)	46 (1)	43 (2)	42 (1)	47 (1)	46 (1)	43 (1)	44 (1)
Acceleration	MT+,‡	275 (12)	365 (12)	275 (20)	395 (21)	289 (13)	362 (12)	259 (18)	396 (22)
	Distance*,+,†,¶,§	61 (1)	133 (3)	49 (2)	122 (3)	59 (2)	128 (4)	45 (2)	112 (4)
	Velocity*,+,†,‡,§,o	338 (17)	583 (27)	268 (13)	485 (22)	315 (14)	562 (28)	266 (13)	446 (20)
Deceleration	MT*,+,‡	347 (17)	450 (19)	380 (22)	537 (19)	344 (20)	444 (23)	350 (16)	522 (24)
	Error*,+,†,¶,§,o	8 (0.5)	27 (3)	19 (2)	30 (3)	21 (2)	47 (5)	31 (2)	63 (6)
	Variable error*,+,†,§,o	9 (0.5)	20 (2)	19 (1)	23 (3)	15 (1)	28 (2)	19 (1)	35 (3)
Secondary	MT*,+,‡,¶	507 (24)	651 (29)	616 (25)	629 (25)	447 (43)	538 (37)	644 (38)	674 (37)
	Error+,†,§	0.1 (0.04)	0.1 (0.03)	0.1 (0.02)	0.1 (0.03)	20 (2)	34 (4)	18 (1)	32 (4)
	Variable error+,†,§	0.2 (0.1)	0.3 (0.1)	0.2 (0.1)	0.3 (0.1)	14 (1)	21 (2)	12 (1)	22 (2)
	Velocity*,+,‡,¶,o	66 (3)	107 (5)	100 (8)	115 (8)	64 (3)	93 (5)	102 (6)	129 (10)
	% Secondary*,+,†,‡,¶,o	81 (3)	94 (1)	83 (2)	95 (1)	62 (4)	81 (3)	82 (3)	86 (3)

Means with SEMs in parentheses. *main effect of step; all *P*s <0.01; +main effect of amplitude; all *P*s <0.01; †main effect of feedback; all *P*s <0.03; ‡step × amplitude interaction; all *P*s <0.01; ¶step × feedback interaction; all *P*s <0.02; §feedback × amplitude interaction; all *P*s <0.05; ostep × amplitude × feedback interaction; all *P*s <0.04.

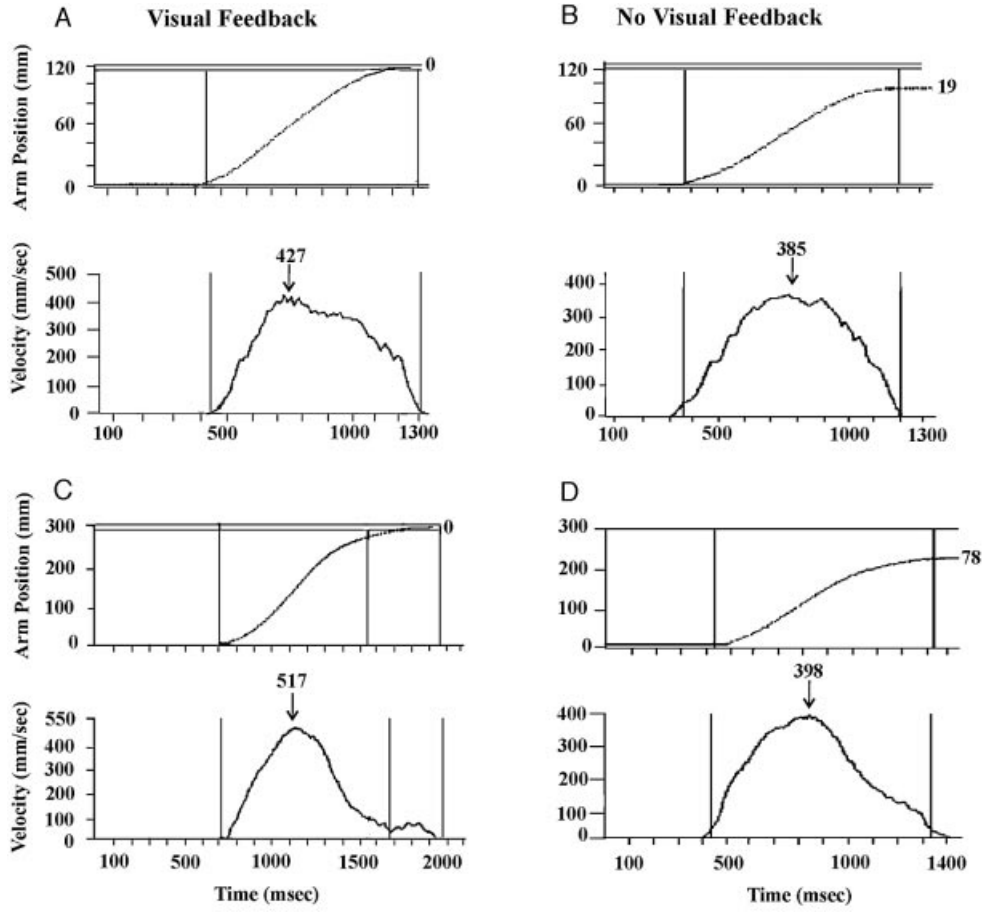

Fig. 4 Position and velocity profiles for the stationary conditions are displayed for an RHD patient: (**A** and **B**) 120 mm condition with and without visual feedback and (**C** and **D**) 300 mm condition with and without visual feedback. See Fig. 2 for other details.

the secondary movement phase, and because secondary movements were more prevalent with visual feedback, it is likely that the combined duration of these components would have increased with visual feedback.

During the initial component, target perturbation or step trials produced changes in all phases of the movement, consistent with response modification very early in the movement. In the initial component, perturbation produced longer MTs, decreased velocity, decreased distance or increased error and prolongation of the deceleration phase, consistent with less efficient performance. In addition, the percentage of trails with a secondary component was greater for the two-step trials, and the relative efficiency of the secondary component increased as evidenced by faster secondary movements for the two-step trials with comparable final error for both conditions. The relative increase in efficiency in the secondary component may reflect compensation for a suboptimal movement plan and/or the difficulty of rapidly modifying the response in the initial components of the movement. This finding of differential performance between the initial component and later components of the movement is consistent with other studies that have examined target perturbation and found high negative correlations between the acceleration and deceleration components (Elliott and Carson, 2000).

These step effects interacted with amplitude in some cases. Despite the fact that the target perturbation occurred at the RT, RT was greater for the two-step condition (step effect), especially when response amplitude was short (step $\times$ amplitude) (two-step – one-step = +22 for 120 mm and +1 for 300 mm). This longer RT for the two-step condition at 120 mm was attributed to greater predictability in the one-step condition because whenever the 120 or 300 mm targets were presented first, the subject knew the target would not be perturbed (recall that the two-step movements began with targets at 80 or 200 mm, which were perturbed to 120 or 300 mm, respectively). This subtle cue may have decreased the RT somewhat in the one-step relative to the two-step condition, especially for the 120 mm movement probably because there was less time to process and react to the unexpected perturbation during the shorter duration 120 mm movement than the longer duration 300 mm movement (Rosenbaum *et al*., 1987). However, it is also possible that the longer RT for the two-step 120 mm movement was related to the fact that the first amplitude displayed for the two-step trials was shorter (80 or 200 mm) than the target displayed for

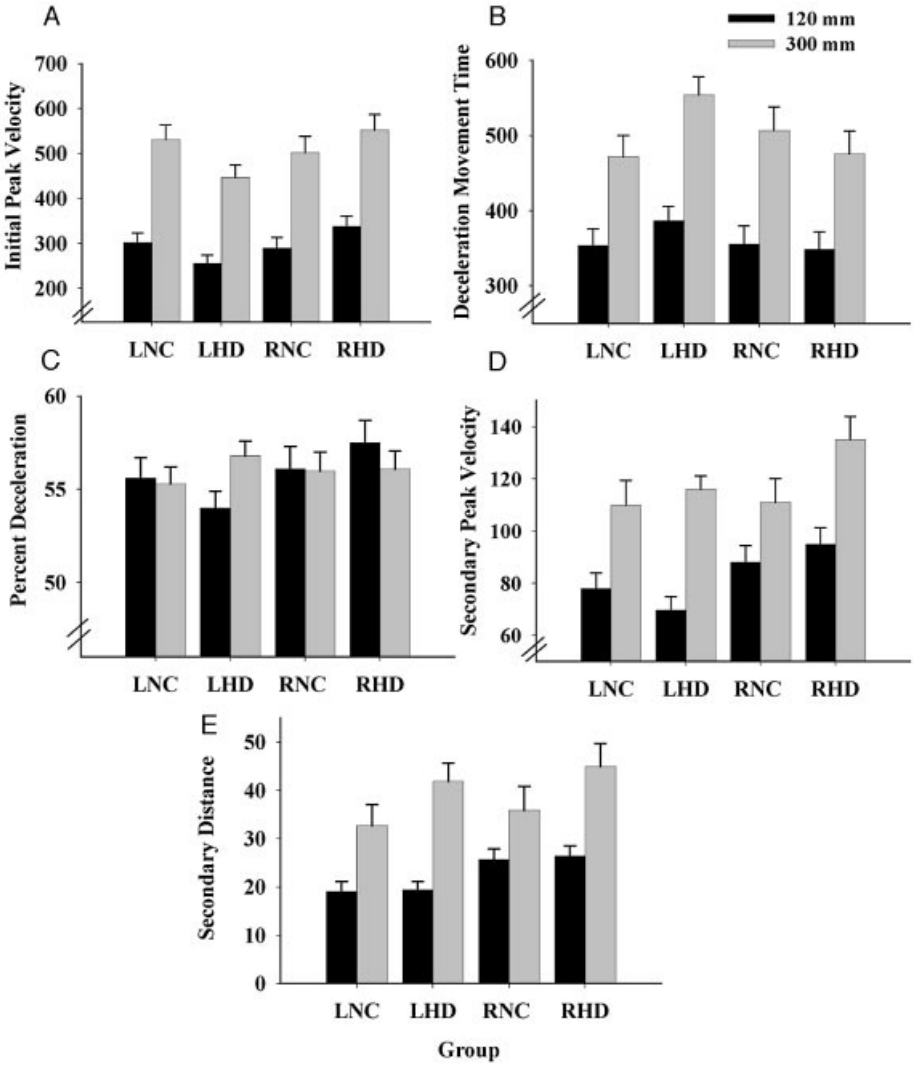

Fig. 5 The figure depicts dependent measures, which show significant group × amplitude interactions for the LHD group. In all cases, the means were derived by pooling across conditions that did not influence the findings (perturbation and visual feedback). Mean with SEM bars for all four groups for (**A**) initial peak velocity, (**B**) deceleration movement time, (**C**) percent deceleration, (**D**) secondary peak velocity, and (**E**) secondary movement distance.

the one-step trials (120 or 300 mm). Shorter amplitudes typically produce longer RTs, and the 80 mm amplitude is likely to be more influenced by this effect than the 120 mm amplitude. Step × amplitude interactions were also present for acceleration MT, initial peak velocity, deceleration and secondary MT, secondary peak velocity, and percentage of secondary movements, suggesting that target perturbation affects normal performance in all phases of the movement. These findings would lead us to predict that if RHD produces deficits in closed loop control, deficits should be seen in all components of the movement.

Deficits associated with LHD

The pattern of findings based upon comparisons between the LHD and LNC groups shows that the LHD group demonstrated greater evidence of temporal than spatial deficits. There were no group differences or group interactions with amplitude or feedback for absolute error or its variability during any of the three movement phases. Significant group differences were seen for RT and secondary MT, reflecting longer RTs [$F(1,38) = 5.19$, $P < 0.05$; mean (SEM) for LNC = 417 (11) and for LHD = 451 (10)] and longer secondary MTs [$F(1,38) = 9.04$, $P < 0.01$; mean (SEM) for LNC = 565 (27) and for LHD = 671 (23)] for the LHD group. These group differences were not affected by amplitude, visual feedback or target perturbation.

Group × visual feedback interactions were seen for initial peak velocity [$F(1.38) = 5.15$, $P < 0.03$], reflecting lower velocities for the LHD group especially when visual feedback was present [mean (SEM) for LNC = 439 (24) for feedback condition, 395 (23) for no feedback condition; and for LHD = 360 (20) for feedback condition, 344 (20) for no feedback condition].

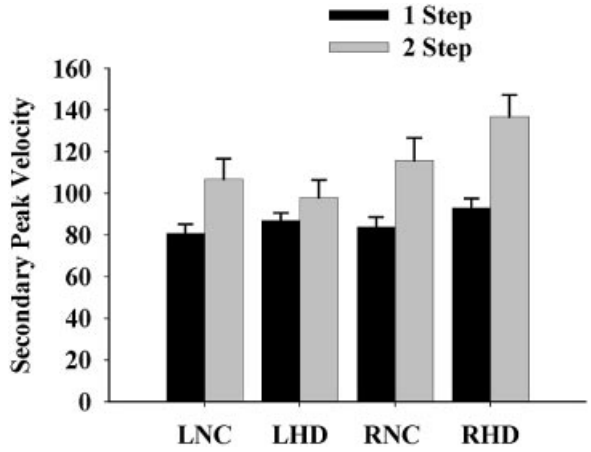

Fig. 6 Secondary peak velocity (mean with SEM bars) for all four groups for one-step and two-step trials to demonstrate the significant group × perturbation interaction for the LHD and LNC comparisons. In all cases, the means were derived by pooling across the conditions that did not influence the findings (amplitude and visual feedback).

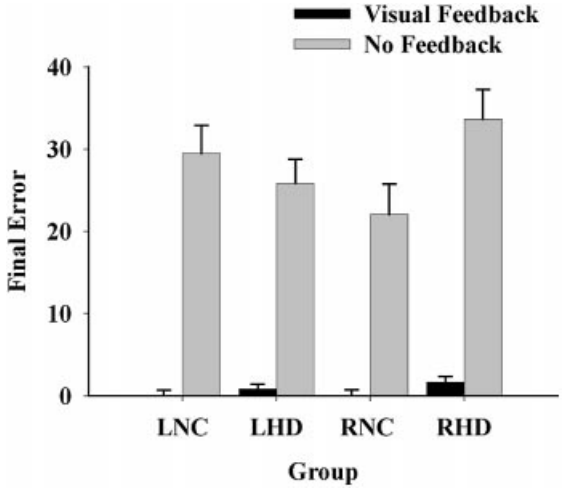

Fig. 7 Final absolute error (mean with SEM bars) for all four groups with and without visual feedback to demonstrate the significant group × visual feedback interaction for the RHD and RNC comparisons and the significant visual feedback effect for the LHD and LNC comparisons. In all cases, the means were derived by pooling across the conditions that did not influence the findings (step and amplitude).

As can be seen in Fig. 5, group × amplitude interactions showed that relative to the LNC group, the LHD group demonstrated a smaller increase in initial peak velocity [$F(1,38) = 4.09$, $P = 0.05$] and a greater increase in deceleration MT [$F(1,38) = 7.98$, $P < 0.01$], deceleration proportion [$F(1,38) = 4.34$, $P < 0.05$], and secondary velocity [$F(1,38) = 5.75$, $P < 0.025$] as amplitude increased. This greater increase in secondary velocity was associated with an increase in the distance moved as amplitude increased during the secondary movement [$F(1,38) = 5.52$, $P < 0.025$] and normal error. Therefore, especially for the 300 mm movement, the LHD group's response efficiency was lower in the acceleration and deceleration phases, but more efficient in the secondary phase. These effects can also be seen qualitatively in Fig. 3, which shows the velocity profiles of an LHD patient at the two movement amplitudes.

In most cases, target perturbation or step influenced the LHD group in the same way that it influenced the LNC group. However, as can be seen in Fig. 6, there was a significant group × step interaction for secondary velocity, reflecting less of an increase in velocity from the one-step to the two-step conditions in the LHD group than the LNC group [$F(1,38) = 4.18$, $P < 0.05$]. This finding suggests that the LHD group's response modification resulted in a slower secondary component with normal accuracy. This effect could not be attributed to the possibility of differential advance planning for the shorter perturbed targets (step × amplitude), which was demonstrated in the control group, because there was not a significant group × step × amplitude interaction.

Deficits associated with RHD

In contrast to the LHD group, the RHD group demonstrated no deficits in RT or any of the initial component measures detailed in the Methods section, and their deficits in the secondary component were spatial rather than temporal. The RHD group demonstrated a significant main effect of group and a group × feedback interaction for final absolute error only. As shown in Fig. 7, final error was greater for the RHD group only when visual feedback was not available [$F(1,27) = 5.39$, $P < 0.05$].

There were no other significant group effects or interactions with group for the RHD and RNC comparisons. The lack of significant group differences or interactions with group in the initial component or in any temporal measures is illustrated in Figs 4, 5 and 7, which, in contrast to the LHD group, show the RHD group's normal performance in initial and secondary peak velocity, deceleration MT, deceleration proportion and secondary movement distance.

Intrahemispheric lesion location

Although the RHD and LHD groups did not differ in lesion volume, intrahemispheric lesion location was somewhat different, with a higher incidence of medial parietal, occipital and lateral parietal damage in the RHD group. In order to ensure that the differences between the RHD and LHD groups were not due to intrahemispheric differences in the parietal lobe, we identified a subset of patients with right ($n = 6$) or left ($n = 5$) lateral parietal damage (RPAR and LPAR). Figure 1B shows that these two groups have similar parietal involvement. Similar to the overall analyses, the RPAR group demonstrated deficits in final error only. Specifically, they showed greater increases in final error for longer movements performed without visual feedback [group × amplitude × feedback; $F(1,18) = 4.4$, $P = 0.05$; no feedback – feedback = +18.1 for 120 mm RNC, +26.1 for 300 mm RNC, +18.9 for 120 mm RPAR, +44.9 for 300 mm RPAR]. Group × amplitude [$F(1,18) = 4.9$, $P < 0.05$] and

group × feedback [$F(1,18) = 5.1$, $P < 0.05$] interactions were also significant. Final error was also affected by target perturbation, such that target perturbation produced greater error for the RPAR group for the 120 mm movement and less error for the 300 mm movement, though these effects were of questionable significance [group × step × amplitude interaction; $F(1,28) = 5.9$, $P < 0.05$; two-step – one-step = –1.5 for 120 mm and –0.9 for 300 mm for the RNC group; +4.4 for 120 mm and –4.0 for 300 mm for the RPAR group].

Consistent with the analyses in the larger groups, the LPAR group demonstrated no error deficits and greater increases in secondary peak velocity as amplitude increased [group × amplitude; $F(1,20) = 5.1$, $P < 0.05$; 300 – 120 mm = +33 for LNC and +56 for LPAR]. However, contrary to the LHD analyses, the LPAR group showed no evidence of trajectory deficits in the initial component

Discussion

These results demonstrate that advance planning for reaching was not affected by LHD or RHD though the LHD group's response initiation was longer, regardless of planning requirements. The most consistent finding in this study was the impairment in response implementation in both groups, with trajectory deficits in the LHD group and final position deficits in the RHD group. These differences were not due to a higher incidence of parietal damage in the RHD group as evidenced by error deficits after right, but not left, parietal damage. Response modification, as reflected by the effect of target perturbations, was associated with decreased secondary velocity in the LHD group and error deficits in the RPAR group, though the latter finding was of questionable significance. However, the LPAR group did not demonstrate velocity deficits seen in the entire LHD group. Due to a small number of patients with damage restricted to the frontal lobes, we were not able to assess directly if the LHD group's trajectory deficits were due to the inclusion of frontal and not parietal patients, which might be expected from previous data in animals and humans (Turner *et al*., 1998; Moran and Schwartz, 1999; Padoa-Schioppa *et al*., 2002). This issue of differential frontal and parietal control of velocity and position requires further evaluation.

LHD produced kinematic impairment primarily in the initial component of the movement, and RHD produced spatial impairment in the secondary component only. Superficially, these findings appear to support the notion that the left hemisphere is specialized for open loop control and the right hemisphere is specialized for closed loop control. However, a closer look suggests that the open and closed loop explanation for hemispheric specialization of reaching was not supported.

The left hemisphere's role in open loop processing and advance planning

Deficits in the LHD group were characterized by slower initial peak velocity with prolongation of the deceleration phase, for the longer amplitude movement. In addition, the secondary component covered a greater distance for longer movements in particular, which probably produced its higher velocity and normal final accuracy. These results are indicative of deficits in the initial component, with differences in the secondary component related to maximizing accuracy. This pattern of results is consistent with previous findings that have found deficits in the initial component after LHD with adjustments in the secondary component after a suboptimal initial movement (Haaland and Harrington, 1989*a*). Other studies (Fisk and Goodale, 1988; Hermsdorfer *et al*., 1999) have also reported deficits in the acceleration and deceleration phases of the trajectory after LHD. While earlier versions of the two-component theory of reaching would have interpreted deficits in the acceleration and deceleration phase as consistent with impaired open loop control after LHD (for reviews see Keele, 1986; Elliott and Carson, 2000), more recent formulations suggest that both phases (Gordon and Ghez, 1987; Desmurget and Grafton, 2000) or the deceleration phase only (Forget and Lamarre, 1987; Heath *et al*., 1998) are dependent on sensory feedback and closed loop control. Therefore, our trajectory data by themselves do not allow a clear conclusion regarding the open loop model of left hemisphere specialization. However, the LHD group's deficit in secondary velocity on two-step trials was suggestive of impaired response modification, which was not predicted with the open loop model of left hemisphere motor control. The fact that the deficit occurred with velocity rather than error is consistent with the notion that LHD affects velocity control, potentially reflecting a deficit selecting the optimal velocity for a given context—whether that velocity is in the initial transport or secondary adjustment phase of the movement.

The LHD group showed no deficits in advanced planning, as reflected by variations in RT, as amplitude or visual feedback cues varied. Advanced planning increased for all four groups whenever the opportunities for planning or response modification during movement implementation were reduced (i.e. when movement amplitude was shorter or when visual feedback of arm position was eliminated), consistent with previous work (Rosenbaum *et al*., 1987; Harrington and Haaland, 1991). The lack of group differences demonstrates that LHD did not produce deficits in planning when this information was cued prior to the RT, which is contrary to what would be predicted if the open loop hypothesis of left hemisphere specialization was true for the reaching task. Deficits in advance planning were also not found after LHD for heterogeneous sequencing tasks even though these sequences required increased planning as sequence length increased (Harrington and Haaland, 1991). It was only LHD patients with apraxia who demonstrated advanced planning deficits for heterogeneous, but not repetitive, sequences. This pattern of results demonstrates that apraxia after LHD is associated with impaired planning when a variety of motor programmes have to be selected, retrieved and organized, but not when a single programme is involved

(Harrington and Haaland, 1992). The low incidence of apraxia in the current LHD sample (n = 5, 22%) precluded the direct examination of this issue. Another study found evidence of greater deficits after LHD than RHD when implementing ipsilesional movements that required greater planning, but RT was not examined (Hermsdorfer *et al*., 1999*a*). In addition, sensorimotor lesions produced ipsilesional deficits in advance planning for movement direction, but no comparisons were made between patients with damage to the right or left hemisphere (Velicki *et al*., 2000). Taken together, these results suggest that advance planning is not differentially impaired after LHD. However, the influence of limb apraxia and intrahemispheric lesion location as well as specific planning requirements should be examined further.

Right hemisphere's role in closed loop processing and response modification

While our finding of greater error only in the RHD group appear to be consistent with greater closed loop processing deficits, consistent with several studies (Haaland and Harrington, 1989*b*; Winstein and Pohl, 1995), the fact that the RHD group demonstrated greater deficits in final error only when visual feedback of hand position was not available suggests that this group used visual feedback to normalize spatial accuracy. They were more dependent on visual feedback than the control group. This is consistent with a previous arm reaching study that reported no error deficits after RHD when visual feedback was available (Haaland and Harrington, 1989*a*), but it is inconsistent with another study, which reported error deficits after damage to either hemisphere (Fisk and Goodale, 1988). However, this latter finding is likely to be due to including trials in which targets were presented in the contralesional hemispace and when target foveation was not allowed. The accuracy deficits demonstrated by our RHD group may be due to proprioceptive or visuospatial deficits, impaired perception of target location or impaired sensory motor transformations for reaching. While it is clear that a variety of visuospatial deficits are more common after RHD than LHD, especially involving the parietal lobes (Benton and Tranel, 1993), deficits in ipsilesional proprioception, target location perception or sensory motor transformations have not been linked consistently to the right hemisphere. Rather, the emphasis has been on the importance of the superior parietal lobe of either hemisphere for controlling perception of target location (Mishkin *et al*., 1983) and visuomotor transformations (Perenin and Vighetto, 1988; Taira *et al*., 1990; Goodale and Milner, 1992). While visuomotor transformation abilities have been examined largely for the contralesional limb, the spatiotemporal deficits seen in the ipsilesional limb with ideomotor limb apraxia are an example of ipsilesional deficits in visuomotor transformations, and these difficulties, in the context of other studies, have been linked to left parietal damage and are characterized by temporal and spatial deficits (Poizner *et al*., 1997; Haaland *et al*., 2000).

Finally, the deficits in final error demonstrated by the RHD group were not associated with deficits in other measures of closed loop processing. Namely, the RHD group showed no deficits modifying their response to large unpredictable changes in target location that occurred at response initiation.

The dynamic dominance hypothesis of hemispheric specialization

Taken together, the kinematic, advance planning and response modification findings after LHD and RHD are not consistent with the open loop/closed loop dichotomy of hemispheric specialization. This pattern of results is more consistent with the dynamic dominance hypothesis (Sainburg, 2002), which was developed to explain hand preference effects in normal right handers. It proposes that the dominant hemisphere/limb system is specialized for controlling limb dynamics as required to specify movement speed and hand-path shape. In contrast, the non-dominant hemisphere/limb system is specialized for controlling static limb position, as required to specify the final position of a reaching movement. This is the first study in brain-damaged patients to suggest that these differences in movement control, which have been reported for the right and left hand of normal right handers, may be related to specialization of the left and right hemisphere. However, Sainburg's model is predicated on studies that have measured intersegmental dynamics and used more rapid movements, which are more likely to be dependent on the control of intersegmental dynamics. Therefore, this notion requires further examination in patients with unilateral hemispheric damage.

The left hemisphere's role in velocity control

All components of the movement were affected by visual feedback, but the relative importance of open and closed loop processing cannot be specified based upon the visual feedback effects because visual feedback was predictable and therefore could affect open or closed loop processing. The preponderance of velocity deficits in the LHD group whenever the context of the task produced increased velocity in the control group suggests that the left hemisphere is particularly important for selecting, retrieving or executing the optimal velocity for a given context. This is consistent with previous studies that have reported deficits after LHD, but not RHD, when paced tapping required a more rapid rate (Carmon, 1971; Tallal, 1983; Sergent *et al*., 1993) and when velocity requirements increased due to increased target size in a Fitts Tapping Task (Haaland and Harrington, 1994). This lack of adaptability in trajectory control was also seen in the movement trajectory of normal right handers who were asked to adapt their movement to a mass placed on the arm (Sainburg and Kalakanis, 2000). Although two other studies reported lower velocities after LHD but not RHD, those velocity differences did not increase as response amplitude increased (Fisk and Goodale, 1988; Haaland and Harrington, 1989*a*). This apparent inconsistency is likely to be related to

reduced speed–accuracy trade-off requirements in the previous studies because movement amplitude was shorter or accuracy demands were lower relative to the current study. In the study of Haaland and Harrington (1989*a*), velocities were less than the current study, and in the study of Fisk and Goodale (1988), velocities were faster, but error was greater. This pattern suggests that the velocity impairment in the LHD group increases when speed requirements are high, but only if the subject is required to adjust velocity in order to maintain accuracy.

Implications of improved performance in the secondary component

The LHD group showed deficiencies in the initial component with improved performance in the secondary component. This improvement was characterized by moving a larger distance at a faster velocity for the larger amplitude movements, such that no group differences were found in final error for this group. The faster than normal secondary response, along with normal velocity for the shorter movements and movements without visual feedback, shows that the LHD group did not demonstrate global velocity deficits. It is unclear if their velocity deficits reflect (i) inability to select, retrieve or execute higher velocities and reliance on the intact right hemisphere's better position control; or (ii) the LHD group's adaptation to the task requirements and the need to decrease velocity in the initial component in order to hit the target accurately (speed–accuracy trade-off) (Harrington and Haaland, 1997). The LHD group's increase in the proportion of the initial movement that was in the deceleration phase is consistent with differential scaling and adaptation after left, but not right, hemisphere damage, suggestive of impaired planning and the necessity of modifying the response during its implementation (MacKenzie *et al*., 1987; Marteniuk *et al*., 1987).

Conclusions

There is considerable variability across studies that have examined hemispheric asymmetry for different aspects of arm reaching probably due to differential requirements of the arm reaching tasks being utilized and differences in the characteristics of the patients being examined. Our findings did not globally support the open and closed loop explanation of hemispheric specialization. Rather, they are consistent with the left hemisphere's greater role in dynamic control of the trajectory and the right hemisphere's greater role in position control, as proposed by the dynamic dominance hypothesis (Sainburg, 2002). However, our previous arm reaching data (Haaland *et al*., 1999) found that there may be limits to the hemispheric separation of temporal and spatial control because apraxics with LHD demonstrated both temporal and spatial deficits. In addition, we showed that relative to a normal control group, apraxic and non-apraxic LHD patients demonstrated slower reaches, which is consistent with the current study. However, in addition, the apraxics demonstrated error deficits. These findings emphasize that hemispheric asymmetry for the temporal and spatial aspects of reaching is a relative rather than an absolute dominance.

Additional work must be done to explore the limits of hemispheric specialization for temporal and spatial control and the importance of the dynamic dominance hypothesis, especially because this hypothesis is based upon hand preference data in healthy controls performing rapid movements, which are more dependent on intersegmental dynamic control. Future studies must control for movement velocity in order to determine if LHD produces positional deficits if patients are forced to perform at normal speeds and determine if there are intrahemispheric differences in position and trajectory control.

Acknowledgements

We wish to to thank Dr Robert Sainburg for valuable discussions, and Gabrielle Mallory, Jennifer Hogan and Lee Stapp for technical help. This research was supported by grants from the Department of Veterans Affairs (K.Y.H. and R.R.L.), the MIND Institute (K.Y.H., J.L.P. and R.R.L.) and NIH NS21135 (R.T.K.).

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