Impaired Dexterity of the Ipsilateral Hand After Stroke and the Relationship to Cognitive Deficit

Alan Sunderland, PhD; Mark P. Bowers, BA; Stella-Marie Sluman, BA; David J. Wilcock, FRCR; Mark E. Ardron, FRCP

Background and Purpose—Previous research has reported impaired hand function on the “unaffected” side after stroke, but its incidence, origins, and impact on rehabilitation remain unclear. This study investigated whether impairment of ipsilateral dexterity is common early after middle cerebral artery stroke and explored the relationship to cognitive deficit.

Methods—Thirty patients within 1 month of an infarct involving the parietal or posterior frontal lobe (15 left and 15 right hemisphere) used the ipsilateral hand in tests that simulated everyday hand functions. Performance was compared with that of healthy age-matched controls using the same hand. Standardized tests were used to assess apraxia, visuospatial ability, and aphasias.

Results—All patients were able to complete the dexterity tests, but video analysis showed that performance was slow and clumsy compared with that of controls (P<0.001). Impairment was most severe after left hemisphere damage, and apraxia was a strong correlate of increased dexterity errors (P<0.01), whereas reduced ipsilateral grip strength correlated with slowing (P<0.05). The pattern of performance was different for patients with right hemisphere damage. Here there was no correlation between grip strength and slowing, while dexterity errors appeared to be due to visuospatial problems.

Conclusions—Subtle impairments in dexterity of the ipsilateral hand are common within 1 month of stroke. Ipsilateral sensorimotor losses may contribute to these impairments, but the major factor appears to be the presence of cognitive deficits affecting perception and control of action. The nature of these deficits varies with side of brain damage. The effect of impaired dexterity on functional outcome is not yet known. (Stroke. 1999;30:949-955.)

Key Words: cognition ■ motor activity ■ rehabilitation

The arm ipsilateral to a unilateral hemisphere stroke is often clinically described as being “unaffected,” but numerous studies have shown that ipsilateral function may not be normal.1–11 Reported impairments range from reductions in strength or speed of movement compared with age-matched normal control subjects4,6 through to the difficulty in producing complex action sequences, which is characteristic of apraxia.2

One might expect that if a patient does not have normal function on the ipsilateral side to help compensate for hemiparesis, this would be a significant obstacle to rehabilitation, but the clinical implications of these impairments remain unclear for a number of reasons. First, studies have varied greatly in sampling procedures and have seldom focused on well-defined groups of stroke patients at a fixed time after stroke. Second, the assessment procedures have often been far removed from everyday functioning. This has been most marked in studies of apraxia in which the most frequently used assessments are gesture imitation or pantomime of object use,2 and some studies have reported no relationship between such assessments and tests of manual skill.12,13 Finally, there has been inconsistency in dealing with the dependence of manual dexterity on cognitive functioning: some studies of ipsilateral performance have excluded patients who showed clinically obvious apraxia or visuospatial problems,4,10 while others have regarded these cognitive deficits as major causes of ipsilateral impairment.5,8

The current confusion over ipsilateral impairment is also reflected in a lack of integration in attempts to explain it. Some papers have sought an explanation in terms of disruption of ipsilateral motor pathways.4,10 Others have proposed an executive role for the dominant hemisphere in skilled motor control of either hand.8,9 There is also a large body of literature on apraxia,14,15 with theories about disruption of the cognitive representation of actions. Finally, some studies have shown that visuospatial impairments such as visual neglect can prevent accurate reaching with the ipsilateral arm.5,9 Each of these different accounts may have a role to play in understanding the motor performance of different patients with different sites of brain damage, but at present...
there is no consensus on what types of deficit might commonly lead to clinically significant impairment of ipsilateral hand function.

This article reports an intensive investigation of ipsilateral hand function in a common clinical group for whom, we hypothesized, it might be most severely affected. These were hemiplegic patients within a month of an infarct in the territory of the middle cerebral artery and with a lesion encroaching on the left or right parietal area. These patients might therefore have suffered the combined effects of loss of ipsilateral motor pathways plus deficits in motor performance secondary to apraxia or visuospatial deficits, which are strongly associated with damage to these cortical areas.16,17 The aims of the study were to assess the impact of ipsilateral deficits on simulations of everyday manual tasks and to discover to what extent these deficits were correlated with cognitive impairment.

### Subjects and Methods

#### Subjects

The study was approved by the Leicestershire Health Authority Ethics committee, and all subjects gave informed consent.

#### Stroke Patients

Two hundred fifty-four admissions to an acute stroke unit and a rehabilitation ward were screened for cases of a first unilateral cerebrovascular accident (CVA) with parietal lobe involvement. Diagnosis was based on medical notes and routine CT scan report. Thirty patients (12% of those screened) were recruited to the study. All had suffered middle cerebral artery stroke (7 hemorrhagic, 23 ischemic). Major reasons for exclusion from the study were evidence of a previous stroke or neurological disease (47%) or internal capsule infarcts with no apparent cortical involvement (9%). The CT scans were later reviewed by an experienced radiologist (D.J.W.) who was blind to patients’ test performances. Table 1 shows the resulting classification of brain lesions. Seven of the patients were judged to have had posterior frontal rather than parietal damage, and another 2 had signs of bilateral damage. These 9 patients were retained in the study but with attention given to these lesion variables in analysis of the results.

Patients were assessed when they were alert enough to cooperate with testing and it was practically possible to arrange a test session. Mean time after left hemisphere stroke (LCVA) was 17.2 days (range, 4 to 29) and after right hemisphere stroke (RCVA) 18.8 days (range, 1 to 31). All of the LCVA group were said to have been right-handed before their stroke. Two of the RCVA group had been right-handed before their stroke. Two of the RCVA group had been left-handed. All patients used the hand ipsilateral to their lesion for hand function. Two hundred fifty-four admissions to an acute stroke unit and a rehabilitation ward were screened for cases of a first unilateral cerebrovascular accident (CVA) with parietal lobe involvement.

### Extent of Infarct

<table>
<thead>
<tr>
<th>Extent of Infarct</th>
<th>LCVA Group (n=15)</th>
<th>RCVA Group (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parietal only</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Parietal+ occipital</td>
<td>1*</td>
<td>0</td>
</tr>
<tr>
<td>Parietal+ posterior frontal</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Parietal+ posterior frontal+ subcortical</td>
<td>4*</td>
<td>7</td>
</tr>
<tr>
<td>Parietal+ posterior frontal+ temporal</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Parietal+ posterior frontal+ temporal+ subcortical</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Posterior frontal only</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Posterior frontal+ subcortical</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

* One case in each category also had old, small, subcortical infarcts on the right but no clinical record or signs of previous stroke (see text).

#### TABLE 1. Brain Lesion Classification From CT Scans

#### TABLE 2. Demographic Data for Subject Groups

<table>
<thead>
<tr>
<th>Age, y</th>
<th>LCVA (n=15)</th>
<th>LC (n=18)</th>
<th>RCVA (n=15)</th>
<th>RC (n=16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD)</td>
<td>67 (13)</td>
<td>63 (12)</td>
<td>58 (11)</td>
<td>67 (11)</td>
</tr>
<tr>
<td>Range</td>
<td>40–83</td>
<td>42–86</td>
<td>33–76</td>
<td>47–83</td>
</tr>
</tbody>
</table>

#### Control Subjects

Healthy volunteers were drawn from a number of community groups. Inclusion criteria were no history of stroke or other neurological disease and no significant problems with hand movement due to arthritis, etc. They were similar to the stroke patients in age and years of education (see Table 2). All of them were right-handed, but equal numbers were assessed using their right hand (right-handed controls, RC) or the left hand (left-handed controls, LC). Groups were allocated alternately as healthy volunteers were recruited.

### Procedure

The subject sat in a chair/wheelchair at a standard height dining table. The stroke patients used the hand ipsilateral to lesion site throughout, while the healthy volunteers used either their left or right hand, depending on control group.

### Dexterity in Simulations of Everyday Tasks

These tests were performed on a rectangular baseboard measuring 75×31 cm, located on the tabletop and centered at body midline. A video camera was positioned above the baseboard and pointing downward to provide a record of task performance for later analysis. Instructions were given verbally and by demonstration. Subjects were asked to do the tasks “as quickly as you can,” and the subtests were always given in the following sequence.

#### Jhelsen Hand Function Test

This was designed to simulate common everyday manual skills.20 Subtests from this battery were given with slight modifications to the original published procedure in an attempt to increase reliability. These modifications were that all subtests began with the subject’s hand resting on the table at midline and each subtest was repeated 3 times. The subtests were the following: (1) Card Turning, the subject turned over each of 5 index cards (7.5 cm×12.5 cm) positioned evenly along the baseboard. (2) Picking Up Small Common Objects. Two paper clips, 2 bottle tops, and 2 coins were spaced along the baseboard with a tin can (13 cm high with a diameter of 9 cm) at the far end. The subject had to make cross-body movements to pick up each object in turn and drop it into the can.
A wooden board 2 cm thick was positioned at the back of the baseboard to provide the ledge used in the last 2 subtests. (3) Simulated Feeding (Bean Spooning). A teaspoon was used to spoon 5 kidney beans spaced along the back of the ledge into a can at the body midline. (4) Stacking Checkers. Four red wooden checkers spaced against the front of the ledge were stacked on top of the ledge.

Williams Doors
This test assesses dexterity in opening and closing small doors and has been shown to be a correlate of dependency in the elderly. Modifications to the original procedure were that only 1 door (15 × 23 cm) was used, fastened with a small bolt. The door was angled at 45° so that a clear view of manipulation of the bolt could be recorded by the overhead video camera. The opening edge of the door was at body midline, and it opened toward the ipsilateral side. The subject began with the hand on the baseboard at body midline and then had to unfasten the bolt and pull the door open until it reached 90°. The hand was then returned to the resting position before the subject reached up again to close the door. This procedure was repeated for 3 trials.

The videotapes of these dexterity tests were analyzed to obtain measures of speed and accuracy. Speed was measured by adding a 1/100-second timebase to the tape and reviewing it, frame by frame, to time the period from when the hand first moved from the rest position until completion of the trial. Accuracy was independently assessed by 2 raters (A.S. and M.B.), and 1 error was scored whenever there was not smooth, uncorrected execution of the component actions in each task. Thus, the maximum number of possible errors on each trial was 5 for card turning (1 for each card), 6 for picking up objects (1 for each object), 5 for spooning beans (1 for each bean), 4 for checkers (1 for each checker), and 2 for the Williams doors (1 for opening and 1 for closing). For the Jebsen subtests, the κ statistic was used for interrater agreement on errors ranged between “good” and “poor” (.64 to .33, P<0.001; extremes of 95% CI, 0.82 to 1.9), but on the Williams test it was below significance at .13 (95% CI, 0 to .28). However, total error scores calculated by averaging percentage error rates across all 5 dexterity tests showed high interrater reliability (r=0.81, P<0.001). A total error score was therefore used in the analysis. This was the average score for the 2 raters except where there was a disparity of >1 point on any trial when consensus was reached by joint viewing of the video.

Cognitive Performance

Apraxia Assessment
The subject was asked to produce each of 9 actions. The instructions were “Show me how you would use a...cup...key...ball...pencil...toothbrush...hammer,” and “Show me how you...salute...threaten with a fist...wave goodbye.” If performance was not perfect, the action was then demonstrated for the subject to imitate. Each item was scored by the examiner on 3-point scales: 0=unable/gross errors, 1=possible errors, and 2=correct.

Line Cancellation
A test for visual neglect in which the subject has to put a pencil mark through each 40 oblique lines scattered across an A4-size page.

Judgment of Line Orientation (Benton Lines Test) A test of visuospatial perception in which the subject has to identify short oblique lines that are at matching angles to the horizontal. The score is the number of correct matches with a correction for sex and age.

Token Test Parts I and V
A measure of receptive dysphasia in which the subject has to follow verbal commands by moving colored tokens.

Grip Strength
A Jamar dynamometer (model 00304) was used to measure grip strength over 3 trials (alternating with trials using the contralateral hand). The dynamometer was held in front of the subject so that a comfortable power grip was possible with the elbow slightly flexed.
Ipsilateral Dexterity and Cognitive Deficit

(F(1,57)=4.2, P<0.05). Figure 1 shows that this interaction arises because the LCVA patients were very much slower than their control group (t(31)=5.43, P<0.001), whereas those in the RCVA group were only slightly slower (t(29)=2.41, P=0.05).

Cognitive Performance

Dysphasia
Table 3 shows that, as expected, only the LCVA group was impaired compared with controls on the Token Test (stroke×side interaction, F(1,59)=26.0, P<0.001). Eight LCVA patients scored less than 20 of 32 and were definitely dysphasic. A direct contrast of LCVA and RCVA groups confirmed significantly poorer performance for the former (t(28)=5.33, P<0.001), whereas the LC and RC groups showed no significant difference (t(32)=.45, NS).

Apraxia
Table 3 shows that the LCVA group was impaired on both action to verbal command and on action imitation. On imitation, 10 of the 15 patients were poorer than any control. Two RCVA patients scored 15 of 18 on action imitation, but otherwise patients in this group were unimpaired, and the stroke×side interaction was highly significant (F(1,59)=14.8, P<0.001). A direct contrast of LCVA and RCVA groups confirmed significantly poorer performance for former (t(28)=3.88, P=0.001), whereas the LC and RC groups showed no significant difference (t(32)=.08, NS).

Visuospatial Deficits
Members of the RCVA group were impaired on line cancellation. No control subject or LCVA patient omitted any lines, whereas 4 RCVA patients made omissions. There were no cases of severe visual neglect, with the maximum number of omissions to the left being 2. On the Benton Lines test, 3 aphasic patients were unable to comprehend the test and were unassessable. Table 3 shows that for the remaining subjects there was a large main effect of stroke (F(1,56)=12.9, P=0.001), with the LCVA and RCVA groups showing a similar level of impairment.

Grip Strength
Table 3 shows that there was a wide range of grip strength within both stroke and control groups, probably reflecting normal variation with age and sex. Analysis of covariance confirmed that these variables were significant covariates of grip strength (P<0.001 for both). Mean scores corrected for age and sex were LCVA=19 kg, LC=34 kg, RCVA=22 kg, and RC=28 kg. The effect of stroke was highly significant (F(1,58)=17.6, P<0.001), but the trend toward differential impairment of the LCVA group did not reach significance (stroke×side interaction, F(1,58)=3.2, P=0.07).

Correlates of Impaired Everyday Dexterity
The relationship between impairment on the dexterity simulation tests and performance on other measures was analyzed both in terms of group regression analyses and individual case profiles.

Multiple Regression
For each of the stroke groups, multiple regression was used to investigate the extent to which the summary measures for speed and accuracy of dexterity could be predicted from...
variables of increasing complexity. The starting point was to look at the predictive value of age and sex alone and then to include in the equation simple motor function (grip strength). The cognitive measures of aphasia, apraxia, and visuospatial deficit were then compared as final additions to the regression equation. The results are illustrated in Figure 2, which shows the changes in $R^2$ as variables are entered into the equation with adjustment for the number of variables in the equation.\textsuperscript{27} Test for significance of change in F, $^*P<0.05$, $^{**}P<0.01$.

Figure 2 shows that for the RCVA group, visuospatial scores significantly predicted dexterity errors, whereas none of the other variables approached significance. There were no significant predictors of dexterity speed for this group. For the LCVA group, there was a different pattern of results for dexterity tests. The results are illustrated in Figure 2, which shows the changes in $R^2$ as variables are entered into the equation with adjustment for the number of variables in the equation at any point.\textsuperscript{27}

Table 4 shows that all LCVA patients who had abnormally high error rates on the dexterity tests were also apraxic on action imitation. However, 1 apraxic patient had normal error rate and speed on the dexterity tests. For the RCVA group, the relationship between high dexterity error rates and visuospatial problems was also imperfect: 1 patient was normal on dexterity but made an error on the cancellation test, and 1 patient had a high dexterity error rate but was normal on visuospatial tests.

All LCVA patients who had abnormally weak ipsilateral grip were slow on the dexterity tests, and in 2 of these cases this was an isolated deficit with normal error rates for the dexterity tests. No such relationship was apparent for the RCVA group, despite 5 patients whose grip strength fell below the normal range.

**Additional Neurological Variables**

Degree of paresis of the contralateral arm as measured by the Extended Motricity Index was not a correlate of ipsilateral dexterity speed or accuracy for the LCVA or RCVA group ($r<.3$, NS, for all). A comparison of the 7 patients judged to have posterior frontal and no parietal damage with the remainder showed no significant differences for dexterity speed or errors (Mann Whitney $U$ tests, $P>0.1$ for all).

**Discussion**

This study has shown that within a month of an infarct involving the parietal and/or posterior frontal lobe, the majority of left hemisphere patients and a smaller proportion of right hemisphere patients have impaired dexterity of the
Ipsilateral Dexterity and Cognitive Deficit

Ipsilateral hand. This result is in agreement with a number of previous studies,\textsuperscript{1,3,10,11} that have reported impaired dexterity in less tightly defined groups of stroke patients. The impairment is typically a subtle one. There was slowing and clumsiness, but no patient in this study was unable to eventually complete these tests that simulated everyday dexterity.

Our results indicate that these dexterity problems are probably due to cognitive deficits affecting perception and control of action. Others\textsuperscript{4,10} have suggested that ipsilateral sensorimotor losses might be important for hand function. We found that ipsilateral grip strength was reduced, but this coexisted with normal dexterity for patients with right hemisphere damage, indicating that this mild weakness was not a sufficient cause for dexterity problems. We did not measure strength in ipsilateral proximal muscles that might be more sufficient cause for dexterity problems. We showed that severity of impairment of action imitation after left hemisphere damage was predictive of the error rate on dexterity tasks. Also, the problems in orienting the spoon that were observed in the bean-spooning task strongly suggested an inability to remember or perceive the correct action. On the other hand, the association between impaired action imitation and frequent dexterity errors was not perfect. In particular, 1 patient who was severely impaired on action imitation was at the normal level on all dexterity tasks. On balance, we feel that the multiple regression results plus the video error analysis argue strongly for the validity of the concept of ideomotor apraxia as a major cause of dexterity problems after left hemisphere damage, and that dissociations with action imitation performance in some cases may arise from patient variability over time or the limitations of our methods of assessment.

A puzzle for researchers in this area has been anecdotal evidence that apraxic patients behave normally in everyday settings. So, De Renzi et al\textsuperscript{2} comment, “The most striking dissociation found in apraxic patients is their inability to perform on command an action which is perfectly executed when roused by a congruent situation: eg, waving goodbye when the doctor is leaving.” Furthermore, some studies have found no correlation between scores on action imitation tests and performance on tests of motor skill, such as pegboard performance,\textsuperscript{7} repetitive motor sequence learning,\textsuperscript{8} or reaction time.\textsuperscript{13} The present study offers a resolution to these problems. First, a general observation was that all our patients were finally successful in completing the dexterity tasks and that their clumsiness was often only evident from detailed video analysis. The apparent normality of apraxic patients in everyday settings may therefore simply reflect the subtle nature of their deficit, which becomes clinically obvious only under the controlled conditions of an action imitation test. Second, the failure of some studies to find correlations with tests of motor skill can be explained by the reliance of those studies on measures of speed rather than error. An important finding in the present study was that apraxia appeared to have a consistent effect on error rate but not speed. This is in agreement with other studies\textsuperscript{7,8} which have found that apraxic patients have increased error rates on motor tasks and that apraxic movement is characterized by spatial disturbance rather than by abnormalities in the timing of movements.\textsuperscript{15}

Slowing After Left Hemisphere Damage
Patients with left hemisphere damage were often slow in their performance of the dexterity tasks. This is consistent with a body of research showing that left hemisphere damage causes greater problems than right-sided damage on tasks which require rapid or accurately timed responses\textsuperscript{5,8} and has led to the hypothesis that the left hemisphere is dominant for the control of preprogrammed, ballistic movements.\textsuperscript{9} A new finding from this study was the correlation between slowing and weakness of ipsilateral grip. The absence of any such correlation for the LCVA group suggested that weakness was not in itself the cause of slowing. Perhaps the correlation for the LCVA group was due to the anatomic proximity in the left hemisphere of areas that are the origin of ipsilateral corticospinal pathways and areas that may be important for ballistic control. The anatomic data in this study was not detailed enough to test this hypothesis. What is certain from this and previous studies is that the slowing seen after left hemisphere damage cannot be attributed to apraxia and, therefore, that there is a separate underlying deficit which affects dexterity in this group.

Visuospatial Deficits
The multiple regression analysis showed that visuospatial deficits (omissions on line cancellation or problems in judgment of spatial orientation) were predictive of dexterity error rates for the RCVA group. Also, the errors observed on the dexterity tasks suggested problems of visual attention or spatial judgment. There was no such association in the LCVA group.
group despite equivalent performance on the Benton Lines test. However, dysphasia or dyslexia can impair comprehension of this task. Benton et al.\(^5\) suggest that performance may not reflect visuospatial ability when patients are “aphasic and confused,” and there was no evidence of visuospatial problems within the LCVA group on the line cancellation test, which all patients could readily comprehend. In summary, although these results are not completely clear cut, they are consistent with the proposal that visuospatial deficits were the major cause of dexterity errors after right but not left hemisphere damage. This is in agreement with previous research indicating reduction in the accuracy of rapid reach- 

**Clinical Implications**

The presence of subtle losses in dexterity particularly after left hemisphere stroke has clinical implications. There has been an understandable tendency to ascribe clumsiness in use of the ipsilateral hand to normal difficulty in learning to use the nondominant hand for most tasks, whereas this study makes it clear that these patients are also struggling against high-level deficits in skilled motor control. This may have an impact at 2 levels: first, it may slow or reduce the acquisition of left hand skills to compensate for hemiplegia, and second, these deficits may impair relearning of control of a hemiparetic arm. A follow-up study of patients at 6 months after stroke will attempt to describe the impact of these obstacles to recovery and the potential for therapeutic intervention.

**Acknowledgments**

This research was supported by the British Stroke Association.

**References**

Impaired Dexterity of the Ipsilateral Hand After Stroke and the Relationship to Cognitive Deficit
Alan Sunderland, Mark P. Bowers, Stella-Marie Sluman, David J. Wilcock and Mark E. Ardron

Stroke. 1999;30:949-955
doi: 10.1161/01.STR.30.5.949
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1999 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/30/5/949

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/