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The effects of unilateral brain damage on visually guided reaching: hemispheric differences in the nature of the deficit

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Summary. Groups of patients suffering from unilateral damage to the left or right cerebral hemisphere were compared to a group of age-matched normal controls in a visually guided pointing task. Subjects were required to reach quickly and accurately to small visual targets as soon as they appeared on the screen in front of them. All reaches, which were quite unrestricted, were videotaped by rotary-shutter cameras and analyzed by a computer-assisted system which allowed analysis of the kinematic parameters of the movement in three-dimensional space. The groups were compared on the basis of their latency to initiate a reaching movement, the accuracy with which they achieved the target's position, and various measures derived from the instantaneous velocity of the movement. Both patient groups were found to be less accurate than controls and to require more time after the target was illuminated to complete the reach. But while the right-hemisphere group took longer to initiate a reach, the kinematic parameters of the movements they produced did not differ from those of the control group. In contrast, the left-hemisphere group did not differ from the control group in the time required to initiate a reaching movement but did require a greater period of time to execute the reach once it had been initiated. It is suggested that the right hemisphere group were deficient in the speed with which they could determine the spatial position of the target, while the left hemisphere group were deficient in their ability to select an appropriate motor program to achieve the target position and/or to monitor the movement and update the motor program as it was being executed.

Key words: Human movement – Visually guided reaching – Unilateral brain damage

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Introduction

Abnormalities in the production of visually guided movements of the upper limbs are characteristic of a number of patients suffering from damage to the cerebral hemispheres. Indeed, elegant descriptions of these behavioural disturbances have been available since the early part of this century (Balint 1909; Holmes 1918). Although these very early descriptions were limited to patients who had sustained bilateral damage to the cerebral hemispheres, it became evident later that unilateral damage to either the left or the right cerebral hemisphere could also result in inaccurate reaching movements (Riddoch 1935; Brain 1941). Since then, numerous cases have been described in which bilateral or unilateral damage has resulted in deficits in visually guided movements of the upper limbs (see for example: Hecaen and de Ajuriaguerra 1954; Rondot et al. 1977; Russell 1977). While the clearest clinical evidence of disturbed visually guided reaching is typically seen in the limb or visual field contralateral to the side of the unilateral damage (Levine et al. 1978), evidence of more subtle disturbances, which may be less readily recognized, has also been presented (Ratcliff and Davies-Jones 1972). One of the major obstacles to developing a satisfactory account of these deficits has been the lack of adequate measures of performance in neurological patients (but see Perenin and Vighetto 1983).

Investigations of similar disturbances in monkeys in which unilateral lesions have been made in parietal cortex or supplementary motor and/or premotor cortex have generally found that the deficits in visually guided reaching are restricted to the contralateral limb (Haaxma and Kuypers 1974; LaMotte and Acuna 1978; Moll and Kuypers 1977). It should be noted, however, that small but measurable deficits in the ipsilateral limb have occasionally been found

after unilateral parietal lesions (see for example: Hartje and Ettlinger 1973). More recent single-unit studies of parietal cortex (Kalaska et al. 1983) and premotor cortex (Weinrich et al. 1984) in monkeys, while providing a good deal of information about the role of these regions in motor control, have limited their recordings to the cortical hemisphere contralateral to the reaching limb. Single-unit activity in the ipsilateral hemisphere during visually guided reaching movements has rarely been studied despite the fact that there are extensive interhemispheric connections between parietal and premotor cortex. In humans, "preparatory" cortical activity can be recorded as a Bereitschaftspotential or "readiness potential" over both hemispheres prior to even simple voluntary movements, although the pattern of activity becomes more lateralized to the contralateral hemisphere as the movement is executed (for a review of this literature, see Deecke et al. 1984). This latter finding would suggest that the production of voluntary movements in humans involves, at least initially, mechanisms within both hemispheres.

Even though both hemispheres may play a role in the programming of a unimanual movement, one must also consider the possibility that there may be important differences in the way each hemisphere contributes to that programming. Certainly in humans there is an overwhelming body of evidence for hemispheric specialization in motor control. Hand preference and speech production are perhaps the clearest and most obvious examples of this lateralized control, but more subtle examples may be found in other aspects of human motor behaviour. It is commonly found, for example, that patients suffering from damage to the left hemisphere, in addition to having problems with speech, are often unable to produce appropriate movements of their limbs to imitation or to verbal command. This syndrome has been termed manual apraxia (Liepmann 1908) and refers to the patient's inability to produce an appropriate sequence of changes in limb posture despite adequate sensation and strength. As noted by Liepmann, this dysfunction of motor control is not limited to movements of the contralateral hand but is evident in both the left and right limbs of apraxic patients. Clinical tests for apraxia typically require the patient to perform a common behaviour (such as demonstrating the use of a toothbrush) to either command or imitation. Although deficits on this type of task have been termed "ideomotor" apraxia (see for example Kertesz and Ferro 1984), the same sort of deficit can also be seen in the production of movements which have no inherent meaning to the patient (Kimura and Archibald 1974). It has been suggested that apraxia represents a generalized deficiency in the selection or programming of appropriate sequences of motor acts (Kimura 1982) and may reflect disturbances in the process of motor learning (Geschwind 1975). Following Liepmann (1908), Kimura (1982) has argued persuasively that the mechanisms responsible for this higher-order motor programming are most often lateralized to the left hemisphere. Unfortunately, this area of research, like the work on deficits in visually guided reaching in neurological patients, is characterized by a lack of detailed measurement of the movements produced by apraxic patients.

In recent years, however, advances in the technology of kinematic recording have made it possible to conduct quite detailed studies of limb movements in normal human subjects (Atkeson and Hollerbach 1985; Soechting and Laquaniti 1981). Investigators using these techniques have generally focussed their efforts on defining the invariant characteristics of the limb movements that are produced rather than attempting to determine whether the variability in the movements could be a reflection of hemispheric specialization for this motor behaviour. We addressed this latter issue in an earlier study of visually guided reaching in normal subjects (Fisk and Goodale 1985) and found that certain characteristics of their performance appeared to reflect the function of a lateralized control system. Indeed, the differences in performance that we saw between the left and right hands and between trials in which the targets were located in the left and right visual fields all suggested that the left hemisphere has a special role to play in the control of pointing movements directed at small visual targets. In other words, the programming of this apparently 'simple' behaviour, like that of more complex behaviour such as speech, appeared to depend heavily on mechanisms within the left hemisphere. The present study was an attempt to examine whether the same kind of fine-grain analysis of movement when applied to a neurologically impaired population, would reveal further evidence of hemispheric specialization for the control of visually guided pointing movements.

Although we set out to compare the pointing movements made by patients with damage restricted to the left or right hemisphere, we limited our comparisons to the limb ipsilateral to the lesion. It is well known that unilateral cerebral damage often results in sensory loss or weakness in the contralateral limb. We were interested, however, in examining the generalized effects of unilateral cerebral dysfunction (such as those observed in manually apraxic patients) on the execution of visually guided pointing movements made with either limb rather than the effects of sensory or motor disabilities

restricted to a single limb. Thus, by limiting our measurements to the ipsilateral limb, we avoided the problems of interpreting any hemiparesis or sensory deficit that might have been present in the contralateral limb. Since we had already shown that were differences in the performance of normal subjects with their dominant and nondominant hands (Fisk and Goodale 1985), it was necessary to establish an appropriate comparison group for the left- and right-hemisphere damaged patients. This meant that reaches with the left hand by patients with left-hemisphere damage had to be compared to reaches with the left hand in age-matched control subjects, and reaches with the right hand by patients with right-hemisphere damage had to be compared to reaches with the right hand in the same control subjects.

Table 1. Summary of clinical data for patients

	Left hemisphere	Right hemisphere	Control
N	17	11	13
Age (yr)	49.1 (24–68)	42.2 (20–66)	50.6 (19–69)
Sex M	12	5	7
Sex F	5	6	6
Etiology vascular	14	6	–
Etiology tumour	2	4	–
Etiology other	1	1	–
Hemiplegia	6	8	–
Visual hemifield deficit	4	1	–
Hemi spatial neglect	1	5	–
Aphasia	3	0	–
Verbal IQ	103.1 (73–132)[a]	97.2 (86–110)	–
Performance IQ	100.5 (79–120)	90.5 (56–105)	–

[a] Aphasic subject scores not included in these data

Methods

Subjects

The patients who served as subjects for this study were consecutive admissions to the Clinical Neurological Sciences division of University Hospital in London in whom a diagnosis of unilateral brain damage was made. All patients were evaluated by neurological examination and CT scan. The presence of visual field defects was established by confrontation testing during the neurological examination. Additional information from angiography, EEG, and surgical records contributed to the diagnosis of some patients. All patients underwent a neuropsychological examination, although this information was not used for the purposes of establishing the laterality of brain damage. A handedness questionnaire, administered during the neuropsychological examination (Kimura 1986) revealed that all patients were right-handed. The possible presence of hemispatial neglect was also evaluated during the neuropsychological examination with the use of a visual search test (Kimura 1986). Informed consent was obtained from all subjects before they participated in the study.

The left-hemisphere damaged group included a total of 17 cases. In 14 of these, the etiology was of vascular origin (12 infarct, 2 AVM). In 12 of these cases, the area of damage was visualized on CT scan. One of the remaining cases had evidence on angiography of a narrowing of the left internal carotid artery. Two cases of tumor were also included in the left-hemisphere group (1 metastatic carcinoma, 1 oligodendroglioma). The tumors in these cases were visualized on CT scan and confirmed by surgical removal after the subjects had participated in the study. The final left-hemisphere case was one of cortical atrophy accompanied by seizures. This was visualized by CT scan and confirmed by EEG.

The right-hemisphere damaged group included a total of 11 cases. In 6 cases, the etiology was of vascular origin (5 infarct, 1 AVM). The area of damage was visualized by CT scan in 4 of these cases. Four cases of tumor were also included in this group (2 astrocytoma, 1 oligodendroglioma, 1 probable glioma). In all four cases, the tumors were visualized by CT scan with 2 cases confirmed by later surgical removal. The final right-hemisphere case was one of leukemic infiltration of the brain which was visualized by CT scan and confirmed by brain biopsy following the patient's participation in the study.

Thirteen right-handed, age-matched volunteers were recruited as control subjects. This group included relatives of the patients as well as local community members who were contacted through a newspaper advertisement. None of the control subjects reported a history of neurological disease and none were taking medications at the time of testing. The relevant subject information is summarized in Table 1.

Apparatus for data collection

The subject was seated facing a 31 cm high by 90 cm long screen that contained a horizontal array of target lights. The position of the subject's head was maintained by a chin and head rest with a distance of 50 cm between the subject's eyes and the screen. For each subject, the position of the screen was centred with respect to their eye level. The screen was covered with black cloth which eliminated any view of the unilluminated targets. A 1° diameter white fixation point was situated directly in front of the subject, at eye level. Target lights, 0.25° in diameter, were presented at four positions, 10° and 20° to either side of the fixation point in the horizontal plane. Once illuminated, they remained visible until the subject completed the reach. The luminance of the screen was 2 cd/m^2 while the luminance of the fixation point and the targets was 69 cd/m^2 and 91 cd/m^2 respectively. A start platform for the subject's hand was situated at the base of the chin rest such that the hand was always within the field of view of the subjects without visual field defects. All reaches were initiated from this platform although the actual position of the hand and fingers was allowed to vary slightly across trials.

All reaches were videotaped using two rotary shutter cameras (Sony RSC 1010) which provided clear images at 60 frames/s. One camera viewed the subject from the side, while the second camera, situated above the subject, provided a top view of reach. The two camera signals were synchronized and fed to a split screen of a single video frame. Onset of the target was indicated on the videotape by the illumination of a separate target light which was outside of the subject's field of view. A video counter/timer (TEL Video Products) recorded the elapsed time of the test session on each video frame with a resolution of 10 ms.

Procedure

For the control subjects, each hand was tested separately in four alternating blocks of 16 trials. Testing of the patients was

conducted in two separate blocks with the hand ipsilateral to the side of brain damage only. The subject was asked to use his index finger to point quickly and accurately, immediately following illumination of the target, to the position on the screen where the target was presented. All trials began with the subject fixating the central fixation point. After a variable interval following a "ready" command, the experimenter illuminated one of the targets. The position of the target was varied randomly throughout the block of trials. Practice trials were provided at the start of each block to familiarize the subject with the test conditions.

For half of the blocks (one block for the patients, two for the control subjects, one with each hand), the subject was instructed to look to the target as well as reach to it. For the other half of blocked trials, he was instructed to maintain fixation on the central fixation point while reaching. During the latter blocks of trials the subject's fixation on the central position was monitored by the experimenter who was seated behind the screen facing the subject. From this position, the eye position of the subject could be easily observed. Any trials in which the subject failed to maintain fixation were repeated at the end of the block. Patients who did not respond to targets in one visual hemifield due to a visual field defect or hemispatial neglect were presented with targets in their good field only. While hemispatial neglect was identified in five patients with right-hemisphere lesions and one patient with a left-hemisphere lesion, only two patients failed to respond to targets presented in the visual field contralateral to the side of pathology due to hemispatial neglect. The patient with a left-hemisphere lesion failed to respond to the most peripheral contralateral target while maintaining central fixation and one patient with a right-hemisphere lesion failed to respond to either contralateral target while maintaining central fixation. Thus, the presence of hemispatial neglect, as defined by neuropsychological examination, had a relatively minor effect on the ability of patients to respond to the visual targets in the study. The test session lasted approximately 60 min and the subject was given rest periods between blocks if fatigued.

Apparatus for data analysis

The method of analysis of the videotape records has been described in greater detail previously (Fisk and Goodale 1985). A computer-assisted digitizing system was used to extract the position of the tip of the index finger, in three-dimensional space, from each video frame of the subject's reach. Measures of latency and accuracy, as well as kinematic measures of the reach, were calculated from this information. Latency was recorded as the time of the first visible movement of the index finger following illumination of the target light. The kinematic measures included the maximum velocity attained, the duration of the acceleration phase of the movement, the mean velocity of the movement, and the duration of the movement. Accuracy of the movement was calculated in cm as a signed error score (positive = overshoot, negative = undershoot) which indicated the horizontal distance between the known target position and the position of the subject's finger as it contacted the screen. All measures of performance were collected for all reaches performed by the subject.

Results

For the reasons outlined in the Introduction, we compared the reaches made by patients with right-hemisphere damage, all of which were made with the right hand, to the reaches made by the control subjects with their right hands. Similarly, we compared the left-handed reaches made by the patients with left-hemisphere damage with the left-handed reaches made by the same control subjects. The latency, accuracy, and kinematic measures of the different reaches were all compared by analyses of variance.

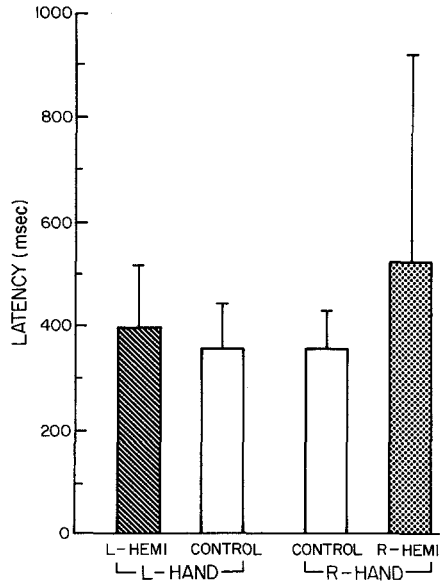

Fig. 1. Mean latency of movement onset for the reaching movement in the patient groups and control subjects. Left-handed reaches by the patients with lesions of the left hemisphere (cross-hatched bar) are compared with left-handed reaches by the control subjects (open bar). Right-handed reaches by the patients with lesions of the right hemisphere (dotted bar) are compared with right-handed reaches by the control subjects (open bar). Standard deviations are indicated by the lines on top of the bars

Relative to the control subjects, the two patient groups showed large differences in the execution of this relatively simple visually guided behaviour. Both groups of patients took much longer than control subjects to attain the final position of their movement following onset of the target light. The average time from target onset to end of reach for the left-hemisphere patients (using their left hand) was 1100 ms and for the right-hemisphere patients (using their right hand) was 1104 ms. While these two values were virtually identical, they differed considerably from the average times for the control group (left hand, 925 ms; right hand 883 ms). As Figs. 1 and 2 indicate, however, the reasons for the increased times were quite different in the two patient groups. The right-hemisphere group (X = 516 ms) demonstrated a greater latency to initiate a reaching movement following onset of the target than the controls (X = 358 ms). Despite a large amount of intersubject variability on this measure, this

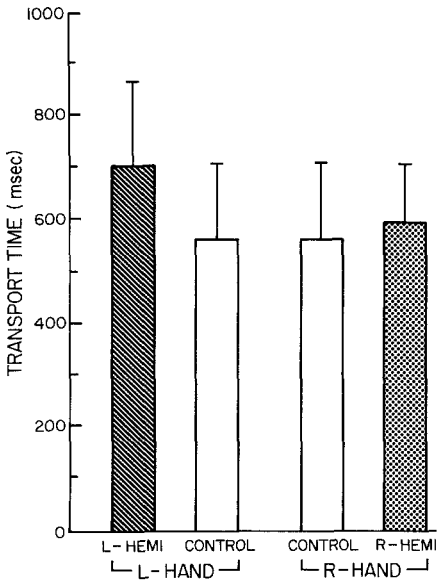

Fig. 2. Mean transport time for the reaching movement in the patient groups and control subjects. Left-handed reaches by the patients with lesions of the left hemisphere (cross-hatched bar) are compared with left-handed reaches by the control subjects (open bar). Right-handed reaches by the patients with lesions of the right hemisphere (dotted bar) are compared with right-handed reaches by the control subjects (open bar). Standard deviations are indicated by the lines on top of the bars

latency difference was evident in an analysis of variance as a trend which approached statistical significance ($F(1,22) = 3.85$, $p = 0.06$). No such difference was evident between the left hemisphere group and the controls (see Fig. 1). Moreover, the prolonged latency to initiate a reaching movement that we observed in the right-hemisphere patients was not limited to targets presented to the contralateral visual field since the latencies for both ipsilateral and contralateral targets were higher in this patient group and no significant group by visual field interaction was found ($F(1,22) = 1.59$, $p > 0.2$).

An examination of the time required to execute the movement after it had been initiated revealed a very different pattern of results from the measures of movement latency. As Fig. 2 illustrates, the left-hemisphere group took nearly 150 ms longer than the control group to transport their limb from its initial to its final position ($F(1,28) = 8.9$, $p < 0.005$). This large increase in movement duration was not present in the right-hemisphere group who did not differ significantly from the control group on this measure. Thus, although both brain damaged patient groups demonstrated an impairment in their ability to produce rapid visually-guided movements, the nature of that impairment was dependent on the laterality of the brain damage. While the patients with right-hemisphere damage took longer than control subjects to initiate a pointing movement, once limb movement began they were no slower than the control subjects in reaching the target. In sharp contrast, the patients with left hemisphere lesions, while they were able to initiate a pointing movement as quickly as control subjects, took much longer to reach the target.

Despite these clear impairments in the patients' performance, they still showed many of the characteristic effects of target location on movement kinematics that we had observed in an earlier study of normal young adults (Fisk and Goodale 1985). Thus, reaches to ipsilateral targets (i.e., targets ipsilateral to the hand being used) attained a higher maximum velocity and were completed in a shorter period of time than contralateral reaches ($F > 37.0$, $p < 0.001$ for all comparisons). In addition, the maximum velocity of reaches to the further of the two ipsilateral targets was greater than that of reaches to the nearer one ($F > 21.0$, $p < 0.001$ for all comparisons). Just as we had observed in the earlier study (Fisk and Goodale 1985), the scaling of movement velocity for changes in movement amplitude, while clearly present in ipsilateral reaches, was not at all evident in the reaches to contralateral targets. One final similarity between the performances of all three groups in the present study and the data we obtained earlier from the young adults was the skewed velocity profile of the pointing movement in which the acceleration phase was quite a bit shorter than the deceleration phase. These findings are shown in Fig. 3 which illustrates the averaged velocity profiles of all reaches to the four target positions for the two patient groups and the control groups. Although a small increase in movement duration for trials in which the subject was allowed to look at the target had been noted in the previous study, this was not apparent in the present study. None of the kinematic measures differed significantly between these conditions.

Figure 3 also shows the differential effect of left- versus right-hemisphere damage on a number of kinematic parameters. The lack of a significant difference between the right-hemisphere group and the control group is readily apparent in this figure and was confirmed by statistical analyses ($F < 1.0$ for all kinematic measures). Thus, when compared to the control subjects, the right-hemisphere group required a similar period of time to transport the limb from initial to final position, achieved the same peak velocity, and demonstrated a similar temporal pattern of acceleration/deceleration. A very different pattern was evident from the comparison of the left-hemisphere group and the control group. Reaches by

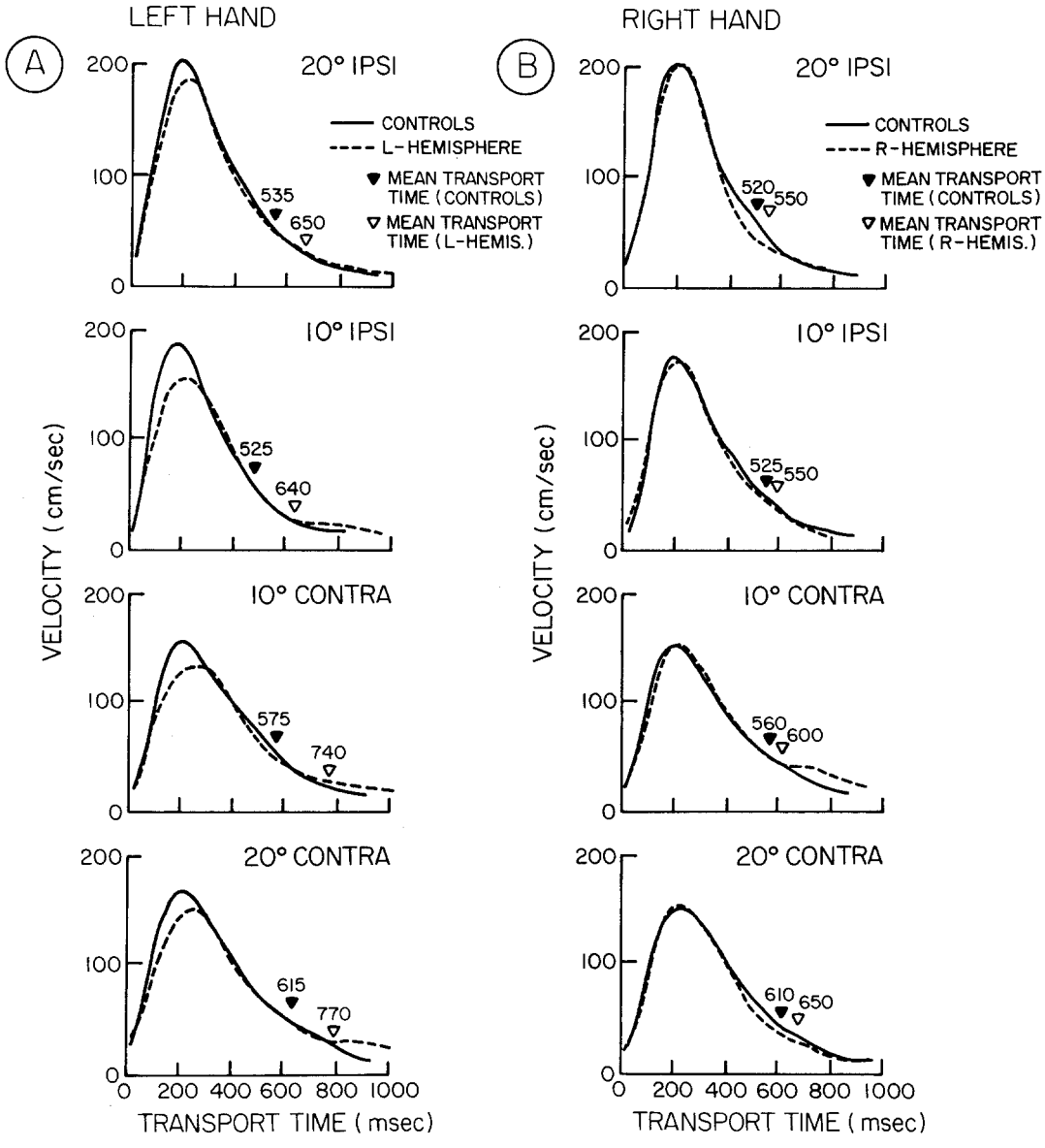

Fig. 3A, B. Average velocity profiles for reaches made by patients and control subjects to targets ipsilateral and contralateral to the arm being used. **A** Left-handed reaches made by patients with lesions of the left-hemisphere (dashed lines) are compared with left-handed reaches made by control subjects (solid lines). Mean transport times in each case are indicated by inverted triangles. **B** Right-handed reaches made by patients with lesions of the right-hemisphere (dashed lines) are compared with right handed reaches made by the control subjects (solid lines). Mean transport times in each case are indicated by inverted triangles

the left-hemisphere group tended to attain a lower maximum velocity ($F(1,28) = 3.6, p < 0.07$) but since all groups produced lowered maximum velocity for reaches to contralateral targets the difference between the left-hemisphere group and the control group was most evident for ipsilaterally directed reaches (Group X Target Laterality interaction, $F(1,28) = 7.3$, $p < 0.05$). In addition to a lower maximum velocity, the left-hemisphere group also demonstrated a prolonged period of low velocity movement at the end of the reach which is evident in the velocity profiles of Fig. 3. These two factors resulted in the greatly increased duration of reaches by the left-hemisphere subjects noted above, as well as a significantly lowered mean velocity of movement ($F(1,28) = 8.9$, $p < 0.005$). Analysis of the time taken to accelerate to maximum velocity revealed, however, that the duration of the acceleration phase of the movement did not differ between the left-hemisphere group and the control group ($F < 1.0$), even though the peak of the averaged velocity appears to be shifted to the right (Fig. 3). However,

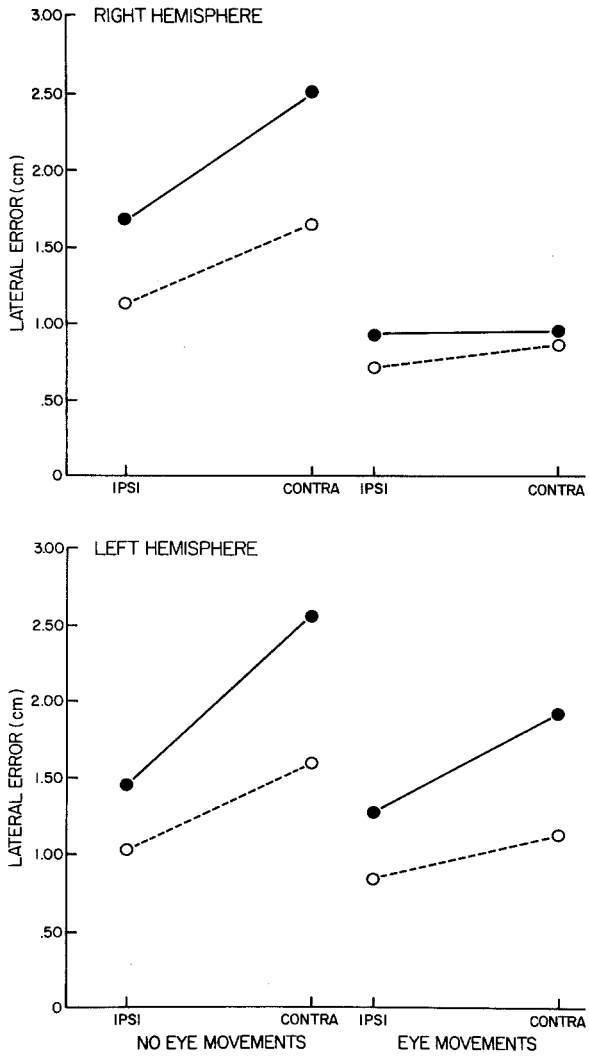

Fig. 4. Mean lateral error (cm) of reaches made by patients and control subjects. The top graph compares right-handed reaches made by the patients with lesions of the right-hemisphere (filled circles, solid lines) with right-handed reaches made by the control subjects (open circles, dashed lines). The bottom graph compares left-handed reaches made by the patients with left-hemisphere lesions (filled circles, solid lines) with left-handed reaches made by the control subjects (open circles, dashed lines). Errors in the no eye movement condition are compared to errors made when eye movements were permitted. Errors for reaches made to contralateral targets are also compared to errors for reaches made to ipsilateral targets

for the left-hemisphere patients a greater proportion of the distance of the reach was completed at a lower velocity and during deceleration.

The effects of targets laterality, eccentricity, and foveation that had been reported previously (Fisk and Goodale 1985) were noted again in the performance of the control subjects. Thus, the poorest accuracy was found for reaches to the most eccentric, contralateral targets while significantly greater accuracy was found when the subjects were allowed to move their eyes and foveate the target ($F > 16.0$, $p < 0.001$ for all comparisons). Both patient groups were less accurate than the control group ($F > 4.3$, $p < 0.05$ for both comparisons). These results are summarized in Fig. 4.

Discussion

Before discussing the differences in the performance of the two patient groups on the visually guided pointing task, it is important to emphasize that there were striking similarities in performance not only between the two patient groups and the control subjects, but also between all three of these groups and the normal young adults we tested in an earlier experiment (Fisk and Goodale 1985). Thus, while the patients showed specific deficits, the general pattern of their performance was not affected by the brain damage. Just as we had found in our earlier work with normal young adults (Fisk and Goodale 1985), reaches to targets ipsilateral to the hand being used reached a higher peak velocity than reaches to targets on the opposite side of the body. Moreover, increases in peak velocity as a function of target eccentricity were observed only for these ipsilateral targets. It must be remembered, however, that all patients suffered from restricted unilateral pathology and were tested only with the limb ipsilateral to the side of brain damage. The basic sensory and motor functions of that limb were quite intact as was the cerebral hemisphere contralateral to that limb. Georgopoulos et al. (1982) have reported that some cells in the motor cortex of the monkey vary their discharge with the direction of visually guided movements of the contralateral arm in such a way that a "tuning curve" for preferred directions of movement are apparent. The frequency distribution of preferred directions which Georgopoulos et al. (1982) presented implies a general preference on the part of the cell population for movements directed into ipsilateral space. This suggests that some of the laterality differences we observed in normal human reaching which are maintained after damage to the ipsilateral cerebral hemisphere may represent the influence of a directional preference on the part of the neural systems of the contralateral cerebral hemisphere which are involved in the programming and/or execution of the reaching movements.

One of the unique contributions of the present study was the application of systematic kinematic recording techniques to the study of neurological patients. Moreover, the results we obtained underline the importance of having appropriate compari-

son groups when examining the effects of brain dysfunction in patient populations. For example, the right-hemisphere group demonstrated a longer latency and movement duration, as well as reduced accuracy, when reaching for targets in their left as opposed to their right visual hemifield. These differences do not reflect the effects of lateralized brain damage, however, since these same differences in latency, movement duration, and accuracy are demonstrated by normal subjects when they reach to targets on the side of the body contralateral to the hand used for reaching. A simple within-subjects comparison of reaches by a single patient group could have resulted in some very misleading conclusions. Although both patient groups in the present study were less accurate than the control subjects, this difference was not limited to any one combination of lateralized brain damage and visual hemifield. Findings such as these point out the need to be cautious when interpreting results such as inaccurate reaching into the "impaired" visual field (i.e., the field contralateral to the side of damage) with the hand ipsilateral to unilateral brain damage (see for example: Levine et al. 1978). Such inaccuracies could reflect nothing more than a generalized reduction in accuracy due to nonspecific brain damage combined with the decreased accuracy which occurs in normal subjects when they reach across the body axis.

While damage to either the left or right hemisphere resulted in clear deficits in visually guided pointing, there were important differences between the two patient groups relative to the control subjects. Patients with right hemisphere damage required a longer time to initiate a movement, but produced reaches whose kinematics were indistinguishable from the control subjects'. In sharp contrast, patients with damage to the left hemisphere, while they took no longer than control subjects to initiate a pointing movement, produced reaches of much longer duration. Indeed, while all three groups of subjects showed skewed velocity profiles with prolonged deceleration, the extent to which the profile was skewed was much greater for the left-hemisphere patients. The long tail on the velocity profile was a characteristic of visually guided pointing movements that we had observed in our earlier study of normal young adults (Fisk and Goodale 1985) and undoubtedly reflects the demand for relatively accurate reaches. An elegant description of the effects of varying target size on the kinematics of accurate unrestricted pointing movements in normal subjects has been presented by Soechting (1984) who also noted prolonged periods of low velocity movement at the end of reaches to small visual targets. Presumably, it is during this period of time that modifications of the movement trajectory can serve to improve the final accuracy. For this reason, it is important to note that it was in this late phase of the movement that the deficit was most apparent in the left hemisphere patients.

A number of explanations, none of which are mutually exclusive, can be put forward to explain the differences between the performance of the right- and left-hemisphere damaged patients on this task. With respect to the increase in movement latency in the patients with right-hemisphere damage, Heilman et al. (1985) have noted similar deficits in right-hemisphere damaged patients with contralateral neglect as compared to groups of patients with left-hemisphere damage and control subjects. Unlike the present study, Heilman et al. (1985) used a simple reaction time task in which the subjects were required to displace a handle in a predetermined direction immediately following the onset of an auditory stimulus. They interpreted the finding of an increased latency for the right hemisphere group as reflecting the dominance of this hemisphere in what they termed "motor activation or intention". The idea that the right hemisphere plays a major role in preparing a motor response is certainly not inconsistent with the findings of the present study. Nevertheless, many of Heilman et al.'s (1985) other results, such as the effect of the starting position and the direction of the movement, were not found in the current study and may reflect differences in the experimental paradigm as well as the patient selection criteria. In the present study, patients were included on the basis of unilateral hemispheric pathology alone and not on the basis of the presence of a specific clinical sign such as contralateral neglect.

Another plausible explanation for the right-hemisphere deficits in the present study is that these patients had difficulty determining the position of the target in extrapersonal space and thus required a greater period of time in which to access the neural systems responsible for programming a movement to that position. Once this program had been accessed, however, these patients were able to execute it in the same manner as normal subjects. The lack of any significant differences in the kinematic measures between the right-hemisphere group and the control group suggests that they were equally proficient in their ability to update and modify the movement trajectory as the target was approached. Thus, the slightly increased error in final position of the right hemisphere group would reflect errors in their localization of the target position in space.

In contrast to the right-hemisphere group, the patients with left-hemisphere damage did not differ significantly from the controls in the time required to

initiate the pointing movement, and yet they produced reaches which required a greater period of time to complete the movement and differed considerably in their kinematic characteristics. Not only did the left-hemisphere patients execute their reaches at a much lower velocity than controls, they were also less accurate in their localization of the target. These findings suggest that although the left-hemisphere patients were able to localize the position of the target in extrapersonal space, they were less able to make use of the visual, proprioceptive, and efference-copy information that was available as the movement was executed to update and modify the trajectory of the reach.

In a study of visually directed limb movements by monkeys, Georgopoulos et al. (1982) have shown that an evolving motor program can be modified by changes in the visual afferent information of the location of the target. Furthermore, this modification of the motor program is reflected in a modulation of motor cortex cell discharge. Their paradigm involved large shifts in the target position which required a change in the direction of the movement although it seems clear that more subtle changes in limb trajectory, based on visual afferent information, can take place during reaches to stable targets. The evidence suggests that it is during the period of relatively low velocity, in the latter part of the movement trajectory, that modifications in the trajectory most likely occur. Beaubaton and Hay (1986) have demonstrated that while feedback about the position of the moving hand that was limited to the initial phase of the trajectory did little to improve the accuracy of rapid pointing movements, feedback limited to the terminal phase of the movement resulted in a level of accurracy equal to conditions of complete feedback. Thus, it is apparent that even for the rapid movements which they studied (less than 270 ms duration), information processed during the later stages of the movement can be used to improve its accuracy. Although the nature of the information on which such improvements are based remains controversial (see for example Mather and Fisk 1985; Prablanc et al. 1986), the time required to update or modify a previously selected and, in most instances already implemented, motor program necessitates that these modifications take place in the terminal stages of the movement.

Goodale et al. (1986) have demonstrated that updating of the limb position occurs throughout the execution of a visually directed pointing movement without the subject necessarily being conscious of producing any corrections in the trajectory. They also demonstrated that this updating can be based solely on visual information of the position of the target without requiring vision of the moving limb. This suggests that for normally executed limb movements, corrections of the limb's trajectory are not likely to be based on the visual comparison of the relative positions of the limb and a target. Rather, as Goodale et al. (1986) suggest, the comparison of limb and target position is likely to be based on an internal representation of external space which can utilize visual information to determine the target position and proprioceptive and/or program efference information to determine the instantaneous position of the limb. Cooke and Diggles (1984) have demonstrated that very rapid corrections in movement trajectory can be made by normal subjects in the performance of a visual tracking task. They note that the rapidity with which these corrections occur suggest that they "cannot be based on information from the moving limb... (but are) based on central monitoring of the commands for movement" (page 348). The prolonged period of low velocity movement and lowered accuracy of the left-hemisphere group in the present study suggests that they may have been unable to provide efficient monitoring of these central commands and thus were unable to update the program adequately as the trajectory of the movement unfolded. Another possibility which must be considered, however, is that the left-hemisphere damaged patients may also have been deficient in their ability to utilize proprioceptive information to determine the instantaneous position of the moving limb. This would result in a greater dependence on the analysis of visual information to achieve an accurate end-point of the movement which, in turn, could result in an increase in the time needed to modify the trajectory as well as a decrease in the accuracy with which the movement was completed. It would be useful to compare the performance of left- and right-hemisphere damaged patients with and without vision of the moving limb since this might yield additional information about the use of visual feedback by the two patient groups.

Although the patient groups were selected on the basis of lateralized cortical pathology, it would be an oversimplification to suggest that the complex integration of proprioceptive or motor efference information into an internal representation of external "motor" space involves cortical structures to the exclusion of subcortical regions. Clearly, the normal execution of complex motor acts requires the interactive participation of numerous cortical and subcortical systems. It is tempting to speculate, however, that the disturbances in reaching that we observed may be related to other behavioural disturbances which often arise from unilateral cortical pathology. Thus, the difficulty that the patients with right-hemisphere

damage had in initiating reaching movements to the target could well be related to the sorts of problems commonly observed in this kind of patient in traditional clinical settings, such as hemispatial neglect and disturbances in visual perception and construction. Similarly, the abnormal kinematics observed in the reaches of patients with left-hemisphere damage could be related to the phenomenon of manual apraxia, a deficit often associated with damage to this hemisphere. The nature of the relationship between the abnormal reaches observed in the present experiment and manual apraxia is not a straightforward one, however. Manual apraxia is most evident when a sequence of changes in posture is required rather than single or repetitive movements. Indeed, Kimura (1982) has argued that apraxic patients may be deficient in their ability to select the appropriate motor programs which allow such sequential changes in limb posture to be executed. It should not be forgotten, however, that movements requiring sequential changes in posture will often require on-line monitoring of the movement and updating of the motor program. Certainly in the present study, it is not clear whether the impairment we observed is attributable to a failure to select the appropriate motor program when one of the four targets was illuminated or whether there was a failure to monitor and correct the movement during its execution. It is possible that left-hemisphere mechanisms predominate in both these aspects of the control of skilled manual movements and that disturbances in both these processes may also contribute to the clinical phenomenon of manual apraxia.

The present study has demonstrated that the application of detailed quantitative methods to the analysis of relatively simple movements can reveal subtle differences which are not evident in clinical observation. The examination of more complex movements, such as those used in traditional clinical tests of apraxia, may make these deficits visible to a human observer in real-time. Nevertheless, our ability to describe the nature of these deficits will remain limited until we develop a better understanding of how relatively simple unrestricted movements, such as those examined in the present study, are produced by normal and by brain-damaged subjects.

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