OBJECTIVE. We determined (1) whether active range of motion (AROM) of shoulder flexion and wrist extension measured at the initial therapy evaluation in the acute hospital predicted upper-extremity (UE) motor function 3 mo after stroke and (2) whether the presence of nonmotor impairments influenced this prediction.

METHOD. We collected AROM data from 50 people with stroke during their initial acute hospital therapy evaluation and UE motor function data 3 mo later. Multiple regression techniques determined the predictive ability of initial AROM on later UE motor function.

RESULTS. Initial AROM explained 28% of the variance in UE motor function 3 mo poststroke. Nonmotor deficits did not contribute to the variance.

CONCLUSION. Compared with later AROM measurements, initial values did not adequately predict UE motor function 3 mo after stroke. Clinicians should use caution when informing clients of UE functional prognosis in the early days after stroke.


The ability to predict upper-extremity (UE) motor function poststroke is important for planning rehabilitation service needs and patient discharge. For the purposes of this article, we define UE motor function as the capacity to use the UE for skilled actions, such as reaching, grasping, and manipulating objects used in daily life. UE motor function is frequently determined by having the patient perform various skilled actions in the clinic, by patient self-report of performance, or both. Prediction of future UE motor function can provide prognostic information about functional motor activities achievable by the patient at the end of the rehabilitation process. This information assists clinicians with selecting appropriate interventions, setting goals, and educating patients with stroke and their caregivers.

A handful of studies have investigated the predictive validity of various assessment tools on UE motor function (De Weerdt, Lincoln, & Harrison, 1987; Feys et al., 2000; Katrak et al., 1998; Kwakkel, Kollen, van der Grond, & Prevo, 2003; Loewen & Anderson, 1990; Smania et al., 2007). Interpretation of the collective data is challenged by the use of various predictor variables, outcome variables, prediction time points, and analytical models. One general conclusion can be drawn from these data: People with less severe early impairments, particularly less motor loss (i.e., milder paresis), achieve better UE motor function later. This general conclusion, however, does not provide the depth of information necessary to inform the clinician about potential level of independence, achievable activities, appropriate interventions, goal setting, or individual and caregiver education.
In a sample of people with relatively pure motor hemiparesis, we recently showed that the simple measurement of UE active range of motion (AROM) 3 wk after stroke was predictive of UE motor function 3 mo after stroke (Beebe & Lang, 2009a). AROM measurements of shoulder flexion and middle-finger flexion and extension taken an average of 3 wk after stroke explained 71% of the variance in UE motor function 3 mo later, when UE motor function was measured with a comprehensive test battery quantifying the motor capacity of the affected UE. An AROM measurement can be conceptually considered as a measure of the ability to voluntarily activate the spinal motor neuron pools that move a given segment and thus as an index of paretic severity (Hislop & Montgomery, 2002). Because AROM measures are predictive of later UE motor function, they may be a quick, inexpensive way to obtain prognostic data regarding UE motor function near the end of the rehabilitation process.

Two issues need further refinement, however: (1) the timing of the measurements and (2) the sample of people in which they are evaluated. With respect to the first issue, people with stroke typically enter the rehabilitation system within the first day or first few days after stroke, not at 3 wk. With respect to the second issue, people with stroke experience deficits in nonmotor domains, not just in the motor and somatosensory domains, as previously tested (Beebe & Lang, 2008, 2009a; Lang & Beebe, 2007). Of the many nonmotor deficits seen after stroke, three are particularly common and debilitating: (1) aphasia, (2) neglect, and (3) cognitive deficits (Cherney & Halper, 2001; Wade, Hewer, David, & Enderby, 1986; Zinn, Bosworth, Hoenig, & Swartzwelder, 2007). The purpose of this study, therefore, was to determine whether AROM at a proximal and distal joint taken at the initial therapy evaluation in the acute hospital was predictive of UE motor function 3 mo later in people with deficits in motor and nonmotor domains. We chose shoulder flexion and wrist extension as the representative proximal and distal movement assessed in this study because they had high zero-order correlations with function in earlier studies (Lang & Beebe, 2007) and were easy to measure systematically in the acute care hospital. Our hypotheses were that (1) initial AROM measurements of shoulder flexion and wrist extension would predict UE motor function 3 mo after stroke and (2) nonmotor deficits of aphasia, neglect, and cognitive deficits would not influence the predictive relationship between initial AROM measurement and UE motor function 3 mo after stroke.

Method

We used a prospective cohort design to evaluate the relationship between AROM measurements taken at the initial therapy evaluation at the acute hospital and UE motor function 3 mo later. Fifty participants provided data on poststroke recovery. Participants were recruited from the Cognitive Rehabilitation Research Group and Brain Recovery Core Registries. The registries are comprehensive databases of nearly all people admitted to Barnes–Jewish Hospital with stroke; the only criterion to be entered into the registries is a diagnosis of stroke or transient ischemic attack. All participants in the registries gave informed consent after admission to the hospital, and the study was approved by the Washington University Human Research Protection Office.

All eligible participants were contacted first by means of written correspondence, which was followed by a recruitment phone call. Inclusion criteria for participation in the study were as follows:

- Adults with clinical diagnosis of ischemic or hemorrhagic stroke meeting International Classification of Diseases, 9th Revision (Centers for Disease Control and Prevention, 1998) criteria
- Data from initial physical and occupational therapy assessments ≤7 days after stroke
- Unilateral UE paresis, as indicated by a score of 1–4 on the National Institutes of Health Stroke Scale (NIHSS) Arm item
- Persistent deficits after stroke, as indicated by a total NIHSS score of ≥2
- Ability to follow two-step commands.

Exclusion criteria for participation in the study were history or current diagnoses of any other neurological or psychiatric condition, including previous stroke; current orthopedic condition involving the affected UE; recent fall or UE surgery involving the affected UE; or unavailable for 3-mo follow-up assessment.

Acute-care therapists performed the initial physical and occupational therapy evaluations just after participants’ admission to the hospital; we obtained data from these evaluations by electronic medical record review. The data included (1) two independent variables, AROM measurements of shoulder flexion and wrist extension of the affected, contralateral side using goniometric techniques (Gajdosik & Bohannon, 1987; Norkin & White, 2009); (2) the UE Motricity Index (Collin & Wade, 1990) score on the affected side, used as an additional descriptor of overall UE motor impairment; and (3) the Short Blessed Test (Katzman et al., 1983), an executive-function screening tool administered at the
initial occupational therapy evaluation, used to index the presence and severity of cognitive deficits. We trained acute-care therapists to use standardized procedures for these and other measures. The therapists attended four training sessions, and we monitored procedures monthly. Other descriptive items recorded from the medical record to characterize the sample include total scores from the NIHSS, used as a descriptor of overall stroke severity (Brott et al., 1989), and individual NIHSS item scores for aphasia and neglect, used as indexes of the presence and severity of stroke-related deficits in the language and attention domains. The NIHSS was administered by trained nurse practitioners on the acute hospital stroke service.

Three months after stroke onset, we assessed each participant’s UE motor function in the laboratory. The dependent variable of UE motor function was assessed using a comprehensive battery, similar to the one described in Beebe and Lang (2008, 2009a; Lang & Beebe, 2007). We used a battery instead of a single measure because no single tool adequately captures the breadth of UE motor function (Beebe & Lang, 2009b; Wade, Langton-Hewer, Wood, Skilbeck, & Ismail, 1983). A participant’s results from the battery provide a thorough picture of what functional activities can be accomplished with the affected UE, including aspects of strength, coordination, functional ROM, and the participant’s perception of difficulty using the UE. The measures in this battery consisted of the following assessments:

- Grip strength, assessed with a hand-held dynamometer (Mathiowetz, Kashman, et al., 1985; Schmidt & Toews, 1970) and reported as strength of the affected side as a percentage of the unaffected side
- The Nine Hole Peg Test (Mathiowetz, Weber, Kashman, & Volland, 1985), reported as pegs placed or removed per second

We synthesized the results of the test battery to yield a single measure of UE motor function for each participant using principal components analysis (Lang & Beebe, 2007; Ward, Brown, Thompson, & Frackowiak, 2003a, 2003b), a statistical methodology in which the dimensionality of a data set is reduced to one or a few scores. All battery scores loaded onto the first principal component (eigenvalue = 2.96), explaining 74% of the variance in the test scores, and no other components yielded eigenvalues >0.6. According to Kaiser’s criterion, only eigenvalues >1 are retained (Field, 2009); thus, we used the first principal component as a score of relative magnitude of UE motor function among participants. The UE motor function score for each participant can be conceptualized as a statistical composite of that participant’s individual scores on the battery.

**Statistical Analysis**

We used SPSS Version 17.0 (SPSS Inc., Chicago) for analyses; the two-tailed significance criterion was $p < .05$. Normal distribution of variables was confirmed, and descriptive statistics were generated appropriately. Pearson product–moment correlations were used to evaluate relationships between initial assessment measures and UE function. For the initial assessment measures, we entered Pearson product–moment correlations $>0.25$ into a regression model to determine whether they were predictive of UE motor function 3 mo later. Coefficients $<0.25$ were excluded because they would contribute $<6\%$ of the total variance. We evaluated model diagnostics and performed a sensitivity analysis on all outliers. All participants were included in the regression model.

**Results**

Sample characteristics and measurement time points are provided in Table 1. Descriptive statistics for the impairment measurements at the time of initial therapy evaluation and the UE motor function measurements 3 mo after stroke are provided in Table 2. Although the sample varied considerably on all measures collected (see ranges), the sample can be characterized as people with mild to moderate motor and functional deficits after stroke. Aphasia and neglect ranged from absent to severe, and cognitive deficits were generally mild across the sample.

Our first hypothesis was that AROM measurements from the initial therapy evaluation would predict UE motor function 3 mo later. Pearson product–moment coefficients indicated that shoulder flexion and wrist extension AROM measurements were moderately correlated with UE motor function 3 mo after stroke (Table 3). A linear regression model, including both shoulder and wrist initial AROM, explained 28% of the variance in UE motor function 3 mo after stroke, $F(2, 46) = 8.712, p = .001$.

Our second hypothesis was that nonmotor covariates would not influence the predictive relationship between
initial AROM and UE motor function 3 mo later. Correlation coefficients quantifying the relationship between UE motor function and aphasia, neglect, and cognitive deficit were low (Table 3). Only neglect ($r = -.26$) exceeded our threshold for entry into the regression model and was stepwise entered into the model after AROM. This covariate did not significantly contribute to UE variance when entered stepwise and was excluded from the final model.

**Discussion**

We found that AROM measurements of the shoulder and wrist taken at the initial therapy evaluation in the acute hospital were weak predictors of UE motor function 3 mo later, predicting only 28% of the variance in UE scores. Contrary to our first hypothesis, the prognostic information provided by AROM measures within a few days after stroke was substantially less than the prognostic information provided at 3 wk after stroke (71%: Beebe & Lang, 2009a). In support of our second hypothesis, we found that the presence of aphasia, neglect, and cognitive deficits did not influence the predictive ability of initial AROM on UE motor function 3 mo later.

The difference between the explained variances an average of 2 days after stroke and 3 wk after stroke, as in our previous work (see the introduction), most likely involves two key prognostic factors, (1) initial severity of stroke and (2) rate of change of severity or rate of UE motor recovery (Kwakkel, Kollen, & Lindeman, 2004; Teasell, Foley, Salter, & Jutai, 2008). The 2-day AROM measures used in this study reflect the initial severity of motor deficits poststroke but not the rate of change of severity, because it is too early to see much change over time. In contrast, measurement at the 3-wk time point would reflect some combination of the initial motor deficit and the recovery that would occur by then. When viewed from this perspective, the current data, along with our previous data, match the general consensus that functional motor recovery poststroke can be reasonably predicted by 3–4 wk after stroke (Duncan, Lai, & Keighley, 2000; Jørgensen et al., 1995; Kwakkel et al., 2003, 2004; Loewen & Anderson, 1990; Olsen, 1990). Three-week measurements, but not 2-day measurements, therefore, can provide specific information about motor capacity for functional UE activities such as dressing, bathing, and feeding that will be achievable late after stroke. Measurements 2 days after stroke are insufficient to provide the depth of information about 3-mo UE motor function needed to make judgments about functional activities achievable later.

The weak predictive relationship found here contrasts with a recently published report that used measures and time points similar to ours (Nijland, van Wegen, Harmeling-van der Wel, & Kwakkel, 2010). Using logistic regression, Nijland et al. (2010) found a high

Table 1. Participant Characteristics ($N = 50$)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
<th>Range or %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, yr ($SD$)</td>
<td>59 (15)</td>
<td>19–87</td>
</tr>
<tr>
<td>Gender, $n$</td>
<td>26 M, 24 F</td>
<td>—</td>
</tr>
<tr>
<td>UE affected, $n$</td>
<td>22 L, 28 R</td>
<td>—</td>
</tr>
<tr>
<td>Dominant UE affected, $n$</td>
<td>27</td>
<td>54%</td>
</tr>
<tr>
<td>NIHSS total score$^a$ ($SD$)</td>
<td>8 (6)</td>
<td>2–31</td>
</tr>
<tr>
<td>Ischemic stroke, $n$</td>
<td>44</td>
<td>88%</td>
</tr>
<tr>
<td>Race/ethnicity, $n$</td>
<td>29 AA, 20 W, 1 AS</td>
<td>58% AA, 40% W, 2% AS</td>
</tr>
<tr>
<td>Time from stroke to initial assessment, days ($SD$)</td>
<td>2 (1)</td>
<td>0–5</td>
</tr>
<tr>
<td>Time from stroke to follow-up assessment, days ($SD$)</td>
<td>101 (13)</td>
<td>77–129</td>
</tr>
</tbody>
</table>

*Note. AA = African-American; AS = Asian; F = female; M = male; L = left; R = right; NIHSS = National Institutes of Health Stroke Scale, $SD$ = standard deviation; UE = upper extremity; W = White.

$^a$Range $= 0–42$, with higher scores indicating greater stroke severity.

Table 2. Initial and 3-Mo Follow-Up Measurements

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean ($SD$)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial assessment: motor and nonmotor impairments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AROM shoulder flexion; degrees</td>
<td>88 (59)</td>
<td>0–140</td>
</tr>
<tr>
<td>AROM wrist extension; degrees</td>
<td>24 (32)</td>
<td>−30–65</td>
</tr>
<tr>
<td>Motricity index</td>
<td>56 (32)</td>
<td>0–100</td>
</tr>
<tr>
<td>Short Blessed Test</td>
<td>6 (5)</td>
<td>0–22</td>
</tr>
<tr>
<td>NIHSS Aphasia</td>
<td>Median = 0</td>
<td>0–3</td>
</tr>
<tr>
<td>NIHSS Neglect</td>
<td>Median = 0</td>
<td>0–2</td>
</tr>
<tr>
<td>3-mo assessment: UE battery scores and overall motor function score</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grip strength; % of unaffected side</td>
<td>57 (36)</td>
<td>0–122</td>
</tr>
<tr>
<td>Nine Hole Peg Test; pegs/s</td>
<td>1.37 (1)</td>
<td>0–4</td>
</tr>
<tr>
<td>ARAT</td>
<td>42 (18)</td>
<td>0–57</td>
</tr>
<tr>
<td>Stroke Impact Scale: Hand function</td>
<td>58 (31)</td>
<td>0–100</td>
</tr>
<tr>
<td>UE motor function score$^a$</td>
<td>0 (1)</td>
<td>−2–1</td>
</tr>
</tbody>
</table>

*Note. ARAT = Action Research Arm Test (score range $= 0–57$, with higher scores indicating better performance); AROM = active range of motion; NIHSS = National Institutes of Health Stroke Scale; $SD$ = standard deviation; UE = upper extremity.

$^a$Calculated as a standardized $z$ score for each person from the weighted, linear coefficients of the first principal component.
probability (0.98) of regaining dexterity, as defined by an ARAT score of ≥10, when shoulder abduction and finger extension were present at the initial assessment after stroke. Their independent variables (presence of shoulder and finger movement) are conceptually similar to ours (amount of shoulder and wrist movement) in that each variable is an index of the severity of paresis poststroke. Differences in results are probably not explained by the specific independent variable selected, because the severity of paresis is strongly correlated across UE segments poststroke (Beebe & Lang, 2008, 2009a; Lang & Beebe, 2007). Instead, the difference in results may be explained by the logistic regression and dichotomization of the dependent variable. Defining return of dexterity as an ARAT score between 10 and 57 (maximum score on this measure) represents a wide range of UE functional capabilities. People scoring at the low end of this range, 10 or slightly above, would have little functional use of the UE. Such use would be observed as an inability to elevate the arm, an inability to perform tip-to-tip pinch, and moderate assistance required from the unaffected arm for functional gross grasp. People at the higher end of this range, ≥40, would demonstrate independence with activities of daily living and likely require only a little additional time for fine motor activities. Dividing the dependent variable into two broad categories (scores of <10 and ≥10 on the ARAT), unfortunately, does not provide sufficient information about eventual activity achievement to guide clinical decisions about intervention choice, goal setting, and client and caregiver education.

An important limitation of this study is that our sample consisted of stroke survivors who could be reached for follow-up and who could return to the laboratory for the follow-up testing. The sample did not include people with the most severe stroke, because they may have died, were residents in extended-care facilities, were not reachable at their listed phone numbers, or were unwilling to participate. Results presented here, therefore, are most appropriately generalized to stroke survivors living in the community at 3 mo poststroke.

A second limitation was the rather insensitive measures used to index the covariates. More sensitive tools to quantify aphasia, neglect, and cognitive deficits, such as the Boston Naming Test (Lansing, Ivnik, Cullum, & Randolph, 1999), Unstructured Mesulam Test (Mesulam, 1985), and Trail Making Test–B (Reitan & Wolfson, 1995), might possibly have identified at least minimal influences on later UE motor function. Unfortunately, initial evaluation data using these more sensitive tools (or similar ones) were not consistently available for each person serviced by the acute stroke therapists. We speculate that deficits in nonmotor domains do not influence the capacity to use the UE for functional activities (as tested in the laboratory in this study) but might instead influence actual use of the UE or participation in the real world. This speculation requires further study with different dependent variables.

In summary, AROM measures taken at the time of initial therapy evaluation in the acute-care hospital were weakly predictive of later UE motor function. Further work is needed to determine the earliest and most effective time point poststroke to obtain prognostic information about functional UE activities the patient with stroke will be able to achieve by the end of the rehabilitation process.

Implications for Occupational Therapy Practice

The results of this study have the following implications for occupational therapy practice:

- AROM measures, an index of paretic severity, provide good prognostic information about UE function at 3 wk after stroke, but not at 2 to 3 days after stroke.
- Stroke-induced deficits of aphasia, neglect, and cognition measured in the first 2 to 3 days after insult did not influence the capacity to use the UE for function 3 mo later.
- Clinicians should use caution when informing clients of UE functional prognosis in the early days after stroke. ▲

Acknowledgments

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Table 3. Pearson Product–Moment Correlations Between Initial Impairment and 3-Mo Upper-Extremity Motor Function Scores

<table>
<thead>
<tr>
<th>Measure</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>AROM</td>
<td></td>
</tr>
<tr>
<td>Shoulder flexion</td>
<td>.51*</td>
</tr>
<tr>
<td>Wrist extension</td>
<td>.46*</td>
</tr>
<tr>
<td>Short Blessed Test</td>
<td>-.06</td>
</tr>
<tr>
<td>NIHSS</td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>-.07</td>
</tr>
<tr>
<td>Neglect</td>
<td>-.26</td>
</tr>
</tbody>
</table>

Note. AROM = active range of motion; NIHSS = National Institutes of Health Stroke Scale.

* p < .05.
to Catherine E. Lang and NIH Grant UL1RR024992 and subaward TL1RR024995 to Eliza M. Prager.

References


Cherney, L. R., & Halper, A. S. (2001). Unilateral visual neglect to Catherine E. Lang and NIH Grant UL1RR024992 and subaward TL1RR024995 to Eliza M. Prager.


