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Limb-Sequencing Deficits after Left but not Right Hemisphere Damage

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The performance of right and left hemisphere stroke patients was compared to normal control groups on a task where subjects alternately hit two targets which varied in size from 0.5 to 6.5 cm. The stroke patients used the arm ipsilateral to damage, and the control groups used the same arm as their respective stroke group. Lesion size and location were similar for the two stroke groups. No deficits were found for the right hemisphere stroke group. The left stroke group's tapping speed was not slower at the smallest target, but became progressively slower relative to the control group's as target size increased. Variability in tapping speed increased as target size increased for all except the left stroke group. While the entire left stroke group was as accurate as their controls, the apraxic, but not nonapraxic, patients made more errors on smaller targets only. Two explanations for these findings both emphasize the left hemisphere's special role in motor programming; one focuses upon its dominance for movements which are independent of sensory feedback and the other emphasizes its specialization for processing rapid temporal information.

INTRODUCTION

Numerous studies have shown left hemisphere damage produces bilateral deficits on a wide variety of motor tasks whereas right hemisphere damage is more likely to produce strictly contralateral deficits (see Haaland & Yeo, 1989 for review; Wyke, 1967, 1968, 1971; Kimura, 1977; Kimura & Archibald, 1974; Haaland & Delaney, 1981; Haaland & Harrington, 1989). This asymmetry may relate to the predominance of right hand preference in the normal population (Annett, Annett, Hudson, &

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Turner, 1979; Flowers, 1975; Peters & Durding, 1979; Todor & Cisneros, 1985). Left hemisphere motor dominance has been associated with the production of single movements (DeRenzi, Motti, & Nichelli, 1983; Kolb & Milner, 1981; Kimura, 1982; Harrington & Haaland, 1991), sequencing (DeRenzi et al., 1981; Harrington & Haaland, 1991; Kimura & Archibald, 1974), memory for sequences (Jason, 1983), and the perception and production of rapid sequential information (See Hammond, 1983 for review; Sergent, 1982; Stark & Tallal, 1979; Kent & Rosenbek, 1983). However, the specific aspects of motor programming which are controlled primarily by the left hemisphere have not been well defined, and the task domains in which these processes operate have not been specified.

Several theories have been put forth to explain the left hemisphere's specialization for controlling movements. As limb apraxia is seen predominantly with left hemisphere damage, an early theory (Liepmann, 1913; Geschwind, 1965) proposed the left hemisphere was specialized for controlling complex, purposeful movements, such as gestures used by clinicians to test for limb apraxia. However, because the performance of gestures involves many different component processes, one approach to understanding the mechanisms for left hemisphere control of movement has been to systematically vary characteristics of movement that ostensibly affect certain aspects of processing. Two explanations for left hemisphere dominance of movement control have emerged from this method.

One theoretical view (Fisk & Goodale, 1988; Haaland & Harrington, 1989) suggests the left hemisphere is especially important for controlling the ballistic component of movements. The ballistic or open loop component is minimally dependent upon sensory feedback and is largely preprogrammed (Keele, 1986). For example, simple aiming movements to hit large targets are primarily open-loop because they are less dependent on visual feedback whereas movements to hit smaller targets are more closed-loop because the removal of visual feedback affects performance (Wallace & Newell, 1983). The increase in movement time which is seen as target size decreases is attributable to increases in the deceleration phase of the movement rather than the ballistic acceleration phase (Todor & Cisneros, 1985) which suggests that the percentage of the total movement time which is due to more sensory dependent processes increases for smaller targets. Evidence for left hemisphere control of open loop movements was found in one study (Haaland, Harrington, & Yeo, 1987) showing performance in patients with left hemisphere damage was more impaired for large than small targets whereas no sequential aiming deficits were found in patients with right hemisphere damage, regardless of target size.

Studies of hand preference in the normal population have suggested the left hemisphere is specialized for closed-loop control of movement. This conclusion is based upon experiments which have examined the

difference in the performance of the right and left hand, assuming right hand superiority reflects left hemisphere functioning. These studies have consistently demonstrated better right hand performance on tasks which emphasize closed-loop or sensory-dependent processing (Annett et al., 1979; Flowers, 1975; Peters & Durding, 1979; Todor & Cisneros, 1985) concluding the left hemisphere is specialized for closed-loop control. If this is the case, patients with left hemisphere damage should demonstrate greater deficits on movements to small targets which are more sensory-dependent (Wallace & Newell, 1983).

While an earlier study (Haaland et al., 1987) found that a left-hemisphere-damaged group had greater deficits in rapid, sequential movements to a larger than a smaller target, the study had three limitations. First, only two target sizes were used which did not allow a clear separation between open- and closed-loop movements because the left-hemisphere group showed deficits on the smaller target width, even though the deficit was less in comparison to the large target. Second, the left-hemisphere stroke group had more anterior damage than the right-hemisphere group suggesting the possibility that intrahemispheric lesion location could explain the hemispheric differences. Third, left-hemisphere-damaged patients with and without limb apraxia were not compared. While many have assumed the higher incidence of limb apraxia with left-hemisphere damage accounts for the higher incidence of bilateral motor deficits in this group, few studies (Heilman, 1975; Haaland, Porch, & Delaney, 1980) have tested this assumption. One recent report (Harrington & Haaland, 1992) showed some cognitive–motor deficits were associated with left-hemisphere damage regardless of limb apraxia, while other deficits were seen only in the limb apraxic patients suggesting the possibility that apraxic patients may show a different pattern of cognitive–motor deficits than nonapraxic left-hemisphere-damaged patients.

The present experiment was designed to extend the Haaland et al. (1987) study by examining sequential tapping to a wider range of target sizes in left- and right-hemisphere-damaged patients who were matched for lesion size and intrahemispheric lesion location. If the left hemisphere is specialized for closed-loop, sensory-dependent movement, as the hand preference literature suggests, the group with left-hemisphere damage should show greater deficits in movements to the smaller targets. If the left hemisphere controls open-loop movements, the left-hemisphere group should show greater deficits in movements to larger targets. In addition, the performance of left-hemisphere-damaged patients with and without limb apraxia was compared to determine if deficits could be attributed to the inclusion of patients with limb apraxia or if they were more general to left-hemispheric damage.

METHODS

Subjects

Eighteen stroke patients with right-hemisphere damage, 25 stroke patients with left-hemisphere damage, and 32 normal control subjects were examined. All subjects were right-handed, and their handedness scores were comparable ($F < 1.0$) across the four groups using the Edinburgh Handedness Questionnaire [Means and Standard Deviations for Right Control: 79(21); Left Control: 79(26); Right Stroke: 81(15); Left Stroke: 79(17)]. Fifteen of the control subjects performed with their right hand and 17 performed with their left hand. The arm ipsilateral to lesion was examined in the stroke patients. To control for hand preference effects the left stroke group's performance was compared to the control group using their left hand and the right stroke group's performance was compared to the control group using their right hand.

Medical records were reviewed from patients who had had a thromboembolic stroke within the last 10 years and using CT scan had evidence of an infarct. Control subjects showed no evidence of neurologic disease on chart review. Patients or control subjects with admission for alcohol abuse, psychiatric problems, or diagnoses which can produce peripheral motor problems (e.g. peripheral neuropathy, etc.) were excluded from the study.

All groups were matched on age and education. Although in Table 1 the mean time since stroke appears to be greater for the left stroke group, this difference was not statistically significant [$p > .05$], likely because the variability was large in both stroke groups.

Table 1 provides a description of subjects' intellectual, spatial, linguistic, and simple motor skills. The left hemisphere group performed more poorly than the right hemisphere group on all language measures including auditory comprehension [$t(36) = -4.37$, $p < .001$] (DeRenzi & Vignolo, 1962), speech fluency [$t(33) = 3.21$, $p < .005$] (Goodglass & Kaplan, 1983), and repetition [$t(32) = 3.07$, $p < .005$] (Goodglass & Kaplan). There were no differences between the stroke groups on the Information or Block Design subtests of the Wechler Adult Intelligence Scale, Revised Form (Wechsler, 1981). Ipsilateral motor skills were comparable across the two stroke groups on rapid index finger tapping in one location and on grip strength. There were nine hemiplegics in each of the stroke groups based upon neurologic exam. Apraxia was assessed using a 15-item battery (Haaland & Flaherty, 1984). Subjects were videotaped as they imitated unilateral meaningless (e.g., thumb on forehead), transitive (e.g., brush teeth), and intransitive (e.g., wave goodby) limb movements. Those who scored 10 or less (out of a maximum of 15 points) were classified as apraxic, based upon previous data in normal control subjects. Table 1 shows that 12 of the left stroke patients and 3 of the right stroke patients were apraxic.

Apparatus and Procedure

The Fitts Tapping apparatus (Fitts, 1954) was used. The subject was asked to alternately tap between two targets as rapidly and as accurately as possible. The distance between the two targets was 32 cm. Target widths (W) of 0.5, 1, 2, 4, 5, and 6.5 cm were used which yielded Indices of Difficulty (IDs) of 7, 6, 5, 4.03, 3.68, and 2.29, respectively ($ID = \log_2 2A/W$). The ID has been associated with task difficulty and movement time (Keele, 1986). The vertical target plates were surrounded by error plates. Contacts of the stylus on the target and the error plates were electronically counted for each trial.

Subjects held a metal-tipped stylus and alternately hit the two targets for 20 sec on each trial. Five consecutive trials at each target width were presented, and target width order was randomized across subjects. For each subject three dependent measures were computed: (1) Speed: Average number of hits across five trials for each target width; (2) Intertrial Variability: Standard deviation of hits across the five trials for each target width; and (3) Mean

TABLE 1
Means (Standard Deviations) of Descriptive Data

	Left control (N = 17)	Left stroke (N = 25)	Right control (N = 15)	Right stroke (N = 18)
Demographic data				
Age (yr)	64 (6)	63 (7)	68 (7)	63 (12)
Education (yr)	13 (2)	12 (2)	12 (1)	11 (4)
Time since stroke (mo)		36 (9)		17 (24)
Number of apraxics	0	12	0	3
Cognitive data				
General information[b]	12.0 (3.0)	8.0 (4.0)	11.0 (2.0)	9.0 (3.0)
Block design[b]	9.0 (2.0)	7.0 (3.0)	8.0 (1.7)	6.0 (3.0)
Token Test errors[c]	2.6 (2.0)	10.0 (7.0)[a]	2.5 (1.8)	3.3 (3.1)
Speech rating[d]	6.8 (0.2)	5.0 (2.0)[a]	6.9 (0.2)	6.4 (0.2)
Repetition[d]	7.8 (0.6)	5.0 (3.0)[a]	7.9 (0.4)	7.4 (1.0)
Ipsilateral motor[e]				
Grip strength	51.0 (7.0)	48.0 (9.0)	43.0 (8.0)	51.0 (16.0)
Finger tapping	47.0 (6.0)	48.0 (6.0)	46.0 (7.0)	47.0 (6.0)

[a] Poorer performance of the left vs. the right stroke group, $p < .005$.
[b] Scaled scores.
[c] Part V of Token Test (DeRenzi & Vignolo, 1962).
[d] Speech fluency ratings and repetition of low probability sentences were from the Boston Diagnostic Examination of Aphasia (Goodglass & Kaplan, 1983).
[e] T-scores were based on normative sample obtained at the University of Wisconsin; the mean of the normative sample = 50.

number of errors which was the number of times the stylus hit the error plate averaged across the five trials for each target width.

CT Scan Quantification

Lesion size was quantified by tracing the area of the infarct and the area of the brain on a digitizing tablet and calculating the total lesion volume and brain volume across all slices. Lesion volume was expressed as a function of brain volume in order to control for variations in brain size. Lesion location was expressed as linear and volumetric measures. The linear measures (averaged across CT slices) were based upon calculating the distance between the anterior or posterior aspect of the infarct and the frontal and occipital pole, respectively. These measures were calculated as a proportion of total distance from the frontal and occipital pole, and the distance measures were weighted according to their respective lesion volume for each slice. The volumetric measures were the percentage of the infarct (averaged across slices) located anterior and posterior to a point halfway between the frontal and occipital poles. For descriptive purposes lesion location was also tabulated as a function of lobe.

RESULTS

The data were analyzed using a mixed model design with brain damage (control and stroke) and hand (left and right) as the between-subject fac-

tors and target width as the repeated factor. The effects of these factors and their interactions were tested in a multivariate analysis of variance (MANOVA) with number of hits, variability in hits, and errors as the multiple dependent measures. A multivariate approach to repeated measures was employed to adjust for heterogeneity of treatment-difference variations. Follow-up planned comparisons first contrasted the two control groups and then compared each stroke group with its respective control group. Due to the small sample sizes in these follow-up analyses, a univariate approach to repeated measures was adopted applying the Huynh–Feldt correction for heterogeneity of treatment-difference variances.

Comparisons across All Groups

Figures 1 and 2 suggest mean number of hits and mean intertrial variability in hits (standard deviation) changed with target width differently for the control and stroke groups, especially the left stroke patients. In contrast, Table 2 shows there was little difference among the groups in the pattern of errors across target widths. The supporting MANOVA with hits, intertrial variability in hits, and errors as the dependent variables showed a significant brain damage (stroke and control) × hand × width interaction [$F(15, 57) = 1.86, p < .05$].

Follow-Up Comparisons between Control Groups

Follow-up analyses of this interaction first contrasted the two control groups to determine if there were effects of performing hand that were dependent on target width. If no effects of performing hand are found, the two control groups can be combined to test the effects of group (control, right stroke, left stroke). If the hand effect is significant, follow-up ANOVAs will be done separately comparing each stroke group with their respective control group.

A MANOVA revealed a significant hand effect [$F(3, 28) = 3.49, p < .05$], but only a trend for a hand × width interaction [$F(3, 81) = 1.60, p = .119$]. Follow up analyses of variance (ANOVAs) revealed no differences between the two control groups in mean hits or errors, regardless of target width. For both control groups, hits increased as target width became larger [$F(2, 68) = 209.20, p < .001$] and errors increased as target width became smaller [$F(3, 98) = 10.24, p < .01$]. However, the intertrial variability in hits was greater for the right than the left control group [$F(1, 30) = 4.41, p < .05$], and hand interacted with target width [$F(4, 121) = 3.32, p < .025$] such that the variability in tapping rate changed with increases in target width differently for the dominant and nondominant hand. Hence, the similar pattern of mean hits and error rates between the dominant and the nondominant hand are deceptive as these

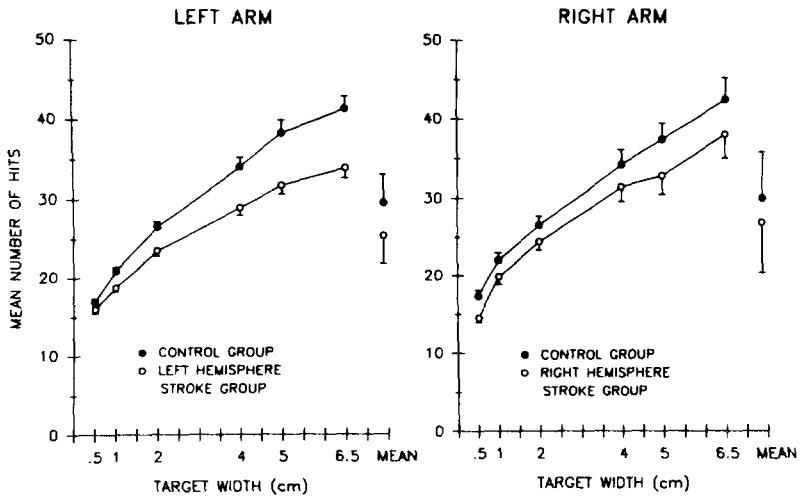

FIG. 1. Mean number of hits (standard error bars) for the left- and right-hemisphere stroke groups and their control groups.

measures are influenced by the intertrial variability in hit rate across the five consecutive trials for each target width. In other words, mean hits and/or error rates may have differed between the control groups if the intertrial variability in hit rates between the two groups were controlled. The greater intertrial variability in dominant-hand performance was consistent with the larger *between-group* standard deviations for the right than the left control group for mean hits [$F(1, 31) = 2.63$, $p = .067$] and

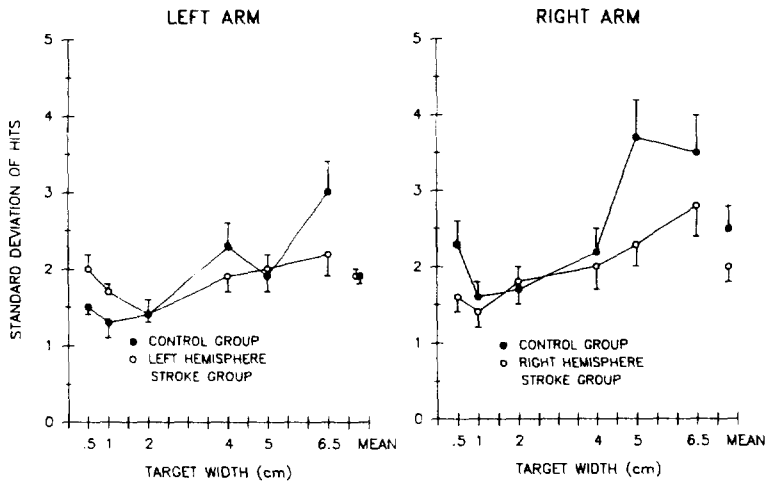

FIG. 2. Mean intertrial variability of hits (standard error bars) for the left- and right-hemisphere stroke groups and their control groups.

TABLE 2
Mean Errors[a] as a Function of Target Width for the Stroke and the Control Groups

	Target width					
	0.5	1.0	2.0	4.0	5.0	6.5
Left control	2.3	2.2	1.2	.7	1.2	.4
	(.6)	(.5)	(.4)	(.2)	(.5)	(.1)
Left stroke	2.5	2.9	2.3	2.0	1.9	.9
	(.8)	(.8)	(.8)	(.8)	(.9)	(.3)
Right control	1.3	1.6	1.2	.4	.9	.3
	(.3)	(.3)	(.3)	(.1)	(.3)	(.1)
Right stroke	3.3	2.6	2.2	1.1	.9	1.0
	(1.2)	(.6)	(.6)	(.3)	(.2)	(.5)

[a] Standard errors in parentheses.

mean variability in hits [$F(1, 31) = 3.23, p < .05$] (see Figs. 1 and 2) but not mean errors (see Table 2). Similarly, when all subjects performing with the right hand (i.e., right control and right stroke groups) were compared with those performing with the left hand (i.e., left control and left stroke groups), between-group standard deviations were larger in subjects performing with the right hand [Mean hits $F(1, 74) = 2.38, p < .01$; Variability in hits $F(1, 74) = 3.32, p < .001$]. In contrast, the between-group standard deviations for mean hits and variability in hits were similar when comparing the two stroke groups with the two control groups. This demonstrates that between-group variability in speed and intertrial variability in speed was due to the performing hand, not brain damage. However, both stroke groups showed greater standard deviations in mean errors than the two control groups [$F(1, 74) = 6.49, p < .001$]. These findings indicate that the psychometric properties of hit rates and variability in hit rates are affected by the performing hand and not by brain damage. Thus, the remaining planned comparisons contrasted each stroke group with its respective control group to eliminate any confounding effects of performing arm and presence of brain damage, and to control for left and right arm differences in between-group variance.

Follow-Up Comparisons between Control and Stroke Groups

A MANOVA comparing the left control group with the left stroke group on all three dependent measures revealed a significant effect of group [$F(3, 38) = 5.58, p < .01$], width [$F(5, 164) = 46.10, p < .001$], and a group × width interaction [$F(5, 165) = 3.26, p < .01$]. The MANOVA contrasting the right control and right stroke groups showed an effect of group [$F(3, 29) = 3.38, p < .05$] and width [$F(2, 63) = 27.23, p < .001$] but no group × width interaction [$F < 1.0$].

Follow-up ANOVAs were conducted for each dependent measure to identify the locus of the group and group × width effects. Figure 1 shows the overall speed of the left stroke group was slower than their control group [$F(1, 40) = 13.91$, $p < .001$]. More importantly, the differences in speed between the groups varied as a function of target size [$F(2, 96) = 6.73$, $p < .01$], such that the left stroke group performed progressively worse as target width increased. Planned comparisons showed that the left stroke group was slower than the control group at all target widths [$p < .01$] *except* the smallest. Further, speed increases between the smallest and largest targets were less for the left stroke group than for their control group [$F(1, 40) = 9.98$, $p < .01$]. Speed did not differ significantly between the right stroke group and their control group, regardless of target width.

Table 2 shows that greater speed with increasing target size could not be explained by speed–accuracy tradeoffs because errors decreased as target widths increased for groups performing with the right [$F(4, 129) = 7.68$, $p < .001$] and the left arm [$F(2, 70) = 4.62$, $p < .05$]. There also were no overall differences in mean errors between either stroke group and their respective control group [$p > .05$], and group did not interact with target width [$p > .05$] for subjects performing with the right or the left arm.

Although the left and the right stroke groups did not differ from their respective controls in the intertrial variability of speed (i.e., averaged across target widths), variability changed differently as a function of width between both of the stroke groups and their respective control groups [left arm $F(4, 144) = 3.19$, $p < .025$; right arm $F(4, 123) = 2.45$, $p < .05$]. As can be seen in Fig. 2, the explanation for these interactions was different in the groups performing with the right and left arm. The left control group's variability increased as target width increased [$F(4, 62) = 9.14$, $p < .001$] whereas no such effect of target width was found for the left stroke group [$F(3, 70) = 2.14$, $p = .10$]. Interestingly, the left stroke group's intertrial variability was not statistically different ($p > .05$) than their control group at any target width. In contrast, both the right control and the right stroke groups showed greater variability with increasing width [Right control group $F(4, 56) = 8.22$, $p < .001$; Right stroke group $F(5, 85) = 7.68$, $p < .001$], and the amount of change in variability between the 0.5- and 6.5-cm target widths was similar between the groups. However, the right stroke group showed less variability in hits only at the 5-cm target width [$F(1, 31) = 6.19$, $p < .02$] which accounted for the group × target width interaction.

In summary, left hemisphere damage did not impair accuracy but did slow performance at all but the smallest target. Unlike their control group, the left-hemisphere-damaged patients showed no change in the variability of speed as target size increased. In contrast, right-hemisphere

damage was not consistently associated with any performance abnormalities. Although the right stroke group's performance differed from their controls in the MANOVA where the linear combination of errors and intertrial variability best discriminated between the groups [Standardized Discriminant Function Coefficients: Hits = −.17; Intertrial Variability = −1.18, Errors = 1.2], this was equally true for all target widths. In addition, the right stroke group was not impaired on any single performance measure except for one isolated finding on intertrial variability. Hence, it is unlikely that right-hemisphere damage can be linked to the processing deficits examined in this study. These findings cannot be attributed to the larger sample sizes in the left stroke group because when a random sample of seven left stroke patients was eliminated from the analyses (to equalize the sample sizes of the stroke groups) similar results were obtained on all performance measures.

Limb Apraxia and Sequencing

To examine the relationship between limb apraxia and sequencing, the left stroke patients who were apraxic were compared with the left nonapraxic stroke patients on all measures of the Fitts task. These data are presented in Fig. 3. There was no difference between the apraxic and nonapraxic patients in speed, and speed increased with target width similarly for both groups. The mean intertrial variability of speed was slightly higher for the apraxic group [$F(1, 23) = 8.16$, $p < .01$], but similar to the total left-hemisphere group, variability did not change significantly as a function of target width for either group. However, for the error data there was an interaction of group × target width [$F(5, 115) = 2.41$, $p < .05$]. Planned comparisons showed that while error rates decreased as target width increased similarly for both groups, the apraxic group made more errors than the nonapraxic group on movements to the three smallest target widths [$p < .05$] but not on movements to the larger targets [$p > .05$].

In summary, the apraxic patients appeared to have similar problems as the nonapraxics in making rapid, sequential movements to large targets. Although, the apraxic group's movements were somewhat less consistently reproduced, this was true regardless of target width. Most interestingly, only the apraxic patients showed impaired accuracy for movements to small targets.

Lesion Size and Location

CT scan measures are summarized in Tables 3 and 4. The left and right stroke groups did not differ in lesion volume or in linear or volumetric measures of lesion location [Mann–Whitney U tests; $p > .05$]. Therefore, the deficits seen with left but not right hemisphere damage cannot be

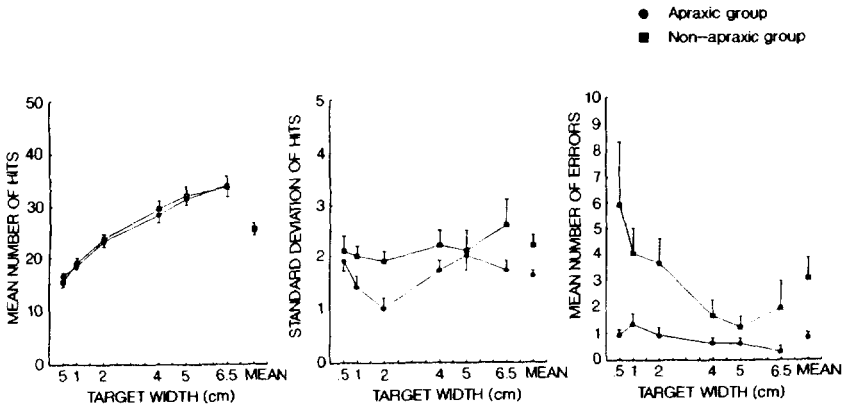

FIG. 3. Mean (standard error bars) number of hits, intertrial variability in hits, and errors for the left-hemisphere stroke apraxic and nonapraxic groups.

attributed to differences between the two stroke groups in lesion size or intrahemispheric lesion location. Table 4 tabulates lesion location as a function of lobe. This information is provided for descriptive purposes to further demonstrate the right and left hemisphere groups were generally comparable in lesion location.

Comparisons (Mann–Whitney U tests) between the apraxic and the nonapraxic left hemisphere groups showed that lesion volume was larger for the apraxic group [$p < .05$] and lesions were located more posteriorly

TABLE 3
Means (Standard Errors) for Lesion Volume and Location Measures

CT measures	Right stroke $N = 18$	Left stroke $N = 25$	Non-apraxic $N = 13$	Apraxic $N = 12$
Lesion volume[a]	4.0 (0.9)	3.6 (0.9)	1.7 (0.5)	5.8 (1.7)*
Anterior lesion volume[b]	65.2 (8.8)	59.4 (7.7)	67.9 (11.3)	50.1 (10.2)
Posterior lesion volume[b]	34.8 (8.8)	40.6 (7.7)	32.1 (11.3)	49.9 (10.2)
Anterior–posterior volume	30.4 (17.6)	18.8 (15.4)	35.9 (22.7)	0.2 (20.4)
Anterior distance[c]	32.6 (4.0)	35.2 (4.1)	33.0 (5.4)	37.7 (5.0)
Posterior distance[c]	36.8 (4.0)	36.2 (4.8)	43.1 (4.9)	28.8 (5.4)*

* $p < .05$ for ANOVAs comparing the nonapraxic and the apraxic groups.

[a] Percentage of total brain volume. Using mean brain volume of 1,237 cc^3 (Blinkov & Glezer, 1968), lesion volume is about 49 cc^3 and 45 cc^3 for the right- and left-hemisphere stroke groups, respectively.

[b] Percentage of infarct volume anterior or posterior to point halfway between the frontal and occipital pole.

[c] Percentage of total distance from frontal to occipital pole.

TABLE 4
Summary of Anatomical Lesion Location in Stroke Patients[a]

Areas of damage	Right stroke	Left stroke	Apraxic[b]	Nonapraxic[b]
Frontal	0	4	0	8
Parietal	6	4	0	8
Occipital	6	8	17	0
Basal ganglia/white matter	11	12	8	15
Frontal–temporal	6	0	0	0
Frontal–parietal	22	16	0	31
Temporal–parietal	6	16	25	8
Temporal–occipital	0	4	0	8
Parietal–occipital	0	8	8	8
Frontal–temporal–parietal	28	20	25	15
Temporal–parietal–occipital	11	8	17	0
Frontal–temporal–parietal–occipital	6	0	0	0

[a] The values in the table are the percentage of stroke patients within a particular group who had damage to an area(s). In some cases the column sum is greater than 100% due to rounding error.

[b] Apraxic and nonapraxic patients all had damage to the left hemisphere.

[$p < .05$], but the percentage of the total lesion volume that was posterior did not differ between the groups. Lesion volume also did not correlate with posterior distance indicating that the apraxic group's more posterior lesions were not larger in size. Table 4 supports the contention that the apraxic group's lesion were more posterior with 92% of the apraxic and 77% of the nonapraxic group demonstrating damage to the occipital and/or parietal area. Greatest differences were seen in occipital, temporal–parietal, and temporal–parietal–occipital groups.

Sequencing and Lesion Location

Partial correlations (which controlled for lesion volume) between the measures of lesion location and the mean hit rate (averaged across target width) showed that more posterior lesions in the left stroke group were associated with decreased speed [$r(15) = -.53$, $p < .01$ for percentage of lesion located posterior; $r(15) = .41$, $p < .05$ for posterior distance]. In the right stroke group, lesions that were more anterior were associated with slower performance but only when anterior distance [$r(22) = .49$, $p < .05$] and not percentage of lesion located anterior was the basis of the calculation suggesting this relationship was weaker. Measures of lesion location did not correlate with the mean error rates or the variability in hit rates. Further, regression analyses with target width as a repeated factor showed no interaction between the measures of lesion location and target width for any of the sequential tapping measures. This finding was true for both of the stroke groups.

DISCUSSION

Ipsilateral limb movements in patients with left hemisphere damage were slower relative to a normal control group, but only as target size increased. No deficits were found for patients with right hemisphere damage. When target size was varied from 0.5 to 6.5 cm the left hemisphere stroke group performed as rapidly as their control group at the smallest target, but for all other target widths this group performed more slowly, especially as target width increased. There also were no differences among the control and stroke groups in finger-tapping speed, a measure of maximum production speed at one location. Hence, these findings cannot be due to an inability of the left-hemisphere group to produce fast responses alone.

These results replicate and extend the findings from an earlier study (Haaland et al., 1987). In both studies tapping speed was slower in the left-hemisphere-damaged group but more so in the 4-cm than the 1-cm condition. Reducing the target size to .5 cm in the present study resulted in no deficits in the left- or right-hemisphere groups which suggests that when closed-loop processing requirements are increased, there is not a clear left- or right-hemisphere advantage for controlling movement. The present study also clarifies the previous work by excluding lesion size or intrahemispheric location as an explanation for the left-hemisphere deficits.

If the left hemisphere is more important for controlling open-loop, rapid movements, it might be predicted that in normals right-hand performance would surpass left-hand performance for these movements. This was not the case in this study, but hand differences were not examined in single subjects which would have been the most sensitive design. Furthermore, hand differences in normals are less likely to reflect hemispheric differences than studies with brain damaged patients because interhemispheric interactions are so pervasive. However, as noted in the introduction, previous studies in normals have found a right-hand advantage for closed-loop movements (Annett et al., 1979; Flowers, 1975; Peters & Durding, 1979; Todor & Cisneros, 1985) which is not supported by the normal control data or the comparison between the right- and left-hemisphere stroke groups in the present study. However, comparisons between apraxic and nonapraxic left stroke patients suggested the left hemisphere may play some role controlling sensory-dependent processing. In addition to performance deficits on open-loop movements, the apraxic group demonstrated deficits on closed-loop movements as evidenced by greater errors than the nonapraxic group on the smaller but not the larger targets.

Open- and Closed-Loop Processing

While the results appear consistent with a left-hemisphere specialization for open-loop processing, several issues concerning this theory re-

quire consideration. First, we are not suggesting movements to larger targets are dependent only upon central programming and entirely independent of sensory feedback. Although ballistic movements have been shown to be centrally programmed they can be influenced by sensory feedback (Angel, 1977; Capaday & Cooke, 1983; Cooke, 1980; Pelisson, Prablanc, Goodale, & Jeannerod, 1986). However, movements to larger targets should be more dependent upon motor programming and less dependent upon sensory feedback. This explanation is consistent with one report (Haaland & Harrington, 1989) of deficits with left- but not right-hemisphere damage in the initial, ballistic component of a simple aiming movement but not in the secondary, corrective component. In the present study this conclusion can be questioned because the duration of the movements even at the largest target (which ranged from 465 to 625 ms) is considered too long to be independent of sensory feedback. Work in young adults has shown that movement times less than 200 ms were independent of visual feedback (Wallace & Newell, 1983). If 200 ms is used to identify open loop movements in the present study, none would be identified. However, we have shown (Haaland & Harrington, 1989) in neurologically intact elderly individuals that movement times of 500 to 600 ms were not dependent upon visual feedback suggesting data from young normals cannot be used to infer a movement's dependence on sensory feedback in the normal elderly or in brain-damaged subjects. Despite these caveats, the movement times in the present experiment are so long that even in the elderly control groups they are not likely to be entirely independent of sensory feedback. Hence, the sensory independence explanation may not be the sole basis for our findings.

Frequency Hypothesis

Another explanation of these findings is that the left hemisphere is specialized for the perception of fine temporal or spatial discriminations (Efron 1963; Hammond, 1983; Kitterle, Christman, & Hellige, 1990; Nicholls & Cooper, 1991; Schwartz & Tallal, 1980; Sergent, 1982; Tzeng & Wang, 1984) and the production of rapid sequences which require timing and coordination of movements, such as in speech (Kent & Rosenbek, 1983; Stark & Tallal, 1979) or nonlinguistic timing tasks (Hammond, 1983). This apparent left-hemisphere specialization for the discrimination and production of fine temporal resolution may be a partial explanation of left-hemisphere dominance for both language and complex motor skills (Tzeng & Wang, 1984).

This hypothesis may explain our speed and intertrial variability data in that ipsilateral motor deficits after left hemisphere damage are more common when movements are faster and consequently more dependent upon the precise timing of the different components (e.g., acceleration and deceleration) of the reaching movement. Other task requirements (e.g.,

location change and manipulation of target size) may also be important as evidenced by the fact that the left hemisphere group demonstrated no significant deficits in ipsilateral rapid tapping at one location.

The intertrial variability data in the present experiment are more consistent with the frequency hypothesis. It predicts increasing variability with increasing target width in the control group because when speed is increased the various components of the movement must be coordinated in less time which is likely to produce greater variability (Darling, Cole, & Abbs, 1988). If processing rapid, sequential information is particularly inefficient in the left-hemisphere group, such that these patients do not significantly alter their response strategy as target size increases, no changes in variability would be expected. Post hoc analyses revealed that the mean hit rate and the intertrial variability in hit rate were positively correlated for all groups (Left Control: $r[17] = .58$, $p < .025$; Right Control: $r[16] = .88$, $p < .001$; Right Stroke: $r[17] = .67$, $p < .01$) except the left stroke group ($r[24] = .21$, $p > .05$). Hence, even though the left-hemisphere group showed some increase in speed as target width increased variability did not increase. In contrast, the open loop model predicts decreasing variability with increasing target size in the control group because the percentage of trials requiring secondary corrective movements should decrease (Soechting, 1984) and a smaller percentage of the total movement should be composed of the more variable corrective component (Todor & Cisneros, 1975). This was not the case in the control or the right stroke groups where variability increased with target size.

The present data, however, are not entirely consistent with the frequency hypothesis which also predicts that slow temporal processing is most efficiently done by the right hemisphere (Kitterle, Christman, & Hellige, 1990). In our study the right hemisphere group showed no evidence of deficits in the speed of a movement, even at smaller target widths which require much slower processing.

Differentiating the Open–Closed Loop and Frequency Hypotheses

The open-closed loop and the frequency theories of hemispheric function emerged from independent literatures so it is not clear if separate mechanisms underlie the processes proposed by the theories. Present conceptualizations of the two are not clearly independent because rapid, sequential movements are also more likely to be sensory-independent. While these two theories appear to differ in terms of the mechanisms supporting response variability, the impulse variability model (Schmidt, Zelaznik, Hawkins, Franks, & Quinn, 1979) would be consistent with modified versions of both. This model suggests faster, more open-loop responses are associated with greater force which produces greater vari-

ability in speed due to the increased variability of neural output. Although this theory typically has been applied to movements of less than 200-ms duration, it may extend to longer movement times, especially in the elderly, to provide a more general account of response variability.

Sequencing and Limb Apraxia

Only error rates dissociated the performance of the apraxic and nonapraxic groups. Therefore, it cannot be assumed that the speed and variability differences seen after left-hemisphere damage are due entirely to the inclusion of limb apraxics. These findings are consistent with a recent study showing some aspects of sequencing hand postures were equally disrupted in apraxic and nonapraxic left-hemisphere-damaged patients whereas other aspects of sequencing were impaired only in the apraxic group (Harrington & Haaland, 1992). Limb apraxia is only one manifestation of complex motor deficits associated with left-hemisphere damage. Thus, in addition to having problems with rapid sequential movements to large targets, the apraxics demonstrated an additional deficit in accurately hitting the small targets. This suggests limb apraxia also disrupts the production of slow movements which are likely to be more sensory-dependent, consistent with the finding that left-hemisphere apraxic but not nonapraxic patients demonstrated deficits steadily holding a stylus in a small but not a larger target (Haaland et al., 1980).

Sequencing and Lesion Location

The relationship of the motor sequencing data to lesion location was not striking, possibly due to the method for specifying location which was quantitative and not directly related to specific cortical areas. However, within the left hemisphere group there was a relationship between slower performance and posterior lesions, suggesting a possible role of the occipital and parietal lobes in some aspects of sequencing. Previous work (Harrington & Haaland, 1991) has also shown that more posterior lesions are associated with diminished speed of sequencing different hand positions. This association in the left- but not right-hemisphere group cannot be attributed to a higher incidence of posterior involvement in the left-hemisphere group using quantitative or qualitative lesion location measures. However, due to the minimal number of focal lesions in this sample it was not possible to examine the role of different parts of the occipital and parietal lobes.

Monkey data also point to the importance of the inferior parietal area in controlling several aspects of movement in the contralateral limb (Lynch, 1980; Mishkin, Ungerleider, & Macko, 1983; Mountcastle, Lynch, Georgopoulos, Sakata, & Acuna, 1975). One study in monkeys (Hartje &

Ettlinger, 1973) examined reaching in the arm ipsilateral to lesion after unilateral parietal lesions in either hemisphere. They found reaching deficits if the movement was performed without visual feedback but not with visual feedback which suggests the parietal lobe may be particularly important for regulating open-loop or faster movements in the limb ipsilateral to parietal lesion even in monkeys.

Summary Remarks

While a variety of motor tasks are impaired after left-hemisphere damage (see Haaland and Yeo, 1989 for review; Wyke, 1967, 1968, 1971; Kimura, 1977, 1982; Kimura and Archibald, 1974; Haaland and Harrington, 1989], the present study demonstrated the degree of left-hemisphere control varied within a single task as a function of changes in target size. It was not possible, however, to determine if the open–closed loop or frequency hypotheses were more explanatory of our data. The speed and the variability data suggest the open–closed loop model is not fully adequate, but the absence of deficits in the small target condition after right hemisphere damage suggests the frequency hypothesis cannot entirely account for the findings. Future research is needed to differentiate these models especially as they relate to hemispheric asymmetry of complex motor skills. In addition, these findings suggest comparisons of limb apraxic and nonapraxic patients on behavioral and neuroanatomical measures should be promising in terms of clarifying the reasons for limb apraxia and elucidating cognitive–motor processes of the left hemisphere that are not explicitly tapped by clinical tests of limb apraxia.

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