Lateralised motor control: hemispheric damage and the loss of dexterity

B Hanna-Pladdy, J E Mendoza, G T Apostolos, K M Heilman

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See end of article for authors' affiliations

Correspondence to:
Dr B Hanna-Pladdy,
Department of Neurology,
University of Florida Health
Science Center, Box
100236 UFBL,
Gainesville FL 32610-0236
USA;
pladdybh@onebox.com

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Objective: To learn if the left compared with the right hemisphere of right handed subjects exerts bilateral compared with contralateral motor control when performing precise and coordinated finger movements.

Methods: The study investigated intertask differences of manual motor asymmetries such as speed, precision, and independent finger movements, in patients with unilateral lesions of the left (LHD) or right hemisphere (RHD) and normal controls (C).

Results: Normal subjects showed the greatest right hand preference on a task that required rapid coordinated and precise independent finger movements (coin rotation). Both hemisphere damaged groups revealed contralateral motor deficits, but the magnitudes of asymmetries were found to be significantly different (RHD>C>LHD) with contralateral and ipsilateral deficits for LHD subjects. The greatest ipsilateral deficits for the LHD subjects were on those tasks that require precision (grooved pegboard and coin rotation).

Conclusions: The degree of hemispheric specialisation is, in part, dependent upon the nature of the motor task, with left hemisphere motor control necessary for tasks that require precision and coordinated independent finger movements.

Hugo Liepmann^{1,2} reported that hemispheric lesions, even in the absence of weakness, could be associated with a loss of the ability to perform skilled movements, apraxia. He described three forms of apraxia—ideomotor, ideational, and limb kinetic. When performing skilled movements patients with ideomotor apraxia make spatial and temporal errors.^{3–4} When attempting to use a screwdriver, for example, instead of rotating the screwdriver on its axis they may rotate it so that the head moves in circles. Patients with ideational apraxia may be impaired in sequencing a series of acts that lead to a goal. For example, when making a sandwich, they may cut the sandwich in half before placing the meat between the bread. Ideational apraxia has also been used to describe patients who make content errors (for example, pound with a screwdriver and screw with a hammer).^{5–6} To reduce confusing terminology, some investigators now call this second form of ideational apraxia, “conceptual apraxia”.^{6,7} According to Liepmann,^{1,2} patients with limb kinetic apraxia have slowed and stiff movements and, when attempting to perform skilled acts, their movements are coarse and clumsy. Kleist⁸ called this form of apraxia, “innervatory apraxia”. He noted that people with this form of apraxia have a loss of independent finger movements, and have problems coordinating simultaneous movements.

Liepmann² provided evidence that, in right handed people, ideomotor apraxia is more commonly associated with left than right hemisphere dysfunction. To learn if limb kinetic apraxia is more frequently associated with left than right hemisphere dysfunction, Heilman and colleagues⁹ studied patients with epilepsy undergoing selective hemisphere anaesthesia. They found that with left hemisphere anaesthesia, right handed subjects with left hemisphere language dominance, had a loss of dexterity or dexterity (limb kinetic apraxia) of both the contralateral right and the ipsilateral left hands. In contrast, with right hemisphere anaesthesia, only the contralateral left hand had a loss of dexterity. Based on this study, the authors concluded that the left hemisphere of right handed people mediates motor dexterity for both hands, while the right hemisphere mediates dexterity for only the contralateral left hand.

The method used to assess for dexterity in the Heilman *et al* study⁹ was observing the patients while they pretended to use four different tools. The apraxic errors classified as being limb kinetic were those characterised by slowness and stiffness with a loss of fine movements. However, no actual measurements of dexterity including: speed, precision, sensorimotor integration, the ability to perform independent finger movements, or coordinate movements, were performed. Additionally, this study included epileptic subjects who could conceivably have aberrant brain organisation.⁹ In the current study, we examined dexterity in right handed experimental subjects with unilateral lesions and matched controls with tests that assess speed, precision, coordinated but independent finger movements.

METHODS

Subjects

The sample consisted of 86 right handed men, as determined by a handedness inventory,¹⁰ who were recruited and tested at the New Orleans Veterans Administration Medical Center. The subject groups included 60 normal controls (C), 13 patients with unilateral lesions confined to the left hemisphere (LHD), and 13 patients with unilateral lesions of the right hemisphere (RHD; see appendix A). The ages of the subjects ranged from 40 to 79 years (mean_{controls}=58.9, mean_{LHD}=58.5, mean_{RHD}=63.4). Patients were screened for previous history of neurological and psychiatric illness, or significant alcohol/drug misuse. Patients were also excluded from the study if they had a non-neurological disease that could cause motor disability, or if they were unable to complete the motor tests with both hands. It is unusual for two patients to have lesions exactly the same size, configuration, and location. In addition, even pre-morbidly there is individual variation in the size, location, and configuration of gyri and sulci. There are no studies, therefore, that completely matched right and left hemisphere lesions, and in this experiment subjects were entered into the study sequentially without an attempt to find subjects whose hemispheric lesions precisely mirrored one another (or each other).

Table 1 Means (SD) for motor tasks

Task	Group	Right hand	Left hand
Hand tapping	LHD	35.8 (11.5)*	38.7 (9.6)
	RHD	41.9 (5)	36.8 (5.4)*
	Control	47.3 (5.7)	44.1 (7.3)
Finger tapping	LHD	38.5 (14.9)*	40.4 (9.8)†
	RHD	39.9 (6.9)†	34.0 (6.2)*
	Control	50.8 (5.7)	45.9 (5.9)
Grooved pegboard	LHD	205.1 (129)*	169.5 (139)†
	RHD	109.8 (37.8)	198.4 (99)*
	Control	91.7 (29.2)	98.9 (29.2)
Coin rotation	LHD	22.2 (8.1)*	18.9 (6.7)†
	RHD	15.4 (3.7)	24.9 (6.9)*
	Control	12.9 (2.5)	14.5 (2.6)

*Contralateral impairment. †Ipsilateral impairment. Finger tapping and hand tapping measured in mean number of taps. Grooved pegboard and coin rotation measured in seconds.

Procedures

Screening for contralateral weakness was performed by testing grip strength with a hand held Stoelting dynamometer.¹¹ The grip strength score was based on the mean of three trials for each hand expressed as kg of pressure exerted.

To assess dexterity we used several tests including: the finger tapping test (Lafayette Instruments) that measures the speed of open looped movements,¹¹ and the grooved pegboard test (Lafayette Instruments) to assess closed loop precision.¹² The grooved pegboard consists of a 5×5 matrix of keyhole shaped holes in various orientations. The score for the grooved pegboard was the amount of time required to complete the task, including the additional time in the event the subject dropped the peg. Because the grooved pegboard primarily measures precision of proximal movements we used a new “coin rotation” task devised by Mendoza and colleagues,¹³ to measure precision of distal movements. This test also requires the coordination of independent finger movements. In this coin rotation task, the subject rotates a nickel 180 degrees as rapidly as possible for 20 times between their thumb, index, and middle fingers. The examiner measures the time it takes to complete this task including the episodes where the coin is dropped. Unlike the finger tapping and grooved pegboard test, the coin rotation test can be used by clinicians at the bedside without having to purchase or carry additional apparatus.¹³ Lastly, we tested subjects with a handheld (Lion) tapping device. Unlike the former tests, this test does not require independent finger movements or precision. The score for finger and hand tapping was the mean number of taps in five 10 second trials. Deviant trials exceeding a 5 point range from fastest to slowest were not discarded. Each subject was given three trials on the coin rotation and grip strength tasks for each hand. However, the grooved pegboard test was administered only once for each hand, while the finger and hand held tapping tests were administered five times for each hand. Each subject was tested on all motor tests beginning with the dominant hand, and alternating between hands, in a counter-balanced order.

To allow for direct comparison of the motor tasks between the RHD and LHD groups, the data were converted to standard scores with a mean of 0 and a standard deviation (SD) of 1 based on the performance of the control subjects. Standardisation of motor tasks was based on the means and SD of control subjects for each motor task ($Z \text{ score} = (\text{mean of controls} - \text{lesion subject performance}) / \text{SD of controls}$) to represent impairment for lesion groups in terms of SD below that of the control group (control group $Z \text{ scores} = 0$; see figs 1 and 2). This standardisation was also performed so that comparison of performance across task demands could be conducted. To

explore hand discrepancies of motor performance between LHD and RHD groups, an asymmetry index was derived. Manual asymmetries were computed for each individual subject in the present study using the formula preferred right hand (R) minus non-preferred left hand (L) divided by the preferred right hand $((R-L)/R)$ for the motor tasks, with positive scores indicating better performance with the preferred hand.

RESULTS

Screening

There was a significant difference between groups for grip strength for both the R preferred ($F(2, 85) = 12.9, p < 0.0001$) and L non-preferred ($F(2, 85) = 12.95, p < 0.0001$) hands. Post hoc analysis with Scheffe's method demonstrated that both groups had significant contralateral weakness relative to the control group, $p < 0.0001$, consistent with the hemisphere that was damaged. That is, the LHD group demonstrated significant impairment for the right hand ($\text{mean}_{\text{LHD}} = 33.9, \text{mean}_{\text{RHD}} = 40.3, \text{mean}_{\text{controls}} = 45.5$), while the RHD group was impaired for the left hand ($\text{mean}_{\text{LHD}} = 36, \text{mean}_{\text{RHD}} = 30.5, \text{mean}_{\text{controls}} = 40.9$).

Motor tasks

The means and standard deviations of raw scores by group on tests of dexterity are presented in table 1. Standard score ($Z \text{ scores}$) conversion of raw scores for each of the tasks, relative to the means and standard deviations of the control group, are displayed in figures 1 and 2. Asymmetry indices for each of the tasks by group are presented in table 2.

A between subjects multivariate analysis of variance was performed on the four dependent motor variables (hand tapping, finger tapping, grooved pegboard, coin rotation) for both the R preferred and L non-preferred hands. Effect of group (C, LHD, RHD) on the dependent variables (DV) was significant for the multivariate equation, $F(8, 160) = 10.2, p < 0.0001$. Further investigation with univariate analyses revealed between groups differences for all motor tasks for both the R preferred and L non-preferred hands: hand tapping ($F_R(2, 83) = 16.03, p < 0.0001; F_L(2, 83) = 6.2, p < 0.005$), finger tapping ($F_R(2, 83) = 18.6, p < 0.0001; F_L(2, 83) = 16.4, p < 0.0001$), grooved pegboard ($F_R(2, 83) = 20.3, p < 0.0001; F_L(2, 83) = 12.9, p < 0.0001$), and coin rotation ($F_R(2, 83) = 28.2, p < 0.0001; F_L(2, 83) = 30.9, p < 0.0001$).

Scheffe's post hoc analysis revealed that the LHD group, displayed R contralateral impairment compared with normal controls across all measures of dexterity ($p < 0.0001$; see table 1). The RHD group's performance with the L contralesional hand was below that of normals on all tasks ($p < 0.02$). LHD subjects also

Table 2 Mean asymmetry quotients ((R-L)/R) by group

	GS	HT	FT	CR	GP
LHD	-0.093**	-0.114**	-0.133**	-0.113	-0.173*
RHD	0.242**	0.147	0.169	0.914**	0.924**
Control	0.097	0.068	0.094	0.141	0.096

((R-L)/R) = Right hand minus left hand relative to the right hand. *Significant difference relative to control group, $p < 0.01$. **Significant difference relative to control group, $p < 0.001$.

Figure 1 Standardised Z scores for motor tasks for the left hemisphere damaged group. **Figure 2** Standardised Z scores for motor tasks for the right hemisphere damaged group.

had ipsilateral impairment on tests of finger tapping ($p=0.03$), grooved pegboard ($p=0.006$), and coin rotation ($p=0.004$) compared with controls, while the RHD group had ipsilateral impairment only on the finger tapping test ($p=0.001$; see table 1).

Asymmetry indices

Inspection of group means revealed similar AQs in terms of magnitude and direction (right hand superiority) for unpaired controls approaching the expected 10% advantage of the R hand for grooved pegboard, grip strength, and for finger tapping (table 2). Although the asymmetry indices for controls on coin rotation and handheld tapping also evidenced right hand superiority, the coin rotation task had slightly greater asymmetry (0.141) while handheld tapping had slightly less (0.068). A multivariate analysis of variance, with AQs for hand tapping, finger tapping, grooved pegboard, and coin rotation as the DVs, was significant between groups, $F(8, 160) = 12.7$, $p < 0.0001$. Univariate F tests were significant for the asymmetry indices of all motor tasks: hand tapping AQ ($F(2, 83) = 13.9$, $p < 0.0001$), finger tapping AQ ($F(2, 83) = 19.7$, $p < 0.0001$), grooved pegboard AQ ($F(2, 83) = 35.5$, $p < 0.0001$), and coin rotation AQ ($F(2, 83) = 24.7$, $p < 0.0001$).

The RHD group evidenced larger positive hand asymmetries compared to the LHD group on all tasks, $p=0.0001$, and larger positive hand asymmetries only on the grooved pegboard and coin rotation tasks relative to control subjects, $p=0.0001$. The asymmetries for the RHD group were greatest for the most

complex tasks that require precision, grooved pegboard and coin rotation (see table 2 for means and significance), and reflect the relatively spared ipsilesional right hand performance (see table 1 and fig 2). The LHD group was found to have negative asymmetries for all tasks, indicating left hand rather than right hand superiority, but with much smaller magnitudes than the RHD group across tasks consistent with a greater degree of ipsilateral impairment (see table 1 and fig 1). The LHD had significantly larger AQ than controls on less complex tasks, hand tapping and finger tapping, $p < 0.0001$, and grooved pegboard which requires precision, but not independent finger movements, $p < 0.05$. The LHD did not have a significantly different AQ for coin rotation when compared with controls, consistent with bilateral impairment on this task.

DISCUSSION

The hand asymmetries for grooved pegboard, finger tapping, and grip strength we observed in this study for our control subjects are similar to those previously reported.^{14, 15} The handheld tapping and coin rotation tasks in the normal subjects, however, were found to have asymmetry indices that are different than the more traditional measures of motor skills used in this study. The handheld tapping task, which does not require independent finger or precise movements, was found to have the smallest hand asymmetry for control subjects. Conversely, the coin rotation task that requires independent finger movements, precision, coordination, and sensorimotor integration, had large hand asymmetries for control subjects. This suggests that the left hemisphere can programme distal independent but coordinated precise movements better than the right hemisphere.

On tasks that require speed but do not require precise open looped movements (finger tapping), our results revealed some ipsilateral control from both hemispheres. However, both the right hemisphere and left hemisphere damaged subjects demonstrated ipsilesional superiority on all tasks. These asymmetries are consistent with hemispheric damage to motor systems. However, the magnitude of the asymmetries was greater for subjects with right hemisphere damage than those with left hemisphere damage. There are two possible explanations for these hemispheric asymmetries. One possibility is that the subjects with right hemisphere damage had more severe damage to motor areas than did the subjects with left hemisphere damage. However, Z scores across motor tasks do not support greater contralesional impairment for the right hemisphere damaged group when compared with the left hemisphere damaged group, making this hypothesis unlikely. Alternatively, these results suggest that in right handed subjects, the right hemisphere primarily controls the left hand, whereas the left hemisphere exerts bilateral control. Our results support the Wada study by Heilman and colleagues⁹ and demonstrate the specific type of motor deficits that can lead to limb kinetic apraxia in non-epileptic subjects, but also conform to previous reports that right cerebral lesions accentuate the pattern of intermanual asymmetries observed, while left hemisphere lesions yield only small discrepancies between the hands.¹⁶⁻¹⁹ The hypothesis that the left hemisphere exerts ipsilateral control compared with the anatomical injury hypotheses is also supported by the hemispheric

anaesthesia (Wada) study. Unlike stroke that may injure specific anatomical areas, the barbiturate is injected into the internal carotid and anaesthetises all the motor areas that are in the distribution of the middle and anterior cerebral arteries.⁹ Our findings also show that those tasks that require the most dexterity (precision, sensorimotor integration, and coordinated but independent finger movements), were the ones that were most impaired in the ipsilateral limb after left hemisphere damage. Although this observation further supports the hemispheric motor asymmetry hypothesis compared with the anatomical hypothesis, it would have been interesting to learn what areas of the left hemisphere when damaged induced these ipsilesional deficits. Unfortunately, specific anatomical data were not obtained and therefore the loci of lesions that induced these deficits could not be determined. Future studies may be directed at determining the anatomical basis for these asymmetries.

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Authors' affiliations

B Hanna-Pladdy, K M Heilman, Department of Neurology, University of Florida, Gainesville, USA

J E Mendoza, G T Apostolos, Mental Health Service, Veterans Affairs Medical Center, New Orleans, USA

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Appendix A Clinical and lesion data for LHD and RHD patients

Subject	Clinical Signs	Neuropathologic Data
01LHD	R hemiparesis	CT – wnl
02LHD	R hemiparesis	Complete occlusion of L common and internal carotid arteries
03LHD	Transient right weakness, comprehension, naming, and verbal memory deficits	Mass occupying L inferior and middle temporal lobe
04LHD	Acute onset of R LE paralysis, PE = RUE 4/5, RLE 1/5	Infarct of L temporal parietal lobes
05LHD	R hemiparesis and paresthesias, residual expressive dysphasia	Lacunes in the L internal capsule and basal ganglia
06LHD	Loss of speech which resolved to a mild expressive aphasia, right homonymous hemianopsia, sensory deficits in U & L ext, mild weakness of RUE	Acute infarct in the L posterior parietal region
07LHD	Mild R sided weakness	Lacune in L basal ganglia, and ischemia of L parietal lobe
08LHD	Sudden onset R hemiparesis and dysarthria, mild residual arm and facial weakness, paresthesias	Lacunar infarct in L parietal periventricular deep white matter
09LHD	R hemiparesis	CT – wnl except atrophy
10LHD	Sudden onset aphasia, blurred vision, loss of sensation RUE and RLE weakness.	L parietal infarct with mild to moderate diffuse atrophy
11LHD	R sided paresthesias and sensory changes, R facial tremor	Variable density in L frontal parietal region suspicious for neoplasm
12LHD	Mild R sided weakness and paresthesias	CT and MRI wnl
13LHD	Mild weakness of RLE, aphasia	L infarct of occipital temporal region, L thalamus
01RHD	LUE weakness, L superior temporal visual field loss	Resection of meningioma from R posterior frontal and R inferior temporal lobe
02RHD	LUE and LLE weakness and incoordination, L facial weakness	CT – wnl except for mild diffuse atrophy
03RHD	Mild, variable weakness in LUE and LLE	Infarct of R anterior limb of internal capsule and basal ganglia
04RHD	L numbness and mild weakness	Lacune in R anterior limb of internal capsule
05RHD	Subtle weakness of LUE	R hemisphere lacune
06RHD	Mild LUE weakness	Small lacune in R basal ganglia adjacent to anterior limb of internal capsule
07RHD	L hemiparesis (4/5), L Babinski, L palmomental	Neoplasm of R parietal lobe posteriorly and R basal ganglia
08RHD	L sided weakness	Large R temporal parietal infarction extending to the posterior R frontal lobe
09RHD	LUE and LLE weakness, L homonymous hemianopsia, LUE 2 point discrimination deficit	R parietal infarct
10RHD	L sided numbness/ weakness	CT – wnl
11RHD	L sided weakness	Infarct in deep white matter of R parietal lobe
12RHD	L sided sensory change, weakness, pronator drift	R infarct at parietal occipital junction
13RHD	Mild L weakness greater in leg than arm	Infarct in R anterior cerebral artery distribution