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RESEARCH NOTE

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Directional control of reaching is preserved following mild/moderate stroke and stochastically constrained following severe stroke

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Abstract Recent evidence suggests that brain injury can impair the ability to independently activate shoulder and elbow muscles. We hypothesized that if muscle activation patterns are constrained, then brain-injured subjects should not be able to accurately grade initial hand movement direction during reaching toward a broad range of target directions. To test this hypothesis, we measured hand trajectories during reaching in three-space by 16 hemiparetic stroke subjects to an array of 75 targets distributed throughout the workspace. Contrary to our hypothesis, we found that the ability to grade movement direction was largely preserved following mild and moderate stroke. However, the most severely impaired subjects exhibited a degradation of directional control consistent with a loss of independent muscle control. Initial and final hand movement directions for these subjects were grouped roughly in two opposing directions, in a plane parallel with the coronal plane of the body, rather than distributed across the normal range. Selection between the two movement directions appeared partially random, in that subjects initiated over 50% of movements in the direction generally opposite the intended target, for targets to one side of the body. These results suggest that individuals with severe stroke are constrained to use only two gross, stereotypical muscle coactivation patterns for reaching control, and that selection between these patterns is stochastically influenced as the actual direction of motion is not strictly predictable given the desired direction.

Keywords Arm · Reaching · Motor control · Stroke · Movement synergy

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Introduction

Damage to motor areas of the brain has long been hypothesized to reduce the ability to independently activate muscles. For example, Twitchell (1951) and Brunnstrom (1970) described a loss of independent joint control in the arm following stroke. One explanation is that destruction of higher motor outflow pathways (for example, corticospinal or rubrospinal pathways) induces a reliance on lower pathways (for example, vestibulospinal, reticulospinal, and tectospinal) to control movement (Bourbonnais et al. 1989; Dewald and Rymer 1993). Since these lower pathways are characterized by a more diffuse muscular innervation, they would be expected to activate multiple muscles, resulting in stereotypical movement patterns. In rehabilitation following stroke, several prevalent paradigms of assessment and therapy are based on clinical observations of stereotypical movement patterns, referred to as "abnormal muscle synergies" (Fugl-Meyer et al. 1975; Sawner and LaVigne 1992; Gowland et al. 1993).

Recent evidence from multiple-muscle EMG and multiaxial force recordings supports the concept of constrained coactivation in the hemiparetic arm. Hemiparetic arms exhibit an increased occurrence of abnormal muscle coactivation during isometric force generation, especially between elbow flexors and shoulder abductors, and between elbow extensors and shoulder adductors (Bourbonnais et al. 1989; Dewald et al. 1995). Hemiparetic arms also have difficulty generating isometric force in stereotypical directions that are geometrically opposed to the clinically described synergies (Beer et al. 1999). Stroke-impaired arms produce substantial torques

in secondary directions when generating maximum torque in a target direction (Dewald and Beer 2001). In addition, when moving along constrained trajectories created by robotic devices, hemiparetic arms generate substantial contact forces against the devices, perpendicular to the intended direction of movement (Lum et al. 1999; Reinkensmeyer et al. 1999a, b).

If muscle coactivation patterns are constrained following stroke, then the kinematics of free arm movement should also reflect these constraints. The purpose of this study was to examine reaching trajectories for evidence of constrained multiple muscle control. We hypothesized that the ability to vary initial movement direction as a function of target direction would be compromised if independent muscle control were impaired. Data from several previous studies that examined reaching kinematics following stroke (Wing et al. 1990; Trombly 1992; Levin 1996; Roby-Brami et al. 1997; Archambault et al. 1999; Beer et al. 2000) provide little evidence of an impaired ability to grade initial movement direction, but none of these studies examined unsupported reaching to a wide range of targets for a broad range of impairment levels.

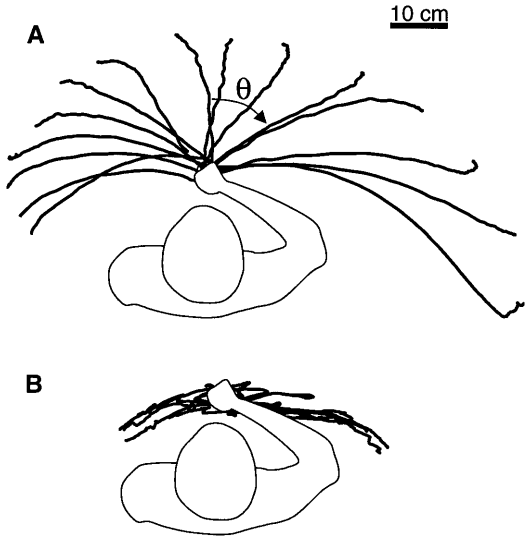

Fig. 1 Example hand trajectories for the ipsilesional (**A**) and contralesional arms (**B**) of a subject with severe stroke. The contralesional (left) arm trajectories are flipped about the sagittal plane. Reaching direction (θ) is defined positive for movements to the right of straight ahead. Note that movement was constrained to essentially two directions (medial and lateral) for the severely impaired subject, even though substantial active range of motion was preserved in these directions

Materials and methods

Twenty subjects, 16 with chronic hemiparetic stroke, participated in the study. The stroke subjects' mean age was 57.4±15.1 (standard deviation) years, and the mean time hyphenate was 2.1±1.6 years. Criteria that excluded participation were cognitive dysfunction, neglect, apraxia, and shoulder pain. Four unimpaired subjects (mean age 32±6.2 years) also participated. Subjects gave informed consent in accordance with the Helsinki Declaration. The work was approved by the local ethics committee of Northwestern University.

The upper extremity impairment of each stroke subject was rated by an experienced therapist using the Stage of Arm section of the Chedoke-McMaster Stroke Assessment scale, a seven-point scale that is based on a hypothesized progression from paralysis (score 1), to movement using abnormal muscle synergies (scores 2–3), to movement out of synergy (scores 4–6), to normal movement (score 7) (Gowland et al. 1993). Subjects were classified as severely impaired if they scored a 2 on the scale (5 subjects), and moderately/mildly impaired if they scored higher than a 2 [distribution (score, number of subjects): (2, 5); (3, 4); (4, 4); (5, 2); (6, 1)]. Each subject's ipsilesional arm was used as a within-subject control for comparison with the contralesional, paretic arm since all subjects exhibited clinically normal usage of their ipsilesional arm, and performed functional tasks regularly with it. Reaching trajectories from both arms of the four unimpaired individuals were also measured to assess possible differences arising between dominant and non-dominant arms.

Following a brief practice period, the seated subjects were asked to perform 150 reaching movements, 75 with the paretic arm, and 75 with the ipsilesional arm, to a semicircular, vertically standing screen of 75 targets placed approximately 1 m from the torso. The 2.5-cm-diameter targets were evenly spaced along five latitudinal (i.e., vertically spaced) arcs and 15 longitudinal (i.e., horizontally spaced) meridians separated by 12° in both directions for spans of 48° in the vertical direction and 168° in the horizontal direction. Each reach was directed at a target chosen at random from the set of 75 targets. The repeatability of repetitive reaching to a single target has been observed previously to be high (Reinkensmeyer et al. 1999a), and therefore only one reach was recorded for each target to maximize the number of target directions that could be tested without fatigue. The subject was positioned such that the sternoclavicular notch was aligned with the middle of the target area. The subject's trunk was restrained with a four-point harness that allowed full scapular mobility. The subject's wrist was splinted to prevent wrist flexion.

From an initial posture with the thumb against the umbilicus and the palm resting against the body, each subject was instructed to reach at a comfortable speed to a point as close as possible to the target, maintain that position for 1 s, and return to the starting position. Subjects were given periodic rest breaks to minimize fatigue. An electromagnetic position sensor (Flock of Birds; Ascension Technology, Burlington, Vt., USA) was attached to the dorsum of the hand to measure the position and orientation of the hand. The transmitter was placed behind the head on a platform. Care was taken to remove ferrous metals from the sensor workspace, and readings were tested to be accurate to at least 1 cm.

During each reach, hand position data were gathered at a sampling rate of 100 Hz and stored on a computer. These data were then digitally lowpass-filtered forward and backward in time at 5 Hz with a 30th-order FIR filter to attenuate high-frequency noise without altering signal phase. Movement direction was assessed by calculating the angle in the horizontal plane between selected movement vectors and a reference vector, defined to point in the direction perpendicular to the subject's torso (i.e., straight ahead). The origin of each movement vector was the position of the hand at the initiation of reaching, defined as the point at which the tangential velocity of the hand exceeded a small velocity threshold (3 cm/s). The tip of each movement vector was a point on the hand trajectory. To assess the open-loop component of motion, this point was taken to be 200 ms after movement initiation; voluntary corrections to perceived errors would likely not occur during this period. To assess final movement direction, the vector tip was defined as the point at which the hand came closest to the target. Positive movement direction angle was defined such that for the right hand, movements to the right of straight ahead had a positive movement angle (Fig. 1). Analysis of the left hand was performed in a mirror symmetric fashion.

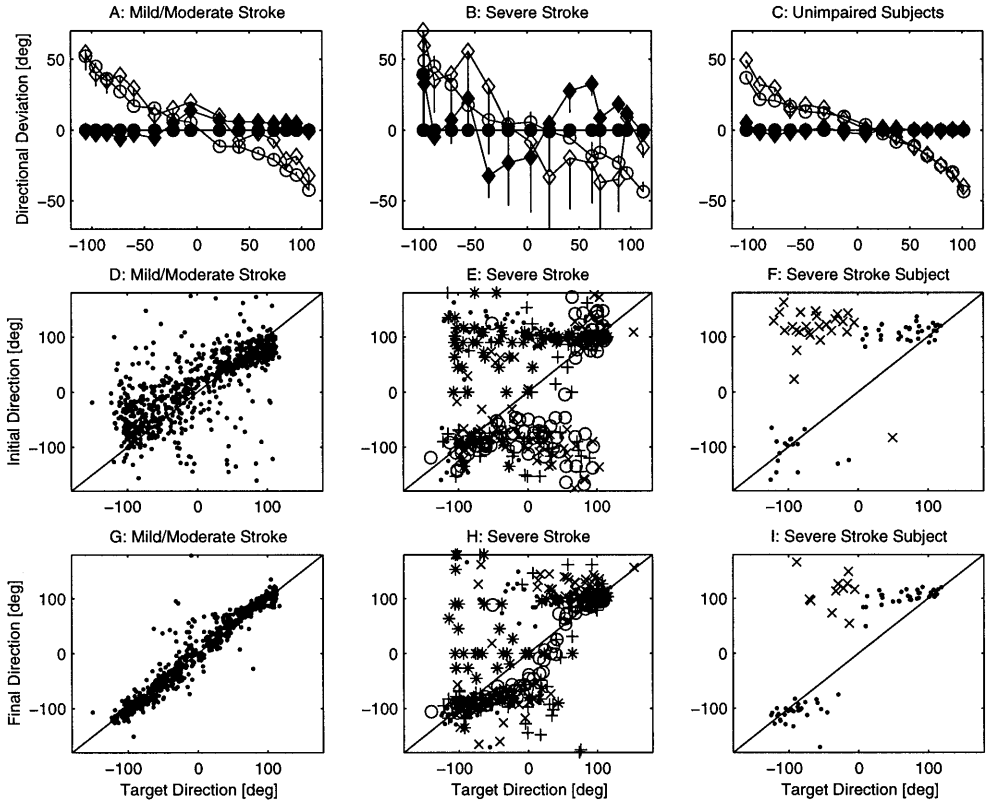

Fig. 2A–I Directional deviation (*top row*), initial movement direction (*middle row*), and final movement direction (*bottom row*) plotted versus target direction for stroke and unimpaired subjects. Directional deviation is defined as the difference between reaching direction and the target direction in the coordinate system shown in Fig. 1. Target direction is defined on a subject-specific basis as the final reaching direction achieved by each stroke subject's ipsilesional arm, or each unimpaired subject's dominant arm. **A** Directional deviation for mildly and moderately impaired stroke subjects. *Open diamond* Contralesional arm, initial direction, *filled diamond* contralesional arm, final direction, *open circle* ipsilesional arm, initial direction, *filled circle* ipsilesional arm, final direction. Initial directional deviation was measured at 200 ms following initiation of movement, and final directional deviation at the end of movement. Mean across subjects plus or minus standard error of the mean. Note that the hand initially did not move directly toward the target for targets away from 0°, but curved back to the target direction (see trajectories in Fig. 1A), similar to the unimpaired subjects in **C**. **B** Severely impaired stroke subjects. *Symbols* the same as **A**. Movement direction was not smoothly graded as a function of target direction, and initial and final directional deviations were significantly larger than for the ipsilesional arms. **C** Unimpaired subjects. *Open circle* Dominant arm, initial direction, *filled circle* dominant arm, final direction, *open diamond* non-dominant arm, initial direction, *filled diamond* non-dominant arm, final direction. **D** Initial movement direction for contralesional arms of subjects with mild and moderate stroke. Each point is a reach, and all reaches for all subjects with a Chedoke score above 2 are shown. Note the strong correlation between target direction and initial direction. **E** Initial movement direction for contralesional arms of subjects with severe stroke. Each of five subjects is shown with a different plot symbol. Note the segregation of initial movement direction into clusters at –100° and 100°, distinctly different from the pattern in **E**. **F** Example of data from the contralesional arm of one subject with severe stroke. The subject moved reliably rightward when the desired movement was rightward (i.e., positive target directions), but moved leftward only 39% of the time when the desired movement was leftward. Movements initiated into the wrong spatial quadrant are denoted with an *x*. Note the large number of reaches initially misdirected to the right side for leftward targets (i.e., positive movement directions were achieved for negative target directions). In contrast, there are few reaches initially directed to the left side for rightward targets. **G** Final movement direction for contralesional arms of subjects with mild and moderate stroke. **H** Final movement direction for contralesional arms of subjects with severe stroke. Note that the segregation of movement direction persisted until the end of movement. **I** Final movement direction for same subject as in **F**. The subject ended 73% of movements rightward when the desired movement was rightward, significantly decreasing the number of misdirected movements (again denoted with an *x*)

Results

Hand trajectories during reaching were measured for dominant and non-dominant arms of unimpaired subjects, for the contralesional, paretic arms of stroke subjects, and for the ipsilesional arms of stroke subjects (Fig. 1). For both arms of the unimpaired subjects and the ipsilesional arms of the stroke subjects, initial movement direction was strongly correlated with target direction (mean r=0.93±0.05, P<0.001 for all regressions), indicating that movement direction was graded with target direction. A characteristic feature of the reaching trajectories for these arms was that they did not move directly toward the target at first, but rather were directed initially more toward the subject's midline, then curved systematically to the target (Figs. 1A, 2A–C).

Reaches performed with the contralesional arms of the subjects with mild and moderate stroke (Chedoke stages 3–6) followed trajectories similar to the reaches with the ipsilesional and unimpaired arms. For these arms, the initial movement direction was also strongly correlated with the target direction (mean r=0.81±0.11, P<0.001 for all regressions; Fig. 2D). In addition, the contralesional arms ended movement at approximately the same final direction as the ipsilesional and unimpaired arms (Fig. 2A, G). Thus, the subjects with mild and moderate stroke were able to smoothly grade initial and final movement direction as a function of target direction with their paretic arms.

In distinct contrast, for the contralesional arms of subjects with severe stroke (Chedoke stage 2), initial movement directions were poorly correlated with target direction (mean r=0.44±0.14; Fig. 2B). These subjects also exhibited significantly larger initial directional errors (mean absolute error 19.4±12.3°) than the mildly/moderately impaired subjects (mean error 9.6±12.3°, t-test, P<0.001), and their final absolute directional deviations (19.0±12.3°) were significantly greater than for the mildly/moderately impaired subjects (4.9±3.1°, P<0.001).

The larger errors resulted from two stereotypical reaching patterns. Initial movement directions were clustered around –100° and 100° for the contralesional arms of the subjects with severe stroke, where 0° is defined as straight ahead from the hand's initial resting point (Figs. 1B, 2E). Thus, the subjects with severe stroke initiated movement with their impaired arms in only two general directions, which can be characterized as medial (i.e., across the midline) or lateral. This was in direct contrast to the same subjects' ipsilesional arms, which did not exhibit clustering of initial movement directions and smoothly graded movement direction. To test for a possible effect of movement velocity (i.e., the more severely impaired subjects moved more slowly with the contralesional arm and thus the hand may not have traveled as far after 200 ms), the same analysis was performed with movement direction calculated when the hand had traveled 5 cm. Similar clustering was found.

Each of the subjects with severe stroke initiated movement with the contralesional arm substantially more reliably in one of the two directions, with the medial direction being more reliable for two subjects, and the lateral for three subjects (see, for example, Fig. 2F). In the subjects' more reliable direction, they initiated movement to the correct side of the hand start position (i.e., to the spatial quadrant containing the intended target) on average 91±8% of the time, while in the less reliable direction they achieved this only 41±19% of the time, a significantly smaller percentage (paired t-test, P<0.001). Thus, when reaching toward targets in their less reliably controlled direction, subjects initially moved into the wrong spatial quadrant over half of the time. For comparison, the ipsilesional arms of the same stroke subjects initiated movement into the correct quadrant 94.4±5.3% of the time. An analysis of the influence of target height on initiation accuracy revealed no significant effects.

For the contralesional arms of the severe stroke subjects, the clustering of movement directions into two preferred directions was largely preserved until the end of the reach (Fig. 2H). However, a fraction of the initially misdirected reaches were corrected. As a result, the arms finished movement in the correct quadrant 69±15.0% of the time (see, for example, Fig. 2I), a percentage significantly greater than the observed 41±19% of errors in the initial movement (P<0.01), but still remarkably low, indicating severe constraints on directional control throughout the entire course of movement.

Discussion

These results support the hypothesis that brain injury reduces the ability to independently control muscles during free arm movement for the case of severe stroke, but not for the case of moderate or mild stroke. For the contralesional arms of the subjects with severe stroke, both initial and final hand movement directions were grouped in two opposing directions, rather than distributed across the normal range. A plausible explanation is that these subjects selected from and were constrained by a set of two gross patterns of muscle activation in order to initiate and complete movements. In contrast, the mildly/moderately impaired subjects smoothly modulated initial movement direction as a function of target direction, suggesting that they essentially maintained the ability to grade muscle activity for directional control.

The constraints in initial movement direction observed for the severely impaired subjects correspond well with previous quantitative descriptions of impaired, multiple-muscle control in isometric and mechanically constrained situations (Dewald et al. 1995; Beer et al. 1999; Reinkensmeyer et al. 1999a; Dewald and Beer 2001), as well as with clinical descriptions of "abnormal extension and flexion synergies" (Brunnstrom 1970). From a starting position with the hand in the lap, the observed movement initiation in the medial direction is consistent with elbow extension and some combination of shoulder internal rotation/adduction, similar to the previously described "abnormal extension synergy." Conversely, the observed movement initiation in the lateral direction is consistent with elbow flexion and shoulder external rotation/abduction, similar to the "abnormal flexion synergy."

A possible alternate explanation for the constrained directional control is severe, selective muscular weakness. Weakness in shoulder flexors may have prevented the subjects from moving their hands outward from the torso, thus constraining the hand trajectories to the approximate coronal plane. However, the medial/lateral movements achieved by the severely impaired subjects also required substantial shoulder strength (for example, adduction and internal rotation, or abduction and external rotation) to lift and move the hand, and previous studies of weakness following stroke have found that shoulder flexion weakness is characteristically accompa-

nied by comparable weakness in other shoulder degrees of freedom (Gandevia 1993). Thus, while weakness was certainly more pronounced in the severely impaired subjects, the observed deficits likely resulted from an inability to achieve the required combination of shoulder muscle and elbow activations, rather than severe weakness in one specific shoulder muscle group. A uniform weakness of multiple shoulder muscles would not be expected to limit directional control in specific directions, but rather to limit active range of motion across multiple directions.

The results also suggest that a stochastic mechanism influenced the selection between the two available control patterns. This mechanism primarily influenced reaches toward only one half of the workspace, i.e., in the "less reliable direction". Movements in the complementary direction were reliably initiated. A possible explanation is that, for a given subject, one of the two available control patterns was dominant due to a decreased threshold in the neural pathways that mediated it. These pathways may have been activated with finite probability even when other, possibly higher threshold, pathways were more appropriate.

The present data also suggest that on-line corrections of initial movement misdirections are possible even following severe stroke, but are also governed by a stochastic selection mechanism. Specifically, while the severely impaired stroke subjects corrected a fraction of the initial movement misdirections, they still completed a substantial percentage (31%) of movements in the wrong spatial quadrant. One explanation is that the motor control system sensed an initial movement misdirection and attempted a feedback correction, but was still constrained to select from the two available patterns, with similar probability of success as at movement initiation. Given the observed initial misdirection percentage of 59%, and a 0.41 probability of success with a second on-line correction, 35% of reaches should remain misdirected, a number consistent with the observed final percentage of 31%.

The relatively preserved directional control observed following mild/moderate stroke does not imply that other aspects of reaching control were not impaired for these subjects. For example, these subjects made small (<10° on average) but statistically significant errors in initial movement direction with their contralesional arms, as has been observed previously for planar, supported arm movements (Beer et al. 2000). In addition, in a related study, we found significant differences in range, velocity, smoothness, and trajectory variability following mild/moderate stroke (Kamper et al. 2002). Further, the present study only measured hand trajectories, and since the human arm is kinematically redundant, the subjects may have used abnormal joint coordination to help gradate directional control. Impaired multiple joint control and sequencing of redundant degrees of freedom has been observed previously following stroke (Levin 1996; Archambault et al. 1999; Beer et al. 2000). Another possibility is that agonist/antagonist co-contraction, and thus arm stiffness, was abnormal despite preserved directional control (Hammond et al. 1988; see, however, Reinkensmeyer et al. 1999b).

Even considering these factors, the present findings clearly suggest that there are two identifiable classes of directional control following stroke: largely preserved and severely constrained. Referring back to the hypothesis that lower pathways substitute for corticospinal ones following stroke, a possible explanation for these two classes is that directional control is largely preserved if some threshold fraction of corticospinal pathways is spared. This hypothesis should be testable in the future via detailed functional imaging of key neural tracts.

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References

Archambault P, Pigeon P, Feldman AG, Levin MF (1999) Recruitment and sequencing of different degrees of freedom during pointing movements involving the trunk in healthy and hemiparetic subjects. Exp Brain Res 126:55–67

Beer RF, Given JD, Dewald JP (1999) Task-dependent weakness at the elbow in patients with hemiparesis. Arch Phys Med Rehabil 80:766–772

Beer RF, Dewald JP, Rymer WZ (2000) Deficits in the coordination of multijoint arm movements in patients with hemiparesis: evidence for disturbed control of limb dynamics. Exp Brain Res 131:305–319

Bourbonnais D, Vanden Noven S, Carey KM, Rymer WZ (1989) Abnormal spatial patterns of elbow muscle activation in hemiparetic human subject. Brain 112:85–102

Brunnstrom S (1970) Movement therapy in hemiplegia. Harper and Row, New York

Dewald JP, Beer RF (2001) Abnormal joint torque patterns in the paretic upper limb of subjects with hemiparesis. Muscle Nerve 24:273–283

Dewald JPA, Rymer WZ (1993) Factors underlying abnormal posture and movement in spastic hemiparesis. In: Thilmann AF, Burke DJ, Rymer WZ (eds) Spasticity: mechanisms and management. Springer, Berlin Heidelberg New York, pp 123–138

Dewald JPA, Pope PS, Given JD, Buchanan TS, Rymer WZ (1995) Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. Brain 118:495–510

Fugl-Meyer AR, Jaasco L, Leyman L, Olsson S, Steglind S (1975) The post-stroke hemiplegic patient. Scand J Rehabil Med 7:13–31

Gandevia SC (1993) Strength changes in hemiparesis: measurements and mechanisms. In: Thilmann AF, Burke DJ, Rymer WZ (eds) Spasticity: mechanisms and management. Springer, Berlin Heidelberg New York, pp 111–122

Gowland C, Stratford P, Ward M, Moreland J, Torresin W, Van Hullenaar S, Sanford J, Barreca S, Vanspall B, Plews N (1993) Measuring physical impairment and disability with the Chedoke-McMaster stroke assessment. Stroke 24:58–63

Hammond MC, Fitts SS, Kraft GH, Nutter PB, Trotter MJ, Robinson LM (1988) Co-contraction in the hemiparetic forearm: quantitative EMG evaluation. Arch Phys Med Rehabil 69:348–351

Kamper DG, McKenna AN, Kahn LE, Reinkensmeyer DJ (2002) Alterations in reaching after stroke and their relationship to movement direction and impairment severity. Arch Phys Med Rehabil (in press)

Levin MF (1996) Interjoint coordination during pointing movements is disrupted in spastic hemiparesis. Brain 119:281–293

Lum PS, Burgar CG, Kenney D, Van der Loos HFM (1999) Quantification of force abnormalities during passive and active-assisted upper-limb reaching movements in post-stroke hemiparesis. IEEE Trans Biomed Eng 46:652–662

Reinkensmeyer DJ, Dewald JPA, Rymer WZ (1999a) Guidance based quantification of arm impairment following brain injury: a pilot study. IEEE Trans Rehabil Eng 7:1–11

Reinkensmeyer DJ, Schmit BD, Rymer WZ (1999b) Assessment of active and passive restraint during guided reaching after chronic brain injury. Ann Biomed Eng 27:805–814

Roby-Brami A, Fuchs S, Mokhtari M, Bussel B (1997) Reaching and grasping strategies in hemiparetic patients. Motor Control 1997:72–91

Sawner K, LaVigne J (1992) Brunnstrom's movement therapy in hemiplegia: a neurophysiological approach. Lippincott, Philadelphia

Trombly CA (1992) Deficits of reaching in subjects with left hemiparesis: a pilot study. Am J Occup Ther 46:887–897

Twitchell TE (1951) The restoration of motor function following hemiplegia in man. Brain 74:443–480

Wing AM, Lough S, Turton A, Fraser C, Jenner JR (1990) Recovery of elbow function in voluntary positioning of the hand following hemiplegia due to stroke. J Neurol Neurosurg Psychiatry 53:126–134