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HEMISPHERIC CONTROL OF THE INITIAL AND CORRECTIVE COMPONENTS OF AIMING MOVEMENTS

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Abstract—This study examined whether the left and right hemispheres play differential roles in controlling the initial and corrective components of aiming movements. A simple aiming task was administered to 31 normal control subjects and 29 unilateral stroke patients (14 with right hemisphere damage and 15 with left hemisphere damage). Movement amplitude was varied (25, 64 and 100 mm) and reaction time, movement time and accuracy were measured. Through a trajectory analysis, initial and corrective movements were separated. The stroke patients performed the task with their ipsilateral arm which was compared to the normal controls' right or left arm performance. Regardless of the movement amplitude the left hemisphere group's reaction time was slower, and the execution of the initial movement component was less accurate than controls. No performance deficits were found on corrective movements. Performance was not impaired for the right hemisphere group on any measures. The results are discussed in terms of the hemispheres' possible roles in controlling movements which are largely open or closed loop.

INTRODUCTION

MOST STUDIES of hemispheric control of movement have emphasized the greater role of the left hemisphere by showing bilateral motor deficits after left hemisphere damage on a variety of motor tasks [6, 9, 11, 14–16, 19, 20, 27–30]. However, some experiments have indicated that the right hemisphere plays a special role in controlling some types of movements [7, 10] or that both hemispheres are equally important [8, 12, 25, 30]. While there is no single explanation for the discrepancies in the literature, significant problems exist in comparing studies because of differences in tasks and because most studies have not verified lesion location and size by CT scan. Further, as most studies have examined performance on only one task, task factors which influence whether the right or left hemisphere is dominant often cannot be identified within the same experiment. This is a serious limitation since tasks vary not only in their complexity but in their emphasis on certain cognitive processes or procedures [13, 21, 22].

One conceptualization of motor skills which differentiates movements in terms of their sensory dependence may be useful in characterizing the motor deficits seen after brain damage. Open loop movements are rapid, programmed and usually sensory independent while closed loop movements are characteristically slower and can be modified by sensory input on a moment to moment basis [17]. Most movements are composed of an initial, open

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loop and a corrective closed loop component, but performance may depend more on one component than the other. This dichotomy may be too simplified as this differentiation typically defines sensory dependence on the basis of visual feedback without regard for the role of proprioceptive feedback and/or efference copies of the motor command [24].

In tasks such as simple aiming or reciprocal tapping [3], the dependence of a movement on open vs closed loop control has been manipulated by changing target size and/or movement amplitude [4, 5, 26]. One recent study [11] which used the FITTS [3] reciprocal tapping task showed that patients with left but not right hemisphere damage had particular difficulty in the larger target condition and thus presumably with movements which are largely open loop. Another study [10] has shown that the accuracy of closed loop aiming movements was impaired after right but not left hemisphere damage.

The purpose of the present study was to investigate whether left or right hemisphere damage differentially affected performance of the initial and terminal components of a simple aiming task in which the initial component was more reflective of open loop processes and the corrective component was more reflective of closed loop processes. In addition, the relative importance of open and closed loop components was varied by manipulating movement amplitude. Open and closed loop components are inferred through variations in amplitude (A) and/or target size (W) which affect the index of difficulty (ID) where $ID = \log_2(2A/W)$ [3]. As the ID increases, so does movement time and, presumably, the extent to which movements are closed vs open loop. WALLACE and NEWELL [26] have directly shown that movements with IDs less than 3.58 were not dependent on visual feedback whereas those to smaller targets with IDs greater than 4.58 were dependent on visual feedback. In the present study, subjects moved a handle as rapidly as possible to targets at various distances with IDs of 3.32, 4.68 and 5.32. Reaction time was measured and the timing (i.e. velocity and acceleration) of these simple aiming movements was directly measured. If left hemisphere damage primarily affects open loop processing, performance deficits of the left hemisphere group should be particularly evident at small ID levels. In contrast, the right hemisphere damaged patients should show deficits only at large ID levels if only their closed loop processing is impaired.

Open loop vs closed loop control can also be partially (but not completely) differentiated by separating the movement into an initial and a corrective component. Aiming responses consist of an initial component which is defined as a symmetrical increase and decrease in velocity followed by a corrective component which is characterized by one or more similar changes in velocity near the completion of the response [1]. The initial movement component has been shown to be unaffected by the absence of visual feedback across a wide range of movement amplitudes [5] even though movement durations have far exceeded estimates (135–300 msec) of the time needed to process and utilize error information [1, 18]. This suggests that although it is possible for visual feedback to be utilized during the initial movement phase, subjects do not choose to do so until they approach the target. Consistent with this analysis are CARLTON'S [1] findings showing that removal of visual feedback had an effect on aiming responses only after 75% of the movement had been completed, again, even though movement durations were greater than 400 msec. However, nonvisual sources of feedback (e.g. proprioceptive and/or efference copy) have been shown to influence what appears to be the initial component without an observable inflexion point in the velocity curve [23] suggesting that this component is not entirely programmed. Nonetheless, it does seem to be less dependent on feedback than the corrective component of movement and this is supported in the present study by our data in normal subjects. Thus, if the initial movement

component is more reflective of open loop processing than ID level, performance should be impaired during this phase after left hemisphere damage regardless of ID level whereas right hemisphere damage should produce deficits in the corrective component across all ID levels.

METHOD

Subjects

Thirty-one right-handed normal control males and 29 stroke (CVA) patients (14 with right hemisphere and 15 with left hemisphere damage) were tested at the Albuquerque Veterans Administration Medical Center (VAMC). Fifteen controls performed the arm reaching task with their right arm, 14 controls performed the task with their left arm, and stroke patients performed with the arm ipsilateral to lesion. The arm contralateral to the lesion was not examined as this would exclude hemiplegic patients and bias the sample. Further, examining ipsilateral performance should minimize factors that are purely motoric or sensory in nature (i.e. hemiparesis or hemianesthesia) allowing for a more accurate account of the anatomical correlates of central control processes in movement.

There were no reliable differences among groups in age or educational level. Subjects in the right arm control group had a mean age of 66.1 (SD = 9.8) and an average of 12.4 (SD = 2.2) yr of education. Subjects in the left arm group had a mean age of 64.8 (SD = 7.1) and an average of 12.1 yr of education (SD = 2.2). The average age of right CVAs was 65 yr (SD = 10), and they had a mean educational level of 12.9 yr (SD = 4.1). The left CVA patients had an average age of 62 yr (SD = 5) and a mean educational level of 12.9 yr (SD = 3.6). Although there was considerable within group variability, Mann–Whitney U tests showed no difference between CVA groups in the average number of months post CVA; 19.6 months (SD = 24.4) for the right CVA group and 26.0 months (SD = 44.6) for the left CVA group.

There were no reliable somatosensory impairments in CVA groups on ipsilateral two-point discrimination of the finger, or ipsilateral finger and arm position sense. Five right CVA patients and five left CVA patients were classified as hemiplegic with hemiplegia defined as contralateral grip strength equal to 0 and ipsilateral grip strength greater than 0. Two right CVA patients and three left CVA patients had visual field cuts, and after patients with field cuts were eliminated, one left CVA patient and five right CVA patients demonstrated visual extinction on bilateral simultaneous stimulation, but neglect was not assessed in other ways.

Apparatus and procedure

Subjects faced a TV monitor and a HI-PAD digitizing table which was interfaced with an Apple II Plus microcomputer that allowed for a 0.125 resolution and sampled points every 10 msec during movement. Subjects held a vertical rod that was attached to a stylus mounted on a track which allowed horizontal movement on the digitizing tablet with minimal resistance. Target circles were projected on the TV monitor to the right or left of the starting point, and the monitor was situated 66.5 cm from the patient. Subjects using their right arm always moved the stylus from the center point to the right, and those using their left arm moved the stylus from the center point to the left. Movements of 25, 64 and 100 mm amplitudes were made to a target that was 5 mm in diameter. The target diameter and movement amplitude combinations produced indices of difficulty of 3.32, 4.68 and 5.32 respectively.

At the beginning of each trial, subjects were presented with a starting point stimulus on the monitor and were instructed to move their arm so that the stylus was on top of the starting point. Fifty to 100 msec after a subject had positioned the stylus on the starting point, a tone provided feedback that the stylus was correctly positioned. Then after a 1–2 sec variable delay, the target circle moved to the left or right, and subjects were instructed to move as quickly and accurately as they could to the target circle.

Stimuli were blocked according to movement amplitude such that within each block there were 30 trials at each movement amplitude for a total of 90 trials. The order of blocks was counterbalanced across subjects. After each 10 trials, there was a brief resting period. If reaction time (RT) exceeded 1000 msec or if movement time (MT) exceeded 3000 msec, the trial was aborted and considered an error. All error trials were repeated at the end of a block. The duration of each session was 20 min per arm.

Measurements

Reaction time (RT) was defined as the time from onset of target to the beginning of the movement. Initial movement time was the time that elapsed from the end of RT to when movement velocity returned to baseline reflecting the beginning of the corrective component of the movement. The end of the corrective movement component was defined by when the subject entered the target circle and remained inside either for 1000 msec, or if they did not remain in the target circle for 1000 msec by the last movement that was made outside of the target circle. Movements made inside the target circle after the target was reached were not considered part of the correct movement.

For both initial and corrective movement components, several measurements were calculated. MT was the interval from the beginning of movement to the end. MTs for the corrective component excluded those trials where MTs were zero; however, analyses of MTs including those trials showed similar trends. Constant error (CE) was the

mean deviation from target at the end of the initial and corrective movement. Variable error (VE) was the standard deviation of CE. Average velocity was computed only for the initial movement component.

CT scan quantification

CT scans were available on all CVA subjects. CT scan parameters were quantified in order to obtain measures of lesion size and location (anterior vs posterior). Computerized procedures were used to quantify lesion volume and location (anterior vs posterior) [31]. Lesion size was expressed as a ratio of lesion volume to brain volume, in order to take into account individual differences in brain size. Lesion location was quantified in two ways: in terms of the proportion of the total lesion volume that was located anterior and posterior and in terms of the distance of the lesion from frontal and occipital poles. To specify the anterior and posterior extent of the lesion, the midpoint of each slice was defined as the point halfway between the frontal and occipital pole. The anterior distance of the lesion was expressed as the average distance (across slices) of the most anterior extension of the lesion from the frontal pole, and the posterior distance was the average distance of the most posterior extension of the lesion from the occipital pole. Measures of frontal and posterior distance were divided by total distance so as to represent a proportion of the slice length.

RESULTS

Data were analyzed separately for the right and the left CVA groups by analyses of variance (ANOVAs) which compared each CVA group with their respective control group. Both sets of analyses were based on a mixed model design where group (i.e. control vs CVA) was a between subject factor and movement amplitude was a within subject factor. Group and the interaction of group with movement amplitude were the effects of interest. Separate analyses were performed for each dependent measure and a 0.01 alpha level of significance was adopted to control for Type II errors.

TABLE 1. Reaction time and initial and corrective movement component means (SD)

	Left Controls	Left CVAs	Right Controls	Right CVAs
Reaction time: mean	404 (64)	510 (89)*	428 (75)	472 (80)
Reaction time: SD	80 (20)	104 (26)*	94 (28)	100 (23)
Initial component				
Movement time: mean	563 (83)	620 (76)	547 (95)	550 (103)
Movement time: SD	92 (27)	150 (34)*	94 (38)	121 (70)
Constant error†	5.1 (2.2)	9.6 (5.4)*	4.3 (2.6)	10.3 (10.6)
Variable error‡	5.3 (1.6)	9.0 (3.4)*	4.9 (2.2)	8.2 (5.9)
Average velocity: mean	98.5 (19)	80.4 (14)*	104.9 (30)	103.6 (32)
Corrective component				
Movement time: mean	488 (120)	695 (142)*	520 (144)	718 (349)
Movement time: SD	278 (99)	366 (81)	293 (109)	343 (121)
Constant error	0.04 (0.05)	0.13 (0.25)	0.03 (0.03)	0.81 (1.85)
Variable error	0.19 (0.18)	0.49 (0.80)	0.13 (0.09)	1.58 (2.92)

Note. Data points are averaged across movement amplitudes. Measures of time are expressed in msec and measures of errors are expressed in mm.

*Significant ($P<0.01$) group effect.

†Constant error is the mean deviation from target.

‡Variable error is the standard deviation of constant error.

Reaction time and initial movements

Table 1 shows that RT was significantly longer for the left CVA group in comparison to their controls [$F(1, 29)=14.76$, $P<0.001$] at all movement amplitudes, but there was not a reliable difference between the right CVA group and their control group. The left CVA group

also showed more within subject variability in RTs relative to their control group [$F(1, 29) = 8.56$, $P < 0.01$] whereas no such differences were found for right CVAs. These findings supported the view that the left hemisphere is particularly important for the programming of discrete aiming movements regardless of movement amplitude.

As for the timing of the initial movement component, the right CVA group was not impaired on any of the speed or velocity measures, regardless of movement amplitude. While MTs for the left CVA group were not reliably longer than their controls at any movement amplitude, left CVA patients showed greater within subject variability in MT across all amplitudes in comparison to the control group [$F(1, 29) = 27.68$, $P < 0.001$]. However, averaging across amplitudes, average velocity was about 18 mm/sec slower in comparison to their controls [$F(1, 29) = 9.16$, $P < 0.01$]. The dissociation between MT and velocity effects for left CVA patients can be explained by the finding that the total distance traveled during the initial movement was on the average 5 mm less for left CVAs than their controls [$F(1, 29) = 8.38$, $P < 0.01$]. No such differences in distance traveled were found for the right CVA group. These findings suggest that the left hemisphere also is involved in the initial execution of discrete aiming movements.

While Table 1 suggests that the right CVA group was less accurate in executing the initial movement component, this was not a statistically significant effect, although there was considerable within group variability in both CE and VE. An examination of the distributions of CE and VE for right CVAs showed that the large standard deviations were related to one or two subjects who showed very large errors; otherwise, the distributions were reasonably normal. To avoid biasing the sample by removing these outliers, these results were reanalyzed using the Mann–Whitney U test and still no differences in CE or VE were found between the right CVA group and their controls, regardless of movement amplitude. As for the left CVA group, they showed significantly greater CE and VE than their controls at all amplitudes [$F(1, 29) = 9.57$, $P < 0.01$ and $F(1, 29) = 15.05$, $P < 0.001$, respectively] suggesting that their initial movements were generally further from the target (CE) and showed greater variability independent of deviation from target. For all significant effects, the pattern of deficits found for the left CVA group was the same regardless of movement amplitude. These findings suggest that the left hemisphere plays an important role in the programming and/or execution accuracy of the initial movement component regardless of movement amplitude.

Corrective movements

While Table 1 suggests that both CVA groups took longer to make corrective movements, the analyses indicated that only the left CVA group was significantly slower in executing corrections relative to their controls [$F(1, 29) = 16.93$, $P < 0.001$]. This finding is not surprising as they had to travel a greater distance to reach the target since on the average the distance traveled during the initial movement was less relative to control subjects. Because there was considerable within group variability in MTs for the right CVA group, a reanalysis of MTs using the Man–Whitney U test was done because of two outliers. This analysis showed no difference in MTs between the right CVA group and their controls.

As for the accuracy of corrective movements, Table 1 suggests that both CVA groups made more errors; however, the analyses failed to find reliable differences in CE and VE for left or right CVA groups in comparison with their respective control group. An examination of the distributions for CE and VE showed one or two right CVAs and one left CVA with unusually large errors, but otherwise the distributions appeared reasonably normal. A

reanalysis of these data to avoid bias by one to two outliers in each group used the Mann–Whitney U test and found no significant impairment in accuracy for either CVA group.

Effects of no visual feedback

To directly test our assumption that the initial movement is largely independent of visual feedback whereas the corrective component is not, five additional normal control subjects (between the ages of 62 and 70) performed the simple aiming task with and without visual guidance. In the no visual guidance condition subject feedback was removed prior to movement initiation and remained unavailable for 1100 msec, after which visual feedback was restored. For four of the subjects the absence of visual feedback during the initial movement had no effect on MT or CE regardless of amplitude. For the other control subject, removal of visual feedback on 25 mm movements did not affect MT or CE for the initial movement, but it was difficult to assess the effects on 64 and 100 mm movements due to large variances and speed/accuracy trade-offs between visual and non-visual conditions. For all control subjects, the removal of visual feedback increased MT and CE on the corrective movement component.

CT analyses

Table 2 presents the lesion volume and distance measurements for each CVA group. Because the within group variability in lesion volume was large, the Mann–Whitney U test was used for comparisons between left and right CVA groups. The analyses showed that both CVA groups were equivalent in total lesion volume. In addition, there was no reliable difference between groups in the proportion of lesion volume that was anterior or posterior. The anterior–posterior lesion volume difference in Table 2 indicates that both group's lesions are more anterior than posterior, and the Mann–Whitney U test showed that there was no difference between CVA groups on this measure. An examination of the distributions showed that four right CVAs and two left CVAs had greater posterior than anterior lesion volumes, but, otherwise, the distributions were similar between groups. Finally, recall that the lesion

TABLE 2. Means (SD) for lesion volume and location measures

	Left CVAs	Range	Right CVAs	Range
Lesion volume*	3.3 (4.0)	0.1–13.0	4.6 (4.7)	0.4–17.1
Anterior lesion volume†	65.2 (37.4)	0–100	66.0 (34.4)	0–100
Posterior lesion volume†	34.3 (37.4)	0–100	34.0 (34.4)	0–100
Difference: anterior–posterior volume†	30.3 (74.7)	−100–100	32.1 (68.8)	−100–100
Anterior distance‡	34.8 (17.8)	12.0–69.3	30.0 (18.3)	4.7–77.2
Posterior distance‡	38.3 (18.9)	0.6–66.2	35.3 (16.7)	6.8–54.6

*Lesion volume measure is proportional to the total brain volume.

†These volume measures are proportional to the total lesion volume.

‡Distance measures are proportional to the total distance from the frontal to the occipital pole. A smaller proportion for the anterior and the posterior distance reflects that the lesion is located closer to the frontal or occipital pole, respectively.

distance measures reflect the extent to which lesions are located near the frontal or occipital poles. Table 2 shows that the anterior and posterior distance measures were similar between CVA groups, which was supported by the statistical analyses.

DISCUSSION

While the role of nonvisual sources of influence (e.g. proprioceptive and efference copy of the motor program) on the initial movement cannot be ruled out [24], from a relative standpoint the initial component appears to be more reflective of programming or open loop control than the corrective component [1, 5]. Using RT and initial movements as more reflective of open loop control and corrective movement measures as indicative of closed loop control, the current results support our previous findings that the left hemisphere may play a more important role in open loop control. These results are consistent with previous findings [6, 14–16, 20, 27–29] emphasizing the greater role of the left hemisphere in controlling a wide variety of movements including discrete aiming. The present experiment also adds critical lesion location and lesion volume data to demonstrate that the greater deficits of the left hemisphere group cannot be attributed to differences in lesion size and location.

Recently, FISK and GOODALE [2] also examined the kinematics of movement in the ipsilateral limb of patients with right or left hemisphere damage. Although this study did not separate the trajectory into distinct initial and corrective components, similar to our findings, the left hemisphere group showed deficits in execution (e.g. slower velocity and greater CE). In contrast to our findings, in their study the left hemisphere group showed no impairment in RT and the right hemisphere group also showed deficits in execution, but statistical verification was not always provided. While there were procedural differences, an important problem with comparing these two studies lies with Fisk and Goodale's subject population. In their study the two patient groups differed in etiology (i.e. 82% CVAs and 12% tumors in the left hemisphere group; 54% CVAs and 36% tumors in the right hemisphere group), and lesion size and location was not consistently specified by CT scan for either group. Because all of our subjects had strokes and lesion size and location was verified by CT, our analyses were able to rule out differences between groups in these factors that otherwise might potentially explain our results.

Our results did not support the prediction of greater closed loop deficits (as reflected by the corrective component) after right hemisphere damage as this group showed no deficits on the corrective movement measures regardless of movement amplitude. However, consistent with our prediction, the right CVA group was not impaired on any measures associated with the initial movement. The findings for the right CVA group are similar to a previous experiment [11] where the right hemisphere group showed no deficits on open or closed loop conditions in a reciprocal tapping task. In contrast, other studies have shown better left hand performance in normals in a ballistic aiming task without visual guidance [7] and poorer ipsilateral arm performance after right hemisphere damage in a nonballistic aiming task [10]. These findings are clearly not supported in the present study. In addition, another study [8] found deficits on tasks that required sensory–motor interaction. Given this finding, larger amplitude movements (64 and 100 mm) which have higher IDs and are presumably more sensory dependent would be expected to show greater ipsilateral deficits after left or right hemisphere damage. This was clearly not the case in the present study or in other studies [10, 11]. However, because we did not vary target size in the present experiment and

because target size in our previous study [11] was not very small, our experiments may not have emphasized sensory–motor integration processes. Future experiments need to extend the range of target widths so as to emphasize the importance of sensory–motor processing, and provide a better test of the role of the hemispheres in controlling closed loop movement.

While the theoretical conceptualization of open and closed loop movements has been consistent, the operational definition of these two components has varied considerably across experiments which may explain the discrepant findings detailed above for the right hemisphere group. It is generally agreed that increasing target width or decreasing movement amplitude increases the open loop component of movement [17, 26]. However, the left hemisphere group demonstrated similar deficits in the initial movement component at each amplitude and the right hemisphere group showed no deficits at any amplitude. There are two possible explanations for these findings. First, the ID levels used in the present study involved primarily open loop processing [26] which would explain our negative findings for the right CVA group. This explanation implies that ID levels may not necessarily be comparable across studies in terms of whether subjects rely on open vs closed loop processing. For example, the degree of open vs closed loop processing may be affected differently by manipulations of target width and amplitude. Alternatively, when aiming responses are decomposed into initial and corrective components using the acceleration/deceleration patterns of the movement, open and closed loop processing can be more accurately specified independent of ID level. Our findings from the no-visual feedback condition on a small sample of control subjects generally supported this view. Also consistent with this explanation are studies showing that execution of the initial movement component of aiming responses is not affected by visual feedback across a wide range of ID levels [5], and that visual feedback does not influence performance until 75% of the movement has been completed [1]. However, because our study did not include a no-visual feedback condition or examine other sources of peripheral and central influence for CVA patients, the underlying mechanisms of hemispheric control of the initial movement component require further verification.

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