Deficits of Reaching in Subjects With Left Hemiparesis: A Pilot Study

Catherine A. Trombly

Key Words: cerebrovascular disorders • electromyography • reaching

Therapy to restore functional movement of stroke patients is based on assumptions about the deficits that occur in motor control as a result of stroke. Success has been limited, perhaps as a result of insufficient information concerning the characteristics of movement after stroke. In this pilot study, the Waterloo Spatial Motion Analysis Recording Technique (WATSMART™), an optoelectric motion analysis system, was used with surface electromyography to measure voluntary reaching in the impaired and unimpaired arms of 5 subjects with left hemiparesis. The ability to reach in a smooth coordinated way was significantly poorer in the impaired arms than in the unimpaired arms, for which scores were essentially normal. The patients were less able to activate the muscles of the impaired arm and, as a result, used a greater percentage of the maximum activity they could generate to complete the resisted reaching task. The electromyographic differences between arms, however, did not reach significance.

The results corroborate previous findings and show that movement deficits of particular patients can be diagnosed precisely with kinematic analysis and electromyography. If greater precision in diagnosis were available clinically, more effective therapy might be developed.

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This article was accepted for publication May 12, 1992.
Figure 1. Examples of velocity profiles and acceleration traces. Note the smoothness of the acceleration and deceleration phases of the velocity profile, plotted in relative time, for the unimpaired arm. The acceleration trace, plotted in actual time, crosses the zero line once; this defines a continuous movement. The graphs for the impaired arm describe a discontinuous movement.

Discontinuous movements, or nonprogrammed movements, are characterized by steps resembling small continuous movements chained together (see Figure 1). At the end of each step, there is opportunity for sensory feedback to guide the movement (Brooks, 1986). Other researchers have referred to these steps as movement elements (Arbib, 1985; Morasso, 1981, 1983; von Hofsten, 1979) or movement units (Petters & Todd, 1987).

Unimpaired reaching has been studied extensively through kinematic analysis, and several theoretical models of control of reaching have been advanced over the past decade (Hogan, 1984; Hollerbach & Atkeson, 1987; Hollerbach & Flash, 1982; Morasso, 1981, 1983; Soechting & Lacquaniti, 1981). All agreed that when unimpaired humans or monkeys reach to a large target, they use one continuous movement that covers most of the distance to the target (Georgopoulos, 1986). In other words, they use an open-loop or programmed strategy to approach the target. This strategy develops gradually during infancy and childhood (Georgopoulos, 1986) and through learning in adult animals (Brooks & Watts, 1988).

The one-peaked velocity profile is further characterized by its shape, which is symmetrical, that is, peak velocity is located at approximately 50% of total movement time (Arbib, 1985; Hogan, 1984; Hollerbach & Flash, 1982; Morasso, 1983; Soechting & Lacquaniti, 1981); or is slightly left-shifted, that is, peak velocity is located at approximately 33% of total movement time (Beggs & Howarth, 1972; Bullock & Grossberg, 1988). In repeated trials of unimpaired reaching to point to a large target, a large percentage of the trials will exhibit the single-peaked velocity profile and the amplitude of peak velocity and overall movement time will be consistent (von Hofsten, 1979). The time to peak velocity in a simple reaching task is relatively invariant over trials (Wing & Miller, 1984).

Few empirical studies have examined the kinematics of reaching by patients after stroke. Reaching to point at a target by such subjects using their unimpaired arms has
been compared with reaching by age-matched controls by Fisk and Goodale (1988). The subjects who had had right cerebrovascular accidents were slower to initiate reach, presumably because of their need to visuospatially locate the target. However, once moving, their movement time, amplitude of peak velocity, and temporal pattern of acceleration and deceleration (velocity profile) were similar to those of the control subjects. These results indicated that the basic programmed strategy of goal-directed reaching was preserved in the unimpaired arms of these subjects. Lough et al. (1984) examined the progress of recovery of forward, gravity-eliminated reaching of the impaired arm of a 26-year-old man with left hemiparesis. They identified two deficits: decreased amplitude of peak velocity and increased movement time, both of which improved with recovery. Additionally, movement discontinuity was noted soon after the stroke, but movement toward target became more direct as recovery progressed.

Knowledge of whether stroke patients use open-loop (preplanned) or closed-loop (guided) strategy would direct therapy differently. For example, if stroke patients used a discontinuous strategy when normally a continuous strategy would be expected, therapy to restore voluntary movement would most appropriately involve practice to relearn the correct strategy or to restore deficit tactics such as muscle activation.

The first question addressed in this study was whether strategies, as measured by velocity and acceleration profiles, differ between the impaired and unimpaired arms of subjects with left hemiparesis. The unimpaired arm was expected to be within normal limits, as described by Fisk and Goodale (1988), and thus to exhibit a continuous, or open-loop, strategy. I expected that the impaired arm would exhibit a discontinuous, or closed-loop, strategy. The second question was whether those tactics of reaching measured in this study by electromyography, that is, level of muscle activation and coactivation, differ between arms. I hypothesized that (a) the impaired arm would generate significantly less muscle activity during maximum voluntary contraction and a greater percentage of maximum voluntary contraction during reaching than the unimpaired arm and (b) the coactivation indexes of the impaired arm would reflect unnatural cocontraction secondary to spasticity or to abnormal synergies associated with hemiplegia as described by Twitchell (1951) and Brunnstrom (1970).

Method

Subjects

Subjects with left hemiparesis were recruited after discharge from three major rehabilitation centers in the metropolitan Boston area. Five subjects consented to participate. No subject showed left-sided neglect, as measured by the Schenkenberg Line Bisection Test (Schenkenberg, Bradford, & Ajax, 1980), therefore none were expected to have difficulty in spatially locating the targets. Additionally, no subject was apractic, as reported by occupational therapists. At the time of the last visit reported here, Subjects 1 and 2 showed mild deficits of awareness of limb position (15/18 and 17/18, respectively), as measured by a test developed by Leo and Soderberg (1981), and Subject 1 had mild spasticity (8.5/48), as measured by the Modified Ashworth Scale of Muscle Spasticity (Bohannon & Smith, 1987). Other characteristics are listed in Table 1.

Instrumentation

The anterior deltoid, biceps, clavicular portion of the pectoralis major, and lateral head of the triceps were monitored with surface electromyography. The signals from the muscles were amplified with a Grass Model 7 poly-

Table 1
Patients' Characteristics

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Sex</th>
<th>Hand Dominance</th>
<th>FMIMT(^{1,2})</th>
<th>Number of Weeks Post Cerebrovascular Accident(^{3})</th>
<th>Site and Type of Lesion(^{4})</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42</td>
<td>F</td>
<td>R</td>
<td>19.0</td>
<td>29.5</td>
<td>Hemorrhage of left lenticular nucleus and the right corona radiata.</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>F</td>
<td>R</td>
<td>28.5</td>
<td>20.3</td>
<td>Lacunar infarct in left lenticular nucleus.</td>
</tr>
<tr>
<td>3</td>
<td>64</td>
<td>F</td>
<td>R</td>
<td>23.0</td>
<td>20.0</td>
<td>Lacunar infarct in the right internal capsule and cerebral atrophy.</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>F</td>
<td>R</td>
<td>36.0</td>
<td>29.0</td>
<td>Right(^1) cerebellar hemispheric infarct with brain stem involvement.</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>M</td>
<td>L</td>
<td>36.0</td>
<td>16.5</td>
<td>Left(^1) cerebellar hemispheric infarct.</td>
</tr>
</tbody>
</table>

Note: F = female, M = male, R = right, L = left.

\(^{1}\)At time of last testing period.\(^{2}\)The Fugl-Meyer Motor Function Test (Fugl-Meyer et al., 1975) quantifies motor control of hemiplegic limbs, a score of 36 in the Upper Limb subtest, the highest obtainable on this scale, is interpreted as meaning that the subject has control of isolated movement of the proximal upper extremity.\(^{3}\)As recorded in the medical record.\(^{4}\)It is not known whether the hemispheric designation represents a real difference or a charting error; however, both subjects manifested left hemiparesis.

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graph and collected with WATSCOPE™ analog to digital converter and data acquisition software at a sampling rate of 500 Hz. The digitized signals were stored with a FM tape back-up system for off-line processing and analysis with ANAPAC waveform analysis package.

The Waterloo Spatial Motion Analysis & Recording Technique (WATSMART™), Version 2.7, a noncontact optoelectric system, was used to track an infrared light-emitting diode (IRED) attached over the second metacarpophalangeal joint of the arm being studied. The two WATSMART™ sensors (cameras) were mounted approximately 1.5 m above the seated subject. Before each experiment with each arm, the sensors were calibrated in relation to the test area with a 1-m cubic grid with 24 IREDS imbedded at known locations. An average of 22 stable IREDS were used to calibrate the space. The average error of location of the IRED in space was 1.73 mm, with no error greater than 2 mm accepted.

The WATSMART™ was used in conjunction with a Hewlett-Packard Vectra personal computer that controlled the strobing of the IRED as well as data acquisition. The position of the IRED in X, Y, and Z planes was digitized at a rate of 100 Hz; these data were stored on FM tape for off-line analysis.

According to Twitchell (1951) and Brunnstrom (1970), persons at various stages of recovery from stroke have different movement capabilities, therefore the experimental setup involved three targets of varying difficulty (see Figure 2). Target 1 was located contralaterally, approximately 45° from midline, to require reaching into extensor synergy; Target 2 was located directly in front of the tested shoulder to require partial movement out of synergy; and Target 3 was located ipsilaterally, approximately 45° from midline, to require movement out of synergy. Each target was a 3-in. circle. Targets were arranged horizontally so that each one was 49.5 cm from the start pad that was located at the edge of the table, directly in front of the shoulder of the active arm. Light emitting diodes (LEDs) positioned above each target provided the go signal. Data collection started automatically when an LED was activated. A microswitch, imbedded in the start pad, signalled when the subject's hand moved off the start pad. When the subject touched anywhere on the target, another microswitch was activated to mark the end of reach. The output from all the microswitches was collected via the WATSCOPE™ A/D converter and software.

According to Fitts’s Index of Difficulty (Fitts, 1954), the task of this study would be rated as 3.70, which is classified by Wallace & Newell (1983) as an open-loop task (4.58). Therefore, reaches composed of one continuous movement would be expected.

Procedure

Each subject was seen for five visits, approximately once every 2 weeks. Results concerning changes occurring over the five visits have been submitted for publication elsewhere (Trombly, in press). No attempt was made to control for outpatient therapy or involvement in daily tasks during the experimental period. At each visit, the Fugl-Meyer Motor Function Test (Fugl-Meyer et al., 1975), the test for awareness of limb position (Leo & Soderberg, 1981), and the Modified Ashworth Scale of Muscle Spasticity (Bohannon & Smith, 1987) were administered before application of the electrodes and IREDS.

For the experiment, the subject sat on a straight-backed chair with the shoulder of the arm to be tested directly in line with Target 2 and the hand resting on the start pad. The subject was told which target to aim for before each trial and was instructed to reach, at illumination of the target LED, to the designated target with normal speed. A randomly delivered verbal ready signal prepared the subject before activation of the target LED. Those trials in which the subject started before the signal was activated were eliminated. Three scorable trials were completed to each target before reaching to the next target. A rest period of 30 sec was provided between trials.

Figure 2. Experimental setup.
Data Reduction and Analysis

The data had to be reduced into usable scores before they could be analyzed. The procedures for data reduction were as follows: The integrated electromyography scores for each muscle during reach were normalized as percentage of maximum voluntary contraction (%MVC). MVC refers to the amount of integrated electrical activity generated in a maximal isometric contraction when the muscle is in its shortened range. MVC was recorded for each muscle before each experiment. The percentage of MVC represents the amount of electrical activity generated by a muscle during a particular sample as compared with the maximum amount of electrical activity the muscle generated during MVC. Coactivity ratios were calculated with Hammond et al.'s (1988) formula:

\[
\text{coactivity ratio} = \frac{\text{antagonist}}{\text{agonist} + \text{antagonist}} \times 100\%
\]

Movement time was calculated, in milliseconds, from the hand off signal to the touch target signal of the WATSCOPE™ data file.

Speed of the endpoint in three-dimensional space, here referred to as velocity, and rate of change of speed, here referred to as acceleration, were obtained for every 0.01 sec from the WATSMART™ processed files with a custom-written program. The terms velocity and acceleration are used to be congruent with the terminology of the literature. However, velocity and acceleration are vector quantities, that is, directional, whereas speed and rate of change of speed are scalar quantities, that is, nondirectional. The latter measures describe the transport of the hand in all three dimensions (X, Y, and Z) simultaneously. The velocity and acceleration files generated were graphed for analysis of shape and characteristics (see Figure 1). From the files and graphs generated, the following kinematic variables were derived: number of movement units or steps within a discontinuous movement (Brooks et al., 1973), amplitude of peak velocity, time to peak velocity, and percentage of reach at which peak velocity occurred.

Only data from the last visit were used in this analysis. The scores taken at this time could have been affected by practice on this task on four previous visits and thus could be expected to represent the subjects' best performances. Contrary to expectations (Brunnstrom, 1970), all subjects were able to reach to all three targets. The only significant difference among the targets was the pectoralis major-anterior deltoid coactivity score of the impaired arm. Therefore, to test the differences between the impaired and unimpaired arms, all scores, except the pectoralis major-anterior deltoid coactivity score, were averaged across the three trials to the three targets for each subject.

The pectoralis major-anterior deltoid scores for both the impaired and unimpaired arms reflected the biomechanically required increased use of the pectoralis major relative to the anterior deltoid when subjects reached across the body to touch Target 1 and the increased use of the anterior deltoid in relation to the pectoralis major when subjects reached out, away from midline, to touch Target 3. The scores for Target 2 fell between those for Targets 1 and 3. When tested by target, the pectoralis major-anterior deltoid scores were not significantly different between arms.

The data were examined for the subjects as a group. This approach was conservative because the use of so few subjects reduced the statistical test's power to detect significant differences (Rosenthal & Rosnow, 1991; Stevens, 1986); however, if such differences or invariances were found, they would most likely be of major importance. The nonparametric Wilcoxon matched-pairs signed-rank test for related samples was used because the sample was small and the assumptions of parametric statistics could not be assured (Siegel, 1956). In this first level descriptive study, the level of significance was set at 15 (one-tailed), corrected for number of comparisons of the kinematic data and of the electromyographic data separately with weighted Bonferroni corrections (Rosenthal & Rosnow, 1991, Stevens, 1986).

Results

The normal continuous strategy of reaching to point at a large target was preserved in the unimpaired arm of all subjects, except Subject 3 (see Figure 3). Although reaching with the unimpaired arm was abnormal for Subject 3, that reach was smoother than the reach with her impaired arm. Other scores of Subject 3 also differed from the patterns of the other four subjects. Cerebral atrophy, which was mentioned in the report of the computed tomography scan for this subject (see Table 1), may account for these differences.

In addition to the preservation of the continuous strategy, the amplitude of peak velocity and time to peak velocity of the unimpaired arm were consistent for each subject; this finding is characteristic of unimpaired reaching. Additionally, the peak velocity occurred between 33% and 50% of the reach, which has been reported for unimpaired reaching to target (Arbib, 1985; Beggs & Howarth, 1972; Bullock & Grossberg, 1988; Hogan, 1984; Hollerbach & Flash, 1982; Morasso, 1983; Soechting & Lacquaniti, 1981).

In the impaired arm, there were significantly more movement units, indicating that the continuous strategy was lost (see Table 2). Movement time was significantly longer for the impaired than for the unimpaired arms. Peak velocity occurred earlier in the reach; that is, the velocity profiles were more left-shifted than for the unimpaired arms (see Figure 4), but this comparison did not reach significance according to the weighted probability level chosen (see Table 2).
Indications of reduced force (weakness) are low amplitude of peak velocity, low voltage during maximum voluntary contraction, and high percentage of MVC during unrestricted movement. All three of these factors were present in these subjects, but not at significant levels (see Tables 2 and 3). The amplitude of peak velocity of the impaired arms of four subjects was less and the velocity profiles more variable than for their unimpaired arms (see Figure 3). Except for the anterior deltoid and triceps of Subject 5 (most recovered) and the triceps of Subjects 1 and 2 (least recovered), all monitored muscles of the impaired arm generated less integrated electromyographic activity during the maximum voluntary control contraction as compared with the muscles of the unimpaired arm (see Figure 5). The biceps, one of the prime movers for forward reaching, showed a comparatively low level of muscle activity, as did the pectoralis major, an assistor to forward reaching across midline.

The monitored muscles of the impaired limbs were generally used at a greater percentage of MVC during reach than those of the unimpaired arms (see Figure 5). For subjects 4 and 5, who scored higher on the Fugl-Meyer Motor Function Test, the values tended to be similar between arms. The coactivity indexes were similar

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**Figure 3.** Kinematic variables for each subject. The standard deviation of amplitude of peak velocity of the impaired arm of Subject 3 exceeded the scale of the graph.

**Table 2**

<table>
<thead>
<tr>
<th>Wilcoxon Matched-Pairs Signed-Rank Test for Kinematic Variables</th>
<th>Weighted Critical Value&lt;sup&gt;a&lt;/sup&gt;</th>
<th>p&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Movement units</td>
<td>1</td>
<td>.04</td>
</tr>
<tr>
<td>Amplitude of peak velocity of first movement unit</td>
<td>2</td>
<td>.04</td>
</tr>
<tr>
<td>Time to peak velocity</td>
<td>6</td>
<td>.01</td>
</tr>
<tr>
<td>Percentage of reach where peak velocity occurs</td>
<td>0</td>
<td>.01</td>
</tr>
<tr>
<td>Movement time</td>
<td>0</td>
<td>.04</td>
</tr>
</tbody>
</table>

*Statistical analysis with Wilcoxon test. †Weightings can be allocated differently among the comparisons with good cause (Rosenthal & Rubin, 1991). In this case, the three variables found to be abnormal in the study by Lough et al. (1984) were weighted most heavily. The alpha level set for this exploratory study was .15; actual total alpha level = .146. One-tailed.

*Significant at .146 level.
Figure 4. (a) Velocity profiles of each subject's three reaches of the impaired arm, superimposed. Note the difference in ordinate scale for Subject 3. (b) Superimposed velocity profiles of three reaches of the unimpaired arms of each subject. Note that there was some replicability for all subjects except Subject 3.
Characteristics of unimpaired reaching described earlier were seen in the unimpaired arm; this finding supports the findings of Fisk and Goodale (1988) and indicates that the ability to plan movement normally was preserved in these left hemiparetic subjects. However, these subjects used a discontinuous guided movement strategy when reaching with their impaired limbs, which is consistent with the reported loss of continuous movement in other patients with central nervous system disorders (Flowers, 1976; Georgopoulos, 1986; Lough et al., 1984). The significantly slower movement time of the impaired arm increased the likelihood that the reaching movement would be guided and be kinematically described as discontinuous. Probable causes for the slowness and discontinuous movement could be spasticity or abnormal synergy that forced the arm off trajectory and required the subject to subconsciously correct the trajectory en route to the target. Each correction would result in another discontinuity. However, this explanation is not supported when the coactivity indexes are considered. The biceps-anterior deltoid coactivity indexes were not significantly different between arms in these particular subjects, indicating that flexor synergy, as described by Brunnstrom (1970), was not strongly controlling movement of the impaired arms. The indexes of the antagonistic pairs—triceps—biceps and pectoralis major—anterior deltoid—would be expected to be increased relative to the unimpaired arms if spasticity were hampering movement. On average, these scores were equal between arms or reduced for the impaired arm. This finding, along with
The variability seen in level of recruitment of individual muscles during repeated reaches by these subjects supports the view of Bernstein (1967), Kelso (1986), and Buchanan, Almdal, Lewis, and Rymer (1986). They proposed that muscle synergies seen in unimpaired persons are not hard-wired but are formed dynamically, as needed, to suit the goal of each movement within a given context. This principle of motor organization seems also to be operative in these subjects.

Weakness could account for the slow, discontinuous movement. If a subject with a weak arm started out using the same programmed strategy that had been correct before the stroke, the arm would fail to progress to the expected point along the trajectory within the time expected by the program. (It is known that the program, or plan, was available to the subjects because they had used the usual programmed strategy in reaching with their unimpaired arms.) Given the failure to approximate the target, the subject would then subconsciously correct the movement, resulting in discontinuities. Signs of weakness were apparent for these subjects, although the reduced muscle activity was not found to be statistically significant. When comparing the muscle activity of stroke subjects with that of unimpaired subjects, Trombly & Quintana (1985) also found low electrical activity during MVC and high percentage of MVC during resisted hand movements.

The finding of increased numbers of movement units also might be explained by the need to learn the new sensation of the impaired limb in relation to effort made. Rules developed before the stroke concerning the relationships of sensory experiences to motor outcomes probably do not represent the relationships after stroke (Winstein, 1987). Subjects 1 and 2 had mild deficits of awareness of limb position at Visit 5, so they may have used vision to guide their reaches, which would result in discontinuities and increased movement time. Movement time would be longer because visual feedback requires a longer processing time than kinesthetic feedback (Keele & Posner, 1968). However, Subjects 3, 4, and 5 seemingly had no need for visual guidance because they scored at maximum on the awareness of position test (Leo & Soderberg, 1981). Subject 4 had regained continuous movement strategy by Visit 5, which indicates her reliance on kinesthetic feedback. Although Subject 5 had regained normal continuous reaching strategy by Visit 4, he evidenced a guided strategy at Visit 5 because he was making an effort to slow his movement as his therapist advised. Subject 3, despite achieving maximal score on the test of awareness of limb position at every visit, evidenced more movement units than any other subject at Visit 5. Her pathology may have interfered with her ability to interpret and use proprioceptive cues during goal-directed movement, thus causing her to use a guided strategy.

The use of guidance is further supported by the shapes of the velocity profiles of these subjects (see Figure 4). The more left-shifted the velocity profile, the greater the indication that the movement is primarily guided by feedback (Nagasaki, 1985), that is, the endpoint of the limb is being decelerated with precision to guide it to the target.

Table 3
Wilcoxon Matched-Pairs Signed-Rank Test for Electromyographic Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Weighed Critical Value</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum Voluntary Contraction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior deltoid</td>
<td>1</td>
<td>.019</td>
</tr>
<tr>
<td>Biceps</td>
<td>0</td>
<td>.019</td>
</tr>
<tr>
<td>Pectoralis major</td>
<td>0</td>
<td>.009</td>
</tr>
<tr>
<td>Triceps</td>
<td>3</td>
<td>.009</td>
</tr>
<tr>
<td>Percentage of Maximum Voluntary Contraction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior deltoid</td>
<td>2</td>
<td>.019</td>
</tr>
<tr>
<td>Biceps</td>
<td>6</td>
<td>.019</td>
</tr>
<tr>
<td>Pectoralis major</td>
<td>0</td>
<td>.009</td>
</tr>
<tr>
<td>Triceps</td>
<td>6</td>
<td>.009</td>
</tr>
<tr>
<td>Coactivity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pectoralis major—Anterior deltoid</td>
<td>5</td>
<td>.009</td>
</tr>
<tr>
<td>Biceps—Anterior deltoid</td>
<td>5</td>
<td>.019</td>
</tr>
<tr>
<td>Triceps—Biceps</td>
<td>5</td>
<td>.009</td>
</tr>
</tbody>
</table>

*Statistics associated with Wilcoxon test. †Per Rosenthal and Rosnow (1991); total of weighted alpha level = 1.45. The anterior deltoid and biceps were given greater weight because they are prime movers for reaching forward (Basmajian & DeLuca, 1985). ‡One-tailed.

Conclusion and Implications
One of the unique contributions of the present study was the application of kinematic recording techniques in combination with electromyography to the study of stroke patients to provide information concerning the strategies and tactics used in goal-directed reaching. The findings of this study corroborate findings of previous studies and add to the knowledge of motor control deficits after stroke. The findings obtained from the subjects with left hemiparesis indicated that, although they were able to accomplish the goal of reaching to target, they had difficulty executing smooth, coordinated movement with the impaired arms. They had no such difficulty with the unimpaired arms. The data suggest a loss of open-loop control of this simple movement in the impaired arms, possibly due to a combination of weakness and the new sensorimotor relationships within the arm, as yet unlearned.

There was no evidence that abnormal muscle activity was occurring to any significant degree; therefore the neurodevelopmental approach to normalize tone or learn more primitive patterns of movement does not seem an appropriate treatment choice for the subjects of this study. Because the goal of therapy to restore functional movement is to reverse deficient aspects of movement, the findings suggest that strengthening and relearning of sensorimotor relationships might be the
appropriate therapeutic goals for these particular subjects. This study should be replicated and expanded to confirm the validity of the findings, to allow generalizability, to determine the mechanism or mechanisms underlying the discontinuous movement strategy observed, and to test whether muscle strengthening or sensorimotor recalibration or both could be effective treatments. ▲

Acknowledgments
I thank Spaulding Rehabilitation Hospital, Boston, Massachusetts; Braintree Hospital, Braintree, Massachusetts; and New England Rehabilitation Hospital, Woburn, Massachusetts, for their cooperation. I especially appreciated the assistance of K. Ramesh Murthy, M.D., Laura DeVore Hollar, M.S.; Michael Baker, M.S.; Daniel Bullock, M.D., Paul Petrone, OTR, PhD, Deborah Reichel Desmond, M.S., OTR, Deborah Palme, OTR, Karen Lindsay, OTR, Lynn Langmaier, OTR, and Shjeldr Crotty, OTR.

Funding for this study was provided through a Mary E. Switzer Distinguished Researcher Fellowship Award from the American Occupational Therapy Association/American Occupational Therapy Foundation Center for Scholarship and Research at Boston University. This paper was part of a dissertation that was guided by Jane Coryell, Ph.D., Linda Fettets, Ph.D., M. J. Blaschak, Ph.D., and Leonard Ziwichowsky, Ph.D.

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