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Limb and hemispatial hypometria

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Abstract

In a previous study, we demonstrated that unilateral cerebral lesions produce hypometric limb movements of the contralateral arm and hemispatial (i.e., directional) hypometria for movements towards contralateral hemispace. In the present study, we investigated 10 patients with right cerebral lesions and 25 healthy controls using a task to uncouple deficits in sensory perceptual systems and motor-action output systems on directional hypometria. This task required participants, with their eyes closed, to reproduce lateral and medial horizontal displacements (15–27 cm) with each arm. Each participant was seated at a waist high table and had their hand placed at an origin point aligned with the axillary fold on the same side. Their hand was moved by the investigator from the origin point to a target point and brought back to the point of origin (input displacement). The participant was then asked to return their hand to either the same target point or to an equidistant target point in the opposite direction. Healthy dextral participants were significantly more hypometric with their right arm, but patients with right cerebral lesions exhibited an opposite pattern with overall left arm hypometria. In addition, patients were significantly more hypometric for movements when output displacements were toward left hemispace. No effect was found for direction of sensory input. The results suggest that the directional hypometria is predominantly produced by hemispatial output deficits. (*JINS*, 2000, *6*, 71–75.)

Keywords: Attention, Hypometria, Motor, Neglect

INTRODUCTION

The effects of cerebral lesions on motor functions including strength, speed, initiation time, and coordination are well delineated (Brookhart & Mountcastle, 1981). However, little attention has been paid to the effects of cerebral lesions on movement size. Reports of hypometria (i.e., movements of abnormally reduced amplitude) have been limited primarily to patients with Parkinson's disease. In this population, hypometric movements are the result of reduced dopaminergic function and are manifested by micrographia, hypophonia, petit ped gait, hypometric saccades, and hypometric arm movements (Angel et al., 1970; Cassell et al., 1973; Flowers, 1976; Wilson, 1925).

Meador et al. (1986) reported contralateral limb hypometria in a patient who had a right medial prefrontal hemorrhage. In a follow-up study, Meador et al. (1988) demonstrated that unilateral lesions appeared to produce both contralateral limb hypometria as well as hypometria for movements directed toward hemispace contralateral to the lesion. However, movements of reduced amplitude might be the result of either deficits in motor programing or in sensory perceptual mechanisms. The above studies did not attempt to separate the motor and sensory contributions to the observed hypometria. In order to contrast afferent and efferent deficits on hemispatial (i.e., directional) hypometria, we developed a task that separated the effects of hemispace on perceptual sensory input and motor response output. Participants were asked to reproduce movements in the same direction as the input or in the opposite direction. By contrasting the responses in these two conditions, it is possible to determine if the hemispatial hypometria results from primarily sensory perceptual or motor programing deficits.

We predicted that if the hemispatial hypometria in patients with right cerebral lesions was due to motor programing deficits, it would occur when the motor output was

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toward the hemispace (i.e., left) that is opposite the cerebral lesion regardless of sensory input direction. If the hypometria was due to perceptual deficits, hypometria would occur when the input movements were toward left hemispace irrespective of the direction of motor output.

METHODS

Research Participants

The project was approved by the Human Assurance Committee for the Medical College of Georgia. Twenty-five healthy dextral adults and 10 dextral patients with unilateral right cerebral lesions served as participants. The study was explained to the participants and/or their legal guardians. Each read and signed an informed consent before the trials began. The controls consisted of 15 women and 10 men with a mean age of 31 years (range 22–48). Patients included 4 women and 6 men with a mean age of 42 years (range 17–70). Four patients had infarctions in the distribution of the right middle cerebral artery. One of these infarctions involved the right opercular frontal branch and caudate nucleus, one involved the right opercular frontal branch, insular cortex, and anterior external capsule, and the other 2 had spotty involvement across the distribution of the right middle cerebral artery. Two patients were status post right frontal surgery for epilepsy. One included the right premotor cortex, supplementary motor area, and anterior cingulate gyrus sparing the frontal pole; the other surgery resected the right medial frontal cortex for an arteriovenous malformation. Another patient had a right dorsal frontal meningioma resected. One patient had a right frontal subcortical infarct, and 1 had a right occipitotemporal infarct. The final patient had sustained a right frontoparietal gun shot wound. Thus, the patient group included proportionately more right anterior involvement. In all patients, motor and sensory functions ipsilateral to lesions were normal, and contralateral strength was mildly impaired in most patients (1 was normal and 1 was moderately impaired). Proprioception was normal for the index finger and proximal arm on both the ipsilateral and contralateral sides in all patients. None of the participants had denial of illness or body part at the time of testing. Although most had impaired visuospatial function, only 2 showed spatial neglect in their drawings. Five participants had mild deviation to the right on line bisection. Four participants exhibited tactile extinction. Mean time between lesion onset and testing was 8 days (range 3–10 days).

Procedure

The task required participants with eyes closed, to reproduce lateral and medial horizontal displacements of each arm. They were seated at a waist-high table with the investigator's midsagittal plane aligned with the participant's midsagittal plane. Each trial was initiated by placing the participant's hand and arm on the table at an origin point that was aligned with their ipsilateral axillary fold. For each trial, a separate sheet of paper was employed, which was marked with the origin point and two equal distant target endpoints in opposite directions. After the participant's hand was moved passively to one of the target positions and returned to the origin, the participant's task was to either (1) reproduce the distance in the same direction (i.e., isomorphic mode), or (2) reproduce the distance in the opposite direction (i.e., heteromorphic mode). The displacements ranged from 15–27 cm and were matched between conditions. The horizontal endpoint of the participant's response was marked so that the distance (mm) and direction of error from the appropriate target position could be measured. The isomorphic and heteromorphic mode tasks were performed with each hand in each axial hemispace (see Figure 1). Order of task mode, hemispace, and arm were counterbalanced across participants. Participants were administered 10 trials for each arm toward each hemispace, in each mode (i.e., same and opposite directions), resulting in a total of 80 trials per participant. Testing was conducted with the participant's eyes closed (i.e., open-looped) for input and output. However, participants were required to demonstrate an accurate performance in a single trial with eyes open (i.e., closed loop) before testing was begun. Participants were monitored to assure eye closure during testing. Two patients, who had difficulty maintaining strict eye closure due to motor impersistence, were blindfolded during testing.

ANALYSIS AND RESULTS

Mean horizontal error ($\pm$ mm) was calculated for each participant in each condition. Means ($\pm$ *SD*s) for each individual condition are depicted in Table 1. Means regrouped by arm and input/output hemispace are listed in Table 2. The data were analyzed by a 2 (space) $\times$ 2 (arm) $\times$ 2 (mode) $\times$ 2 (group) ANOVA. Significant findings included a main effect for space [$F(1,33) = 12.05$, $p < .002$], a Space $\times$ Group interaction [$F(1,33) = 5.10$, $p < .03$], and an Arm $\times$ Group interaction [$F(1,30) = 5.78$, $p < 0.02$]. No main effect of arm or mode were present (Fs < 1), and no other significant interactions were found.

To investigate the Arm $\times$ Group interaction, the left and right arm were compared in each group (see Table 2 for *M*s $\pm$ *SD*s). Healthy participants were more hypometric with their right arm ($t = -3.74$, $p < .001$). In contrast, patients were more hypometric with their left arm; although the magnitude of the left/right difference was similar, it did not reach significance for the smaller patient sample ($t = 1.01$, NS). To further delineate the Space $\times$ Group interaction, left and right space were contrasted in each group for both the output and input space. Patients were also more hypometric for responses (i.e., output) toward left hemispace ($t = 2.24$, $p < .05$) and were even slightly hypermetric toward right hemispace overall. Healthy participants had no significant left–right hemispatial difference for direction of response output ($t = 1.58$, NS). Neither patients ($t = 0.04$, NS) nor healthy

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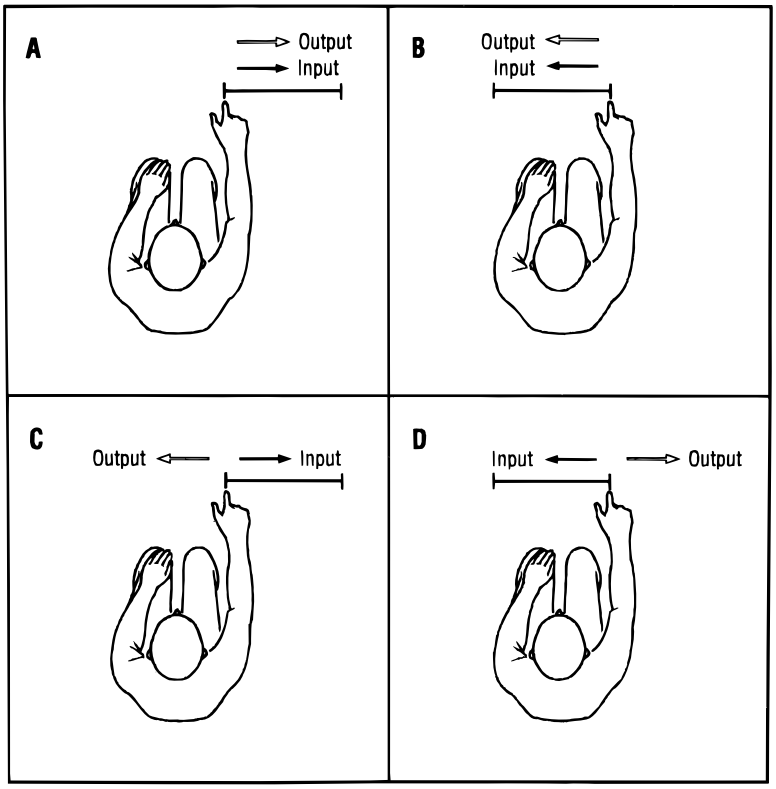

Fig. 1. The four task conditions are depicted for the right arm: A. right hemispace input and output (isomorphic mode); B. left hemispace input and output (isomorphic mode); C. right hemispace input and left hemispace output (heteromorphic mode); D. left hemispace input and right hemispace output (heteromorphic mode). The same tasks were also conducted with the left arm.

controls ($t = -0.77$, NS) had significant left–right hemispatial differences for direction of sensory input.

DISCUSSION

Each cerebral hemisphere is predominately responsible for perception of sensory stimuli from the contralateral hemibody and for motor control of the contralateral limbs. Although left–right brain differences exist, each cerebral hemisphere also mediates both sensory input and motor output in or toward contralateral hemispace (Heilman et al., 1987). Attentional (i.e., perceptual sensory processes) and intentional (i.e., motor preparation and response processes) mechanisms are interactive, but can be dissociated. For example, several investigations have examined the relative roles of attentional and intentional deficits on impaired performance of line bisection and cancellation tasks in patients with neglect syndrome (Bisiach et al., 1990; Coslett et al., 1990; Tegner & Levander, 1991). By uncoupling the direction of sensory perception and motor response, these studies have shown that patients with neglect syndrome may exhibit predominately attentional or intentional deficits. Attention-based deficits (i.e., sensory neglect) appear more prominent in patients with posterior lesions, and intention-based deficits (i.e., motor neglect) appear more prominent in patients with anterior lesions (Heilman et al., 1993).

Table 1. Response deviations for patients and healthy controls for all conditions

			Response deviations (mm)*			
	Hemispatial direction		Patients		Controls	
Arm	Input	Output	*M*	(*SD*)	*M*	(*SD*)
Right	Right	Right	+8	(26)	−7	(8)
Right	Left	Left	−7	(37)	−7	(11)
Right	Right	Left	−14	(19)	−15	(22)
Right	Left	Right	+4	(48)	−2	(22)
Left	Left	Left	−18	(39)	−5	(11)
Left	Right	Right	+4	(22)	−1	(11)
Left	Left	Right	−0.5	(53)	−2	(21)
Left	Right	Left	−18	(23)	−1	(26)

*Negative numbers denote hypometric movements.

Table 2. Response deviations re-grouped and averaged by condition for patients and healthy controls

	Response deviation (mm)*			
	Patients		Controls	
Conditions	*M*	(*SD*)	*M*	(*SD*)
Left arm	−8	(27)	−2	(10)
Right arm	−3	(22)	−8	(10)
Output toward left hemispace	−15	(20)	−7	(12)
Output toward right hemispace	+4	(31)	−3	(10)
Input toward left hemispace	−6	(32)	−4	(10)
Input toward right hemispace	−5	(17)	−6	(12)

*Negative numbers denote hypometric movements.

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Unilateral cerebral lesions can produce both contralateral limb hypometria and hypometria for movements in or toward hemispace contralateral to the lesion (Meador et al., 1986, 1988). As in prior studies, patients in the present study who had right cerebral lesions exhibited greater hypometria with the left arm as well as for output responses toward left hemispace, irrespective of the arm used. In addition, the present study demonstrated that the leftward directional hypometria was related to the hemispatial direction of output responses but not input displacements. Although the hypometria might result from impaired ongoing sensory feedback, this seems unlikely since the direction of the initial sensory input had no effect. Strength was mildly impaired in most patients, but the hemispatial hypometria cannot be explained by motor weakness or rigidity.

Watson et al. (1978) first described unilateral directional hypokinesia, in which there is difficulty in initiating movements toward the hemispace contralateral to the side of the cerebral lesion. Once movements are initiated toward left hemispace in patients with right brain lesions, the present study shows that the movements are also small in magnitude under open-looped conditions. Directional hypokinesia might be considered another example of the action-intentional disorders seen with motor neglect: akinesia, hypokinesia, motor extinction, and motor impersistence (Heilman et al., 1993). We postulate that the directional hypometria is induced by disordered motor preparation and response processing for movements directed toward the hemispace contralateral to the damaged cerebral hemisphere.

Most of the patients in this study had predominantly frontal lesions and thus might be expected to be more likely to exhibit motor output deficits. However, our patient with the most posterior lesion (i.e., occipitotemporal infarct) had greater hypometria for responses toward left hemispace in both the isomorphic and heteromorphic modes, similar to the other patients. Nevertheless, future studies will be required to assess if anterior and posterior lesions affect the metric of movements differentially in patients with predominantly sensory *versus* motor neglect. The right hemisphere appears to be dominant in some aspects of motor activation (i.e., intention) because right hemisphere lesions are more frequently associated with akinesia (motor neglect), hypokinesia (delayed reaction times), contralateral gaze paresis, and motor impersistance (Heilman et al., 1993; Meador et al., 1989). Whether directional hypometria is differentially produced by right *versus* left cerebral lesions remains to be determined.

Our healthy dextral participants had greater hypometria with their right than left arm. This finding is interesting since the right hand would be expected to be more accurate for skilled movements. The left hand in dextrals has a lower sensory threshold (Meador et al., 1998), and the right brain is more specialized in spatial processing (Heilman et al., 1993). However, if the right hand was simply less accurate, then the right-hand errors would be as likely to be hypermetric as hypometric. The right hemisphere is superior for mediating intention in healthy participants during a cued choice–reaction-time task (Verfaellie et al., 1988). In addition, as mentioned above, there is clinical evidence that the right hemisphere plays a dominant role in motor activation.

Since hypometria is a prominent component of Parkinson's disease, is it possible that the relative right hand hypometria seen in healthy dextrals is due to cerebral asymmetries in dopamine? Paw preference in mice is correlated with dopamine asymmetries in the nucleus accumbens; dopamine was found to be higher on the side ipsilateral to paw preference (Cabib et al., 1995). Circling behavior in rats has been found to be related to dopamine asymmetries, although the direction of turning biases may be ipsilateral or contralateral to the side of greater dopamine content depending on rat population (Shapiro et al., 1987). Neurotransmitter asymmetries have been demonstrated in the human brain, and dopamine was found overall to be higher in the right substantia nigra and putamen, but higher in the left caudate and globus pallidus (Glick et al., 1982).

We postulate that the right-hand hypometria in healthy dextrals is related to asymmetries in the dopamine systems, which exist as a function of normal cerebral lateralization. Exactly how this neurotransmitter asymmetry is related to the behavioral asymmetries is uncertain. At least two explanations are possible. The gain on neuronal responses related to motor activation may be relatively higher in the right hemisphere. Alternately, the neurochemical asymmetry may be indirectly related to asymmetries in the functional organization of the left and right cerebral hemisphere.

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