Lateralised motor control: hemispheric damage and the loss of deftness

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

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 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. 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). To reduce confusing terminology, some investigators now call this second form of ideational apraxia, “conceptual apraxia”.

According to Liepmann, patients with limb kinetic apraxia have slowed and stiff movements and, when attempting to perform skilled acts, their movements are coarse and clumsy. Kleist 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.

Liepmann 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 colleagues’ 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 deftness or dexterity (limb kinetic apraxia) of both the contralateral right and ipsilateral left hands. In contrast, with right hemisphere anaesthesia, only the contralateral left hand had a loss of deftness. Based on this study, the authors concluded that the left hemisphere of right handed people mediates motor deftness for both hands, while the right hemisphere mediates deftness for only the contralateral left hand.

The method used to assess for deftness in the Heilman et al study 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 deftness 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. In the current study, we examined deftness 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, 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 C = 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 premorbidly 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).
Procedures
Screening for contralateral weakness was performed by testing grip strength with a hand held Stoebling dynamometer. The grip strength score was based on the mean of three trials for each hand expressed as kg of pressure exerted.

To assess deftness we used several tests including: the finger tapping test (Lafayette Instruments) that measures the speed of open looped movements, and the grooved pegboard test (Lafayette Instruments) to assess closed loop precision. The grooved pegboard consists of a 5x5 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, 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.

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 discounted. 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 counterbalanced 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 score = (mean of controls−lesion subject performance)/SD of controls) to represent impairment for lesion groups in terms of SD below that of the control group. 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 Scheffé’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 (mean L = 33.9, mean R = 30.5, mean asymmetry = 0.40), while the RHD group was impaired for the left hand (mean L = 36, mean R = 20.5, mean asymmetry = 0.40).

Motor tasks
The means and standard deviations of raw scores by group on tests of deftness are presented in table 1. Standard score (Z 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 (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. 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(2, 83) = 16.03, p<0.0001; F(2, 83) = 6.2, p<0.005), finger tapping (F(2, 83) = 18.6, p<0.0001; F(2, 83) = 16.4, p<0.0001), grooved pegboard (F(2, 83) = 20.3, p<0.0001; F(2, 83) = 12.9, p<0.0001), and coin rotation (F(2, 83) = 28.2, p<0.0001; F(2, 83) = 30.9, p<0.0001).

Scheffé’s post hoc analysis revealed that the LHD group displayed R contralateral impairment compared with normal controls across all measures of deftness (p<0.0001; see table 1). The RHD group’s performance with the R contralesional hand was below that of normals on all tasks (p<0.02). LHD subjects also explored R contralateral impairment compared with normal controls across all measures of deftness (p<0.0001; see table 1).
The RHD group evidenced larger positive hand asymmetries compared to the LHD group on all tasks, $p=0.006$, 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 unimpaired 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 (0.68). A multivariate analysis of variance, with AQs for hand tapping, finger tapping, grooved pegboard, and coin rotation as the DVs, was significant between groups, $F(2, 83) = 13.9$, $p<0.0001$. Univariate $F$ tests were significant 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. The hand handed 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 Hellman and colleagues 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. The hypothesis that the left hemisphere exerts ipsilateral control compared with the anatomical injury hypotheses is also supported by the hemispheric

![Figure 1](image1.png) Standardised Z scores for motor tasks for the left hemisphere damaged group.

![Figure 2](image2.png) Standardised Z scores for motor tasks for the right hemisphere damaged group.

| Table 2 Mean asymmetry quotients ((R−L)/R) by group |
|----------------|----------------|----------------|----------------|----------------|
|                | GS             | FT             | CR             | GP             |
| LHD            | −0.093**       | −0.114**       | −0.133**       | −0.113         |
| RHD            | 0.242**        | 0.147          | 0.169          | 0.914**        |
| Control        | 0.097          | 0.086          | 0.094          | 0.141          |

$(R−L)/R = $ Right hand minus left hand relative to the right hand. *Significant difference relative to control group, $p<0.05$. **Significant difference relative to control group, $p<0.001$. 

www.jnnp.com
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. Our findings also show that those tasks that require the most defensiveness (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.

ACKNOWLEDGEMENTS
This work was presented, in part, at the 27th Annual meeting of the International Neuropsychological Society (INS), Boston, MA, February 1999.

Authors’ affiliations
B Hanno-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
Competing interests: none declared.

Appendix A Clinical and lesion data for LHD and RHD patients

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

REFERENCES

www.jnnp.com
Lateralised motor control: hemispheric damage and the loss of deftness

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

*J Neurol Neurosurg Psychiatry* 2002 73: 574-577
doi: 10.1136/jnnp.73.5.574

Updated information and services can be found at:
http://jnnp.bmj.com/content/73/5/574

These include:

References
This article cites 11 articles, 5 of which you can access for free at:
http://jnnp.bmj.com/content/73/5/574#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/