The Good Side after Stroke: Ipsilaterial Sensory-motor Function needs Careful Assessment

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Summary
Twenty subjects were examined 4—6 weeks after stroke to establish whether a sensory-motor ipsilateral deficit occurs early after stroke. Each underwent a timed test of repetitive side-to-side movement of both the upper and lower limbs ipsilateral to the cerebral infarct, and an assessment of motor disability using the Motor Assessment Scale. Results were compared with a group studied almost a year after their stroke, and with 41 age-matched healthy volunteers.

There was a significantly worse performance (p < 0.005) on the right ipsilateral side, but not the left ipsilateral side, compared with normal volunteers, a finding similar to that of a group previously studied about a year after the stroke. There was no relationship between the severity of the motor deficit and performance of the 'good' side. This study suggests that ipsilateral sensory-motor deficit occurs after stroke but only on the right side, possibly owing to reduction in cerebral activation as a result of a right hemispheric lesion.

These observations have importance in rehabilitation and education as well as practical skills, including driving a car and maintaining balance.

Introduction
Rehabilitation assessment and programmes after stroke pay little attention to the so-called 'good side', the side ipsilateral to the cerebral infarct. None the less, stroke patients may report clumsiness in the non-hemiplegic limbs and this may compromise recovery. A number of studies have reported sensory-motor deficit in the limbs ipsilateral to the cerebral infarct of patients with a single focal hemispheric lesion. This deficit varies, possibly owing to differences in test instruments or the nature of the lesion. For example, ipsilateral deficit of similar severity on either the dominant or non-dominant side has been reported in limb strength [1—3], although other studies disagree, reporting ipsilateral limb strength to be normal [4, 5]. Ipsilateral deficits in either side have also been found in tests of hand function [6], visual tracking tests [4]; and static and vertical groove steadiness, maze co-ordination and pegboard tasks [5].

Some researchers have reported a deficit only on one ipsilateral side but not the other. Abnormalities occurring only on the left side ipsilateral to a cerebral lesion (infarct or tumour) have been reported using a variety of tests. These include postural arm drift [7], response to a visual light cue by depressing a target key [8], copying visually presented meaningless hand movement [9], alternate tapping movements between left and right hand [10], sequenced motor tasks [11], simple aiming tasks [12], and tapping between large vertical targets surrounded by error plates [13].

Others have reported abnormalities only in the right side ipsilateral to a right focal hemispheric lesion, and not the left, in tests involving simple reaction time in the lower limb, and coin sorting in the upper limb [14], response to an auditory signal by depressing a telegraph key with the index finger [15], and in side-to-side tapping of both upper and lower limbs [16].

In a previous study we reported significant differences in sensory-motor function compared with age-matched healthy volunteers, but only in the ipsilateral right side after a single focal hemispheric cerebrovascular lesion [16]. These people had had a stroke on average 10.5 months prior to testing (range 2—192 months). It has been postulated that motor weakness in the ipsilateral side is due to disruption of ipsilateral motor fibres [1]. We therefore hypothesized that if weakness were the reason for our findings then immediately after the stroke both sides would be abnormal compared with controls. However after several months left-side performance would improve owing to a practised or learned phenomenon because those now only with a useful left arm and/or leg had been forced to change from right-handed dominance.
and were now reliant on their left side for simple daily activities. On the other hand complex integration mechanisms have been suggested for sensory-motor function whereby the left cerebral hemisphere may be especially important for controlling ballistic functions, and the right cerebral hemisphere in so-called sensory dependent processing [17–20]. If disruption of these processes were the reason for reduced performance in the right ipsilateral side, but not the left, we would expect findings similar to our first study, that is reduced sensory-motor performance only on the right side ipsilateral early after a right hemispheric infarct.

To investigate this ipsilateral sensory-motor abnormality further we repeated the same tests on a new group of patients with a single focal hemispheric infarct. This group was tested within 4–6 weeks of the stroke, and we report the findings.

Approval for the study was given by the University of Auckland Human Subjects Ethics Committee.

Methods

Patients admitted to hospital with a first stroke were entered into the study 4–6 weeks after the initial event. A stroke was defined according to the World Health Organization’s definition as the acute onset of a focal or global neurological deficit, presumably of vascular origin, lasting more than 24 hours. Confirmation of a unilateral infarct was made from a history consistent with a single acute ischaemic infarction of a cerebral hemisphere, appropriate examination findings, and any additional confirmation as available from other sources, e.g. a CT scan.

Subjects excluded from the study included those who had evidence of more than one cerebral infarct, had no motor deficit, evidence of poor cognitive function (including a mental test score [21] of less than 8/10), visual acuity worse than N14 (Reading Test Types, Keeler Ltd); and, on the ipsilateral side, evidence of impaired proprioception, touch and pain sensation, and ataxia. Other exclusion criteria included the presence of medical conditions which might limit arm or leg movement such as pain or arthritis, prescription drugs with known sedative properties which could interfere with sensory-motor function, and those whose English language was such that they might have difficulty comprehending the nature of the research or the instructions for testing procedures.

Each patient was screened for visuo-spatial neglect using the letter cancellation test [22], and clock drawing using a predrawn circle and asking subjects to fill in the hours of the day [23]. All subjects with moderate or severe visuo-spatial neglect were excluded, but those who made only very minor errors were included.

Sensory-motor function was tested using a modification of tests described by Potvin and Tourtellotte [24, 25]. This apparatus consisted of a flat wooden board on which two touch-sensitive target plates (20.4 × 12 cm) were fixed 25 cm apart, and attached to a digital counter. When testing arm function, the board was placed securely on a table at the level of the seated patient’s waist, the seat height being adjusted as required. One target was positioned directly in the subject’s midline, the other target 25 cm away to the side of the patient to be tested, that is to the side ipsilateral to the cerebral infarct. Each target consisted of two equal-sized metal plates separated so that contact on the plate registered only if the patient placed the palm of the hand lightly on both so as to complete the circuit. Having demonstrated the necessary technique the patient was then required to tap each target alternately as fast as possible over a timed 30-s period, using the arm ipsilateral to the cerebral infarct. The number of contacts made on each target was recorded at 10 s, and again at 30 s.

The leg was tested in a similar manner with the subject seated on the adjustable chair with feet resting on the floor. Standard positioning was achieved by placing the foot on one target box directly in front of the side to be tested, seat height then being adjusted so that the knee angle was at 115° flexion (measured with a goniometer). The touch-sensitive target plates were then repositioned so that one was opposite the midline of the patient and the other 25 cm towards the side to be tested. The subject was then required to tap each target alternately with the sole of the foot for 30 s. The number of contacts was recorded at 10 and 30 s. In testing both ipsilateral limbs emphasis was on speed and accuracy.

Assessment of motor function on the hemiplegic side was made using the Motor Assessment Scale (MAS), an instrument which has demonstrated high inter- and intra-tester reliability [26]. It includes eight items representing seven areas of motor function and one item relating to general tone. Each test is scored on a seven-point scale (from 0 to 6). A score of six represents optimal motor behaviour in each test, to a total maximum combined score of 48.

The performance of the 20 patients in this study was compared with that of the 20 we reported previously [16] where stroke subjects were studied 2–192 months after their stroke. Both groups were compared with 41 healthy subjects of similar age acting as controls. Control subjects were selected from a nearby retirement village, all reporting good physical health and who were not on medications known to slow sensory-motor function.

For the timed tests, differences between the groups were tested using the Student’s t test. Pearson Correlation Coefficients were calculated to assess the relationship between severity of motor deficit and performance on the ‘good side’. Multivariate linear regression analysis was used to explore the relationships between age, MAS score, tapping side, group and performance measures.

Results

This study group included 20 subjects, 11 with a left cerebral infarct and nine with a right cerebral infarct. All indicated strong right upper and lower limb preference for activities requiring precision suggesting left cerebral hemispheric dominance [27]. All were studied between 4 and 6 weeks after the stroke, compared with 2–192 months in the previous study. Table I gives the descriptive characteristics of each group.

The average age of the subjects was 70.0 years (range 50–85 years), the control group 71.6 years (range 51–90), and the group previously studied 67.1 years (range 54–85).

Only four of the 20 subjects had had a CT scan and in each case this confirmed the presence of a single cerebral infarct. No subjects demonstrated errors in clock drawing but four had minor errors in the letter cancellation test missing up to three letters. Three of these had a right cerebral infarct, and one a left infarct.
Table I. Descriptive characteristics of each group

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Control</th>
<th>Late after stroke</th>
<th>Early after stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range</td>
<td>71.6 (9.6)</td>
<td>67.1 (7.9)</td>
<td>70.0 (10.6)</td>
</tr>
<tr>
<td>Men</td>
<td>51–90</td>
<td>54–85</td>
<td>50–86</td>
</tr>
<tr>
<td>Women</td>
<td>18</td>
<td>12</td>
<td>8</td>
</tr>
<tr>
<td>Dominance</td>
<td>Right 40</td>
<td>19</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>Ambidextrous</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Time post stroke</td>
<td>–</td>
<td>2–192 months</td>
<td>4–6 weeks</td>
</tr>
<tr>
<td>Side of infarct</td>
<td>–</td>
<td>11 L</td>
<td>11 L</td>
</tr>
<tr>
<td></td>
<td>–</td>
<td>11 R</td>
<td>9 R</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>41</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Mean MAS score</td>
<td>–</td>
<td>27.8</td>
<td>34.5</td>
</tr>
</tbody>
</table>

Age: arithmetic mean (standard deviation). MAS (Motor Assessment Scale): scored on a scale in which 0 represents no physical function, 48 normal function.

Table II gives the mean scores and statistical values for each group. Subjects with a right hemispheric infarct showed significantly reduced slowing of sensory-motor performance in the 'good' right ipsilateral side compared with the performance of control subjects on their right side. No difference was found on the ipsilateral left side (left hemisphere infarct) compared with performance on the left side of the control group. The differences observed on the right side were evident in both the upper and lower limbs and after 10 and 30 s. Exclusion from analysis of patients with minor errors in the letter cancellation test made no substantial difference to the results.

No significant difference was found in side-to-side tapping speed between the ipsilateral right or left side when the recent-onset stroke group was compared with the group tested late after the stroke. It should be noted here that a review of the earlier data from the late group revealed a significant difference on the left good side compared with controls, but only in the lower limb, and only at 30 s (p = 0.007).

Two models were used to predict speed of arm side-to-side tapping over 30 s, using as independent variables the group (late or early, compared with controls) and age. One model included only left-sided strokes, and one the right. The model for the left scores was not useful (r^2 = 0.16, p = 0.13), showing only a slight relationship between age and tapping score. Conversely, the model for the right scores shows a strong relationship (r^2 = 0.48, p = 0.0001), with age and group showing significant effects. The difference

Table II. Mean scores (and standard deviations) for side-to-side tapping in the three study groups

<table>
<thead>
<tr>
<th>Limb</th>
<th>Time (s)</th>
<th>Side tested</th>
<th>Control</th>
<th>Early after stroke</th>
<th>Early vs. control* (p)</th>
<th>Late after stroke</th>
<th>Late vs. control* (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arm</td>
<td>10</td>
<td>R</td>
<td>38.6 (10.7)</td>
<td>28.9 (8.1)</td>
<td>0.02</td>
<td>26.6 (9.3)</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L</td>
<td>30.5 (7.1)</td>
<td>32.3 (16.9)</td>
<td>0.75</td>
<td>26.4 (5.0)</td>
<td>0.14</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>R</td>
<td>112.0 (29.7)</td>
<td>83.9 (26.3)</td>
<td>0.02</td>
<td>65.3 (22.6)</td>
<td>0.0002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L</td>
<td>88.4 (13.8)</td>
<td>91.3 (38.0)</td>
<td>0.82</td>
<td>81.6 (15.3)</td>
<td>0.28</td>
</tr>
<tr>
<td>Leg</td>
<td>10</td>
<td>R</td>
<td>26.3 (6.2)</td>
<td>21.9 (8.6)</td>
<td>0.13</td>
<td>18.1 (5.4)</td>
<td>0.0009</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L</td>
<td>20.8 (5.3)</td>
<td>19.3 (6.3)</td>
<td>0.50</td>
<td>20.2 (1.5)</td>
<td>0.74</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>R</td>
<td>79.4 (15.1)</td>
<td>62.1 (21.3)</td>
<td>0.02</td>
<td>54.6 (15.1)</td>
<td>0.0002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L</td>
<td>70.6 (15.5)</td>
<td>60.0 (14.8)</td>
<td>0.09</td>
<td>57.7 (7.3)</td>
<td>0.007</td>
</tr>
</tbody>
</table>

*Note: p values are based on Student’s t tests for samples of equal or unequal variances as appropriate.
between these two models supports the argument that the right side is affected by different factors from the left.

The motor assessment score was completed in 18 patients, ten with a left cerebral infarct and eight with a right infarct. In a multivariate analysis, there was no correlation between the severity of motor hemiplegia and tapping scores on the ipsilateral side once age, side, or group was taken into account.

**Discussion**

This study has confirmed our earlier observation that the ipsilateral right good side of patients with a single right-hemisphere infarct have slowed sensory-motor responses. The abnormality is present early after the stroke. On the left side ipsilateral to the infarct sensory-motor function was not significantly different from controls. We conclude therefore that this difference is due to a specific impairment of right cerebral function. The results appear to disprove our hypothesis which stated that both sides would be abnormal at stroke onset and that the left improves over time through practice and necessity. Whilst it could be argued that a left ipsilateral deficit could have been compensated for within 4—6 weeks we believe this would be unlikely. Disruption of ipsilateral motor fibres and associated motor weakness does not appear to be the cause of the sensory-motor deficit demonstrated. Furthermore the finding of no correlation between ipsilateral sensory-motor performance and severity of motor deficit raises the question of whether ipsilateral motor fibres have any useful function.

All patients were carefully screened from history and examination to ensure that only one cerebral infarct had occurred, although only 20% had a CT scan to confirm this. At the time, access to CT scanning was limited in our hospital and only a minority of people with a stroke had this examination performed, nor was it possible to justify access for research purposes. A CT scan is clearly a more accurate method of confirming the diagnosis of a single infarct, and lack of routine use of this instrument may lead to criticism. However, all of our subjects fulfilled clinical criteria for a single cerebral infarct and the chances of one or more silent infarcts in a contralateral hemisphere are equal regardless of side, thus any confounding influence should have been minimized.

Side-to-side tapping requires complex integration of sensory and motor function. Such movement involves all muscle groups, together with input from proprioceptors, skin receptors, vision, and labyrinthine mechanisms. In our two studies, impaired sensory-motor function of the good right side was observed in both the recent onset stroke group, and in those studied some time after the stroke. If these differences were due to a 'silent' infarct in the contralateral hemisphere they would be expected to occur randomly on either side. It is likely that such consistent differences in the right ipsilateral arm and leg in both stroke groups are real. A more detailed review of data from the latter group by a different analyst, however, concluded that there was a difference in this group also in the good left ipsilateral side but only in the lower limb at 30s. This was not reported in our previous paper [16]. The observed differences in the left leg of the group studied later after the stroke may be spurious but require further evaluation. Neither the slight age differences, nor the fact that MAS scores were lower in the group studied some time after the stroke accounted for these findings.

Longer simple reaction times in the good ipsilateral arm have been reported elsewhere only in subjects with right cerebral lesions, compared with subjects with left cerebral lesions and with normal controls. When arm function was assessed using coin-sorting tasks, and motor response in the leg by requesting the subject to depress a foot pedal after cueing to an auditory stimulus, the right side ipsilateral to the cerebral infarct performed significantly slower than in control subjects [14]. Those tested in the same way on the left side ipsilateral to a left infarct showed no impairment. In that study, five of eight subjects with a right cerebral lesion had clear evidence of unilateral neglect. In our study three of nine with a right-sided lesion failed to complete the letter cancellation test perfectly, omitting one or two letters, which could possibly be interpreted as left hemispatial neglect. They did complete clock drawing accurately. The abnormalities we detected in sensory-motor function were in the subject's right hemispace, not in left hemispace where unilateral neglect associated with a right hemispheric lesion would be expected. Even in the presence of more severe left neglect, the patient may well still be able to proceed with the testing method we used but this has not been validated. There is therefore no evidence to suggest that the abnormalities we found are due to disruption of hemispatial awareness. However it has been demonstrated that when contralateral unilateral neglect occurs, ipsilateral neglect does occur if the non-dominant hemisphere is damaged, but not in left-dominant hemispheric lesions [28].

In another study, subjects who were asked to depress a telegraph key with the index finger of the side ipsilateral to the infarct showed slower reaction times on both sides compared with controls, and the effect was much greater when the lesion was in the non-dominant hemisphere [15]. Whilst each study has used different instruments to test performance, our work does support the suggestion that the slowing of reaction times in the ipsilateral right limbs after stroke is likely to be due to a reduction of cerebral activation [14, 15]. Slow reaction times on the ipsilateral right side could also be interpreted as an attentional impairment, possibly due to frontal lobe (premotor planning) damage or disconnection.

In contrast with our findings is a recent report which appears to use a fairly similar instrument but found
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