RESEARCH ARTICLE | *Nervous System Pathophysiology*

Spasticity may obscure motor learning ability after stroke

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Subramanian SK, Feldman AG, Levin MF. Spasticity may obscure motor learning ability after stroke. *J Neurophysiol* 119: 5–20, 2018. First published September 13, 2017; doi:10.1152/jn.00362. 2017.—Previous motor learning studies based on adapting movements of the hemiparetic arm in stroke subjects have not accounted for spasticity occurring in specific joint ranges (spasticity zones), resulting in equivocal conclusions about learning capacity. We compared the ability of participants with stroke to rapidly adapt elbow extension movements to changing external load conditions outside and inside spasticity zones. Participants with stroke ($n = 12$, aged 57.8 ± 9.6 yr) and healthy age-matched controls ($n = 8$, 63.5 ± 9.1 yr) made rapid $40°$–$50°$ horizontal elbow extension movements from an initial ($3°$) to a final ($6°$) target. Sixteen blocks (6–10 trials/block) consisting of alternating loaded (30% maximal voluntary contraction) and non-loaded trials were made in one (controls) or two sessions (stroke; 1 wk apart). For the stroke group, the tonic stretch reflex threshold angle at which elbow flexors began to be activated during passive elbow extension was used to identify the beginning of the spasticity zone. The task was repeated in joint ranges that did or did not include the spasticity zone. Error correction strategies were identified by the angular positions before correction and compared between groups and sessions. Changes in load condition from no load to load and vice versa resulted in undershoot and overshoot errors, respectively. Stroke subjects corrected errors in 1–4 trials compared with 1–2 trials in controls. When movements did not include the spasticity zone, there was an immediate decrease in the number of trials needed to restore accuracy, suggesting that the capacity to learn may be preserved after stroke but masked by the presence of spasticity.

NEW & NOTEWORTHY When arm movements were made outside, instead of inside, the range affected by spasticity, there was an immediate decrease in the number of trials needed to restore accuracy in response to a change in the external load. This suggests that motor learning processes may be preserved in patients with stroke but masked by the presence of spasticity in specific joint ranges. This has important implications for designing rehabilitation interventions predicated on motor learning principles.

motor control; spasticity; stroke rehabilitation; motor learning; goal-directed behavior

INTRODUCTION

Stroke often results in spasticity and associated motor impairments in the upper limb (UL; Cheung et al. 2016; Opheim et al. 2014), including decreased strength (Harris and Eng 2007; Li et al. 2014), abnormal coactivation (Levin and Dimov 1997; Musampa et al. 2007; Trumbower et al. 2010), and altered interjoint (Gera et al. 2016; Levin 1996) and intersegmental coordination (Shaikh et al. 2014). With deficits in independent reaching and grasping ability, persistent UL impairments can lead to limitations in activities of daily living, social participation, and quality of life (Winstein et al. 2016). Despite efforts to tailor exercise interventions to patient needs, results of UL rehabilitation have been disappointing (Veerbeek et al. 2014). This may be due to a lack of focus on improving specific motor skills.

One basic motor skill that is impaired in patients with stroke is the ability to rapidly adapt arm movement to changing environmental conditions (Baniña et al. 2017; Boyd and Winstein 2004; Dancause et al. 2002; Takahashi and Reinkensmeyer 2003). For example, individuals with left parietal lobe damage had decreased ability to adapt reaching movements to shifted visual targets (Mutha et al. 2011; Palluel-Germain et al. 2011), and all subjects, regardless of the side of brain damage, had reduced performance on a visual UL obstacle avoidance task (Baniña et al. 2017). Other studies investigating adaptation of arm movements to changing external load conditions also reported deficits in the more affected compared with the less affected arm and the arms of healthy controls (Dancause et al. 2002; Takahashi and Reinkensmeyer 2003) with the deficit in adaptive behavior related to the severity of the motor impairment (Dancause et al. 2002).

Although motor learning is not abolished by stroke (Boyd et al. 2007; Meehan et al. 2011), the ability to adapt motor behavior may vary greatly between individuals because of differences in levels of motor impairment and remaining kinematic redundancy (Levin et al. 2016). More specifically, motor adaptation may critically depend on the range of motion in the joint configuration space in which the movement is performed. The range of elbow joint space in which movement can be made with normal muscle activation patterns is diminished in patients with stroke (Levin 2000; Levin et al. 2000; Musampa et al. 2007). The functional implications of deficits in the control of motion in specific parts of the elbow joint range in this population can be understood in the context of how the normal control of motoneurons (MNs) and muscles is disrupted after stroke-related pathology.

Normal motor control mechanisms based on regulation of the threshold properties of MNs have been proposed and

experimentally supported. In particular, since Leksell (1945), it has been known that afferent feedback to α-MNs is modulated by γ-MNs that innervate muscle spindle receptors. Initially Matthews (1959) and then Feldman and Orlovsky (1972) recorded force-length characteristics of ankle muscles in decerebrate cats during different background levels of γ influences graded by anesthesia of γ-fibers in the muscle nerve. They found that these influences shifted the spatial threshold, i.e., the muscle length (λ) or respective joint angle (R) at which gradual recruitment of α-MNs begins to produce active muscle force (Fig. 1A). The spatial threshold and thus the muscle force-length characteristics can also be shifted by intermuscular reflexes influencing α-MNs via spinal interneurons. In addition, Feldman and Orlovsky (1972) tonically stimulated vestibulo-, cortico-, and reticulospinal pathways to show that direct (monosynaptic) or indirect (via spinal interneurons and/or γ-MNs) influences of these pathways on α-MNs primarily result in shifts in the spatial thresholds, λ. Consequently, the level of active muscle force is not predetermined by these pathways but emerges if the difference between the actual (x) and threshold muscle length becomes positive (i.e., when $x - \lambda > 0$). In addition, Capaday (1995) showed that the
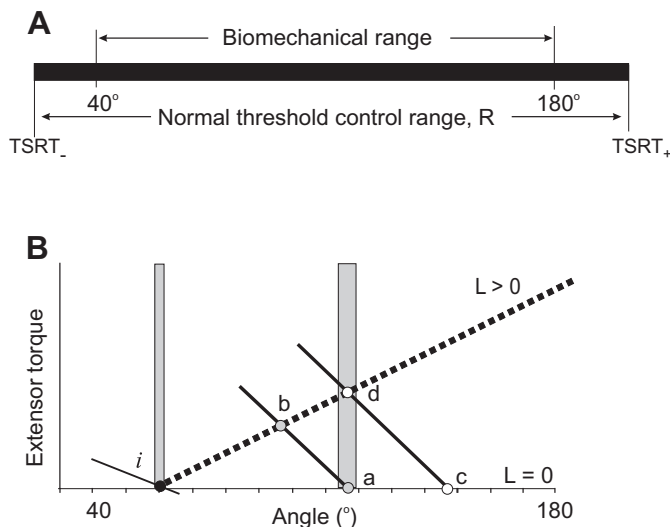

Fig. 1. Threshold position control of movement adaptation to sudden changes in the load (schematic diagrams). A: the biomechanical range of the elbow joint is limited by flexion to ~40° (due to the soft tissue of the upper arm) and extension to 180° (due to bony approximation). The threshold position is the elbow angle (R) at which motoneurons begin to be activated and the tonic stretch reflex (TSRT) becomes functional. The range (TSRT$_-$, TSRT$_+$) is defined by the limits of central facilitation/inhibition of motoneurons and extends beyond the biomechanical range. B: elbow extension is accomplished by changing muscle activation thresholds, resulting in a shift of the torque/angle characteristic of the joint (left solid diagonal line) to the right with a possible increase in the slope (line ab). If movement is adapted to a zero load (L = 0), the equilibrium point (EP) of the joint is shifted from i to a, bringing the elbow position from the initial (left shaded bar) into the target window (right shaded bar). If a spring-like load is suddenly introduced (diagonal dotted line, L > 0), the same shift in the characteristic brings the joint to EP b, resulting in an undershoot positional error. To correct the error, the characteristic should be shifted further to the right to bring the joint to EP d and balance the load within the target window. This shift should be reproduced in the subsequent trials with the load to regain movement accuracy. However, if the loaded trial characteristic is reproduced when unexpectedly no load was presented in the upcoming trial, an overshoot error would occur (EP c). To correct the error, the subject would need to respecify the initial characteristic learned for the nonloaded condition to balance the load at EP a.

activation threshold also depends on the level of presynaptic inhibition of MNs.

Using perturbation methods, Asatryan and Feldman (1965) showed that shifts in the spatial activation threshold and respective force-length curves underlie intentional changes in the elbow position in healthy humans, which has been confirmed by others (for review see Latash 1993). Thus the principal mechanism of muscle control revealed in decerebrate cats, shifts in the spatial threshold, is characteristic of intact motor control in humans. In terms of the dynamic systems theory, by changing λ, the nervous system shifts the equilibrium point (EP) to which the neuromuscular periphery interacting with the external force (load) is attracted. The muscle force and length are thus determined by the point of intersection between the muscle force-length characteristic for a given activation threshold and the load characteristic (Fig. 1B).

The EP hypothesis was based on the key findings described above. With further development, it has been more recently called the threshold or referent control theory of motor actions (Feldman 1986, 2015). These names emphasize different aspects of the same theory: the EP concept characterizes the interaction of body segments with environmental forces, whereas the concept of threshold control indicates the physiological mechanism that influences this interaction via indirect changes in muscle activation and force. The spatial threshold also represents the referent (origin) point in the spatial (length-dimensional) frame of reference in which α-MNs function, thus emphasizing the role of spatial frames of reference in the control of actions.

The theory integrates different ways of achieving muscle activation and relaxation by defining the dynamic velocity-dependent muscle activation threshold (λ^*) such that the muscle is activated if the difference between the actual (x) threshold length becomes positive:

$$x - \lambda^* > 0 \qquad (1)$$

Threshold λ^* decreases with increasing stretch velocity (v) such that, to a first approximation,

$$\lambda^* = \lambda - \mu v \qquad (2)$$

where μ is the sensitivity (gain) of the threshold to velocity.

According to these formulas, the initially silent muscle (when $x - \lambda^* < 0$) can be activated either by passive stretching (by increasing x) or centrally (by decreasing λ). Physiologically, λ^* represents the dynamic stretch reflex threshold (DSRT), whereas its velocity-independent component, λ, is the tonic stretch reflex threshold (TSRT). Parameter μ is likely defined by the level of activity of γ-dynamic MNs. The TSRT differs from DSRT: the latter indicates phasic (transient) muscle activation that can be achieved by a rapid muscle stretch at high velocity, as occurs during the tendon reflex response.

To enable the muscle to relax or balance various loads at any joint angle within the joint biomechanical limits, the range of central regulation of λ or respective angular threshold (R) has to exceed the biomechanical limits (Fig. 1A). For example, suppose a subject is asked to fully relax arm muscles, and then elbow flexors are stretched by slow passive elbow extension. To prevent activation of flexor muscles within the biomechanical range, the TSRT should be shifted beyond the maximal value of this range (Fig. 1A). Conversely, to activate flexor

muscles at the biomechanically minimal length, the TSRT should be shifted below the minimal biomechanical angle. Thus the full range of changes in the threshold (TSRT$_-$, TSRT$_+$) exceeds the biomechanical range of changes in the joint angle.

In contrast, in poststroke subjects with spasticity, the flexor threshold range is diminished and at rest, i.e., despite the instruction to fully relax arm muscles, the maximal threshold for muscle activation (TSRT$_+$) lies abnormally within the biomechanical joint range (Fig. 2*A*, *top* horizontal bar; Levin et al. 2000; Mullick et al. 2013). As a result, passive extension that stretches the flexors beyond their activation threshold evokes muscle resistance that usually persists from the threshold angle to the full range extent, which is clinically defined as spasticity (Fig. 2*A*, open section of *top* horizontal bar; Levin and Feldman 1994).

In this scheme, the angular range in which spasticity is present at rest is called the spasticity zone (Fig. 2*A*). Although determined during passive stretching, the spasticity zone affects the production of voluntary movements. In particular, during voluntary extension of the joint, additional extensor muscle activity occurs to overcome resistance of spastic flexors, manifested as abnormal agonist-antagonist coactivation (Levin et al. 2000; Mullick et al. 2013). In the remaining part of the range, called the active control range, the ability to produce movement is characterized by normal patterns of agonist-antagonist muscle activation (Fig. 2*A*, filled section of *top* horizontal bar; Levin et al. 2000). Indeed, the active control range can be restricted by the presence of a spasticity zone in the elbow flexors as well as in the extensors (Musampa et al. 2007).

The capacity for motor learning may be preserved after stroke but may be masked by the presence of spasticity zones. To test this hypothesis, we studied motor adaptability in patients when elbow movements were made within the patient's active control range (Fig. 2*A*, *middle* horizontal bar) compared with movements that crossed into the spasticity zone (Fig. 2*A*, *bottom* horizontal bar). The hypothesis would be confirmed if individuals with stroke could correct errors more consistently when movements were restricted to the active control range than when movements were made beyond this range. We also anticipated that error correction performance would be correlated with levels of clinically assessed UL motor impairment and activity limitations, aside from spasticity. Preliminary results have appeared in abstract form (Subramanian et al. 2017).

MATERIALS AND METHODS

Participants and Clinical Testing

Based on a sample size estimate of 8 subjects per group (G*Power 3; Faul et al. 2007); effect size = 1.23, α = 0.05, 80% power) and a 30% dropout rate, 12 subjects with UL hemiparesis were included in this cross-sectional study if they *1*) had sustained a stroke in the dominant left hemisphere ≥6 mo previously, *2*) were aged 40–80 yr, *3*) had spasticity in the UL [≥5/16 on the Composite Spasticity Index (CSI; Levin and Hui-Chan 1993)] and full passive range of elbow joint motion, and *4*) scored 3/7–6/7 on the arm subscale of the Chedoke-McMaster Stroke Assessment (CMSA; Gowland et al. 1993). Participants were excluded if they had *1*) cerebellar (Boyd and Winstein 2004), brain stem (Kruger et al. 2007), or thalamic (Exner et
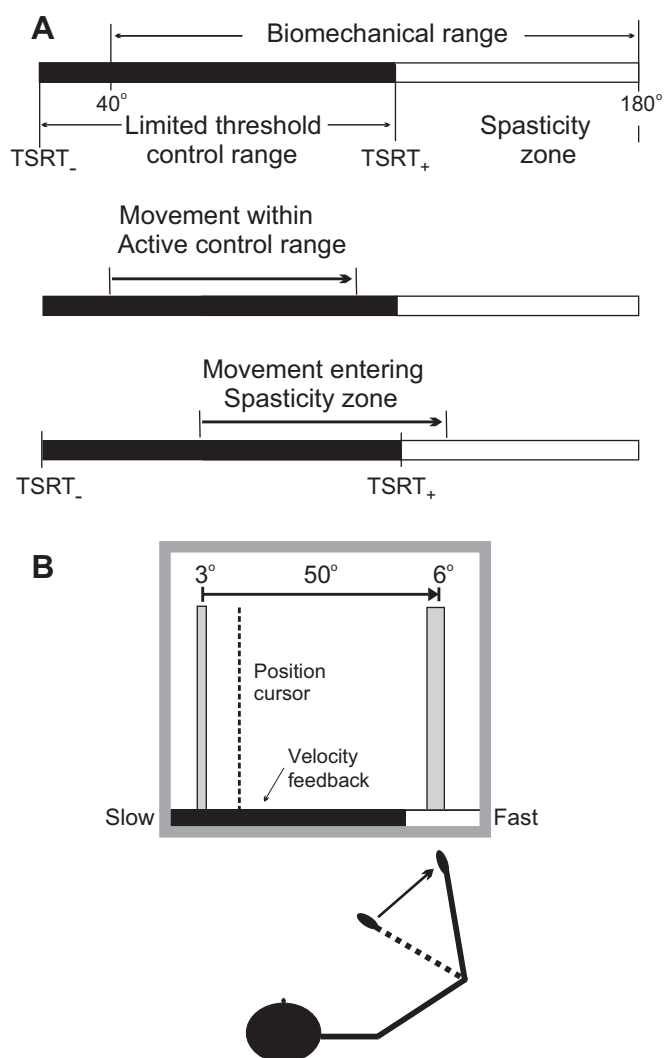

Fig. 2. Influence of spasticity zone on voluntary movement, and experimental setup. *A*: in patients with stroke, the range of regulation of the TSRT is limited. *Top*, in this example, the upper limit of the threshold (TSRT$_+$) for flexors lies within the biomechanical range, resulting in a flexor spasticity zone and a restricted active control zone. *Middle*, active elbow extension within the active control zone can be produced with normal reciprocal muscle activation patterns. *Bottom*, elbow movements extending to the right of TSRT$_+$ (i.e., entering the spasticity zone) would necessitate overcoming resistance of spastic flexors and would be associated with flexor/extensor coactivation. *B*: experimental setup. Participants made a single elbow extension movement from an initial (*left* vertical bar) to a final target window located 40°–50° away. The initial and final targets were 3° and 6° windows, respectively. The actual position of the arm was shown by a cursor (dashed vertical line) that moved across the screen. The horizontal bar at the bottom of the screen provided participants with velocity feedback at the end of each trial. Error correction behavior in the paradigm shown in Fig. 1*B* in the participants with stroke was evaluated in 2 sessions, spaced 1 wk apart, when elbow extensor movements were restricted to the active control range or when they crossed from the active control to the spasticity zone.

al. 2001) lesions that might affect motor adaptation and learning or *2*) shoulder pain (≤4/7 on the CMSA pain subscale; Niessen et al. 2009). Because they had to interact with a computer-presented visual scene, participants were also excluded if they had *3*) marked visuospatial neglect (line bisection test; Menon and Korner-Bitensky 2004), visual field deficits [chart review, medical consults, or proprioceptive deficits in the shoulder or elbow: <8/10 on the proprioception subscale of the Fugl-Meyer Assessment (FMA); Lin et al. 2004], because impaired proprioception results in lower rates of motor learning (Vidoni and

Boyd 2009). Age-matched control subjects recruited from the community ($n = 8$) were included if they had no orthopedic or neurological abnormalities interfering with participation. All participants provided written informed consent by signing forms approved by the institutional review boards of the Center for Interdisciplinary Research in Rehabilitation of Greater Montreal and Hôpital de réadaptation Villa Medica, according to the Declaration of Helsinki. Movements were performed with the dominant right arm.

Participants with stroke underwent a clinical evaluation by a licensed physiotherapist using psychometrically sound assessments. Motor impairment level was assessed using the upper limb section of the FMA-UL (Fugl-Meyer et al. 1975), which rates voluntary movement, reflex activity, grasp, and coordination on a scale from 0 to 66, where higher scores denote lower levels of impairment. Spasticity was assessed using two clinical measures, the CSI and the TSRT. The CSI evaluates the amount of resistance to stretch at a moderate velocity of the passive elbow flexors and the excitability of the deep tendon reflex (biceps brachii) and wrist clonus (Levin and Hui-Chan 1993). CSI scores of 1–4, 5–9, 10–12, and 13–16 points denote no, mild, moderate, and severe spasticity, respectively. UL activity levels were assessed using the streamlined Wolf Motor Function Test–Functional Ability Scale (sWMFT-FAS; Bogard et al. 2009) as the mean value of 6 tasks scored on 6-point scales ranging from 0 to 5.

Tonic Stretch Reflex Threshold Measurement: Data Collection and Analysis

The Montreal Spasticity Measure (MSM; Calota et al. 2008) was used to measure the TSRT of elbow flexors, for which smaller TSRT angles denoted greater spasticity severity (Jobin and Levin, 2000). The TSRT has high interrater and intersubject reliability and low measurement error (Blanchette et al. 2016; Calota et al. 2008).

TSRTs were measured in an elbow flexor (biceps brachii, BB) while elbow extensors (triceps brachii, TB) were simultaneously recorded in the hemiparetic arm of patients sitting in a standard chair with their affected arm supported by dense foam pillows. Bipolar Ag-AgCl disposable surface electrodes (Ambu Blue Sensor P; Ballerup, Denmark) were placed over each muscle belly according to Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) guidelines (Freriks et al. 1999) after the skin was cleaned and abraded. A reference electrode was positioned on the acromion. Electromyographic (EMG) signals were amplified for a total gain of 7,000 and sampled at 1,000 Hz. Elbow angular position was recorded with a servo-type rotational position potentiometer (P2200; Novotechnik U.S., Southborough, MA) with the axis of rotation positioned over the lateral epicondyle. The proximal and distal electrogoniometer arms were secured with straps to the arm and forearm, respectively. Before testing, participants performed a maximal voluntary contraction of elbow flexors to adjust the agonist EMG gain. Baseline EMG activity (with the participant at rest) and the initial elbow angle were determined and recorded. The initial elbow angle corresponded to the fully flexed elbow position without compressing the BB EMG electrodes with the forearm (~30°–40°, where full elbow extension with the arm extended equaled 180°). In this position, all poststroke subjects were able to relax elbow flexors. These muscles were then stretched manually by moving the forearm from the initial position toward full extension at different velocities. Twenty stretches were performed with 10–30 s between stretches. The examiner was trained to perform the stretch with a single bell-shaped velocity profile as is usually done in the clinic, but to vary the stretch velocity. Before each stretch, an auditory signal consisting of seven consecutive tones decreasing in pitch was generated by the computer to indicate how fast to stretch the arm. Each series of tones indicated to stretch at one of three speeds: slow, medium, or high velocity that varied between ~10 and ~400°/s. Accurate reproduction of the instructed stretch velocity was not required because a wide distribution of stretch velocities was desired to calculate the TSRT

(see below). The inter- and intra-evaluator reliability of TRST measurements for elbow flexors has been reported as moderately good (Calota et al. 2008). In addition, the split-half reliability (Spearman-Brown coefficient; Stanley 1971) of TSRT measures was 0.95, indicating excellent reliability of the TSRT measure between sessions. Subjects were instructed to relax completely without assisting or resisting the passive angular displacement. Although all poststroke participants were able to relax shortened flexors at the initial position, they were unable to prevent activation of flexors in response to stretching beyond a specific angle depending on the velocity of stretch.

Full details of the data analysis can be found in Blanchette et al. (2016). Briefly, the MSM software processed the angular signals from the goniometer and determined angular velocities in real time through differentiation. For each stretch, the stretch response onset was determined, based on the EMG envelope as the point at which the envelope rose and remained above 3 SD of the baseline for a minimum of 25 ms. The joint angle and velocity at which EMG was initiated were determined (Fig. 3) and identified as the dynamic stretch reflex threshold (DSRT) for that stretch velocity. In all stroke subjects, once the joint angle corresponding to the DSRT was surpassed, the elbow flexor muscle remained activated until the end of the stretch (Fig. 3). Diversity of stretch timing ensured that the DSRTs were determined over a wide range of velocities. This was important because the MSM
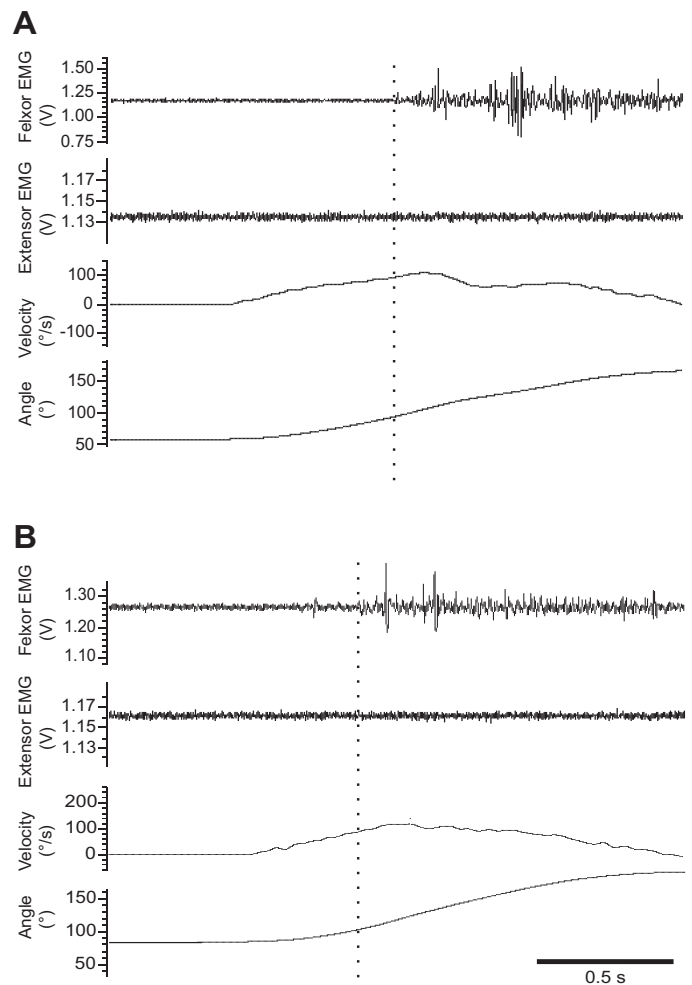

Fig. 3. Examples of individual stretches in 2 stroke subjects, illustrating the EMG activity in elbow flexors and extensors (*top* 2 traces in *A* and *B*). Stretch velocity was 105°/s (*A*) and 120°/s (*B*). Dynamic stretch reflex angles (dotted vertical lines) were 93.2° (*A*) and 107.1° (*B*). Raw filtered EMG is shown for illustrative purposes. Thresholds were defined from EMG envelopes (see text).

computed the best fit regression line through the DSRTs on a velocity/angle plot and determined the intercept of the regression line with the angular (*x*) axis (see Fig. 4*B*). This intercept represented the TSRT angle (TSRT = DSRT for zero velocity) that was used to demarcate the active control range from the spasticity zone.

The measured values of thresholds indicated the position (angle) at which spasticity began to gradually increase when the muscles were stretched, despite the instruction to fully relax arm muscles. This response is in contrast to healthy subjects, in whom no stretch responses are observed in the entire biomechanical range when the person is fully relaxed. Note that in Fig. 2*A*, only the range in which spasticity is present is indicated (beyond the TSRT level) without indicating threshold dynamics in response to stretching (DSRTs). When muscles are stretched at higher speeds, spasticity can be observed to the left of the TSRT, because the activation threshold is velocity dependent (Levin et al. 2000).

One-Trial Learning Paradigm (Theoretical Scheme)

We used a one-trial learning experimental paradigm in which subjects had to adapt 40°–50° elbow extension movements to reach a
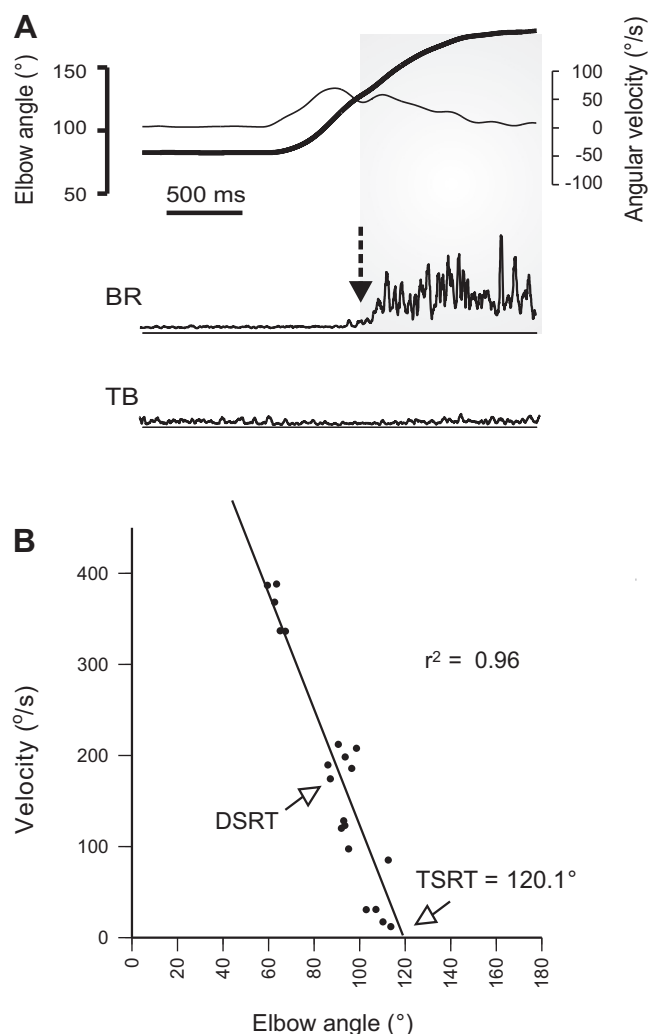

6° target in the presence or absence of an opposing spring-like load (Fig. 1*B*; Dancause et al. 2002). Subjects first learned to make rapid and accurate movements with no opposing load (line L = 0). Based on the equilibrium point (EP) theory of motor control (Feldman 1966, 1986, 2015), the initial movement from point *i* to point *a* results from a central shift in muscle activation thresholds (λ) of flexor and extensor muscles in the same direction (the reciprocal, R, command). This central shift is associated with a shift, in the angular domain, of the net torque/angle characteristic generated by intrinsic muscle and reflex properties (thick solid diagonal lines in Fig. 1*B*). The R command can be combined with a C command that changes flexor and extensor thresholds to increase the slope (stiffness) of the characteristic. The final EP of the system is the point of intersection of the muscle characteristic with that of the load, *point a* in the case of zero load. By adjusting shifts in muscle activation thresholds (i.e., control variables), the system brings the EP into the target window (*right vertical gray bar*) to reach the target. If the same shifts in control variables are reproduced (invariant strategy) when the external load is modified (e.g., from L = 0 to L > 0), the EP of the system will be outside the target zone, resulting in an undershoot error (*point b*). Once perceived, the error could be corrected by a secondary movement produced by shifting flexor and extensor thresholds further to the right via the R command, to specify a new EP (*point d*) inside the target window. If the new load condition is repeated, the system could reproduce, with small variations, the new threshold shifts to maintain movement accuracy. Thus, by reproducing the control variables after correction, specified in the previous trial (recurrent strategy), the system could maintain movement accuracy after one trial. Indeed, after several trials in the new load condition, if the load were unexpectedly removed, an overshoot error (*point c*) would occur that could be subsequently corrected by returning to the first pattern of control variables.

Experimental Procedures

Subjects sat on an adjustable height chair in front of a computer screen (Fig. 2*B*). The trunk was fastened to the chair back by wide Velcro straps to avoid compensatory trunk movements often observed in individuals with stroke during arm movements (Cirstea and Levin 2000). The right forearm and part of the hand were secured to a horizontal manipulandum in the neutral position using a polypropylene bivalve splint with the shoulder in ~70° flexion and 70° abduction. The manipulandum partially supported the weight of the arm against gravity, and the splint assured a firm grip on the handle for those with disturbed grasping ability. The manipulandum was controlled in the horizontal plane by a torque motor (MT2000; Mavilor Motors, Barcelona, Spain). The inertia of the manipulandum was I = 0.03 kg·m² (for comparison, the inertia of the forearm with hand and cast was ~0.05 kg·m²). Three safety features incorporated into the apparatus automatically switched off the torque motor: if the segment reached the limits of the manipulandum range indicated by signals from interrupters placed near the movement limits (~40° and 170°), the manipulandum speed approached the natural speed limits of the arm (2 m/s), or a "panic" button held by the subject was depressed. Finally, the torque of the motor was limited to 30% (15 Nm) of its maximal output.

At the beginning of each trial, the subject placed a vertical cursor within a 3° target window shown on the computer screen (initial position) by moving the handle of the manipulandum (Fig. 2*B*). In this position, the hand was ~30 cm in front of the sternum. The task was to make a rapid 40°–50° elbow extension to move the cursor from the initial to a final 6° target. A 6° final target window was chosen to maintain a consistent index of difficulty (Fitts 1954). In addition, compared with healthy controls, noncorrected movements in post-stroke subjects are characterized by enhanced terminal overshoots and undershoots (lower decrements of decay; Levin and Dimov 1997). The target window was sufficiently wide such that noncorrected

Fig. 4. Identification of stretch reflex thresholds during passive stretching of elbow flexors. *A*: spastic muscle (Brachioradialis (BR)) begins to be activated (arrow) during passive stretching above a certain, velocity-dependent joint angle (DSRT). Triceps brachii (TB) was shortening during the passive elbow extension. *B*: tonic stretch reflex threshold (TSRT) at which spasticity begins to be manifested is determined from linear regression across DSRT points (TSRT = DSRT at zero velocity).

movements could terminate entirely within the window, thus preventing subjects from mistakenly considering terminal oscillations as movement errors. Participants had visual feedback of their moving arm and of the hand position from the cursor on the screen. To ensure that movements were made rapidly, subjects had to match a target velocity indicated by a horizontal line at the base of the screen.

Control subjects participated in one session and stroke subjects participated in two sessions. For control subjects, the initial elbow position was 90°, whereas full elbow extension was 180°. For each stroke subject, the initial position of the movement task depended on the location of the spasticity zone measured before each session (Table 1). An example of a single stretch (peak velocity 130°/s) in one stroke subject is shown in Fig. 4A. The DSRT for the stretch is the angle at which the onset of the BR EMG occurs at the respective stretch velocity (Fig. 4A, arrow). Figure 4B shows the plot of the DSRTs for 20 stretches in this subject and the value of TSRT corresponding to the intercept of the regression line through the DSRTs with the angular axis at zero velocity. In each stroke subject, the elbow flexor $TSRT_+$ was determined by using the MSM software to locate the angle demarcating the start of the elbow flexor spasticity zone. Each stroke subject participated in two experimental sessions, one in which the elbow movement was restricted to the angular zone below $TSRT_+$ (active control range) and one in which the elbow movement started in the active control range and ended within the spasticity zone. Session order was randomized, and sessions were conducted 1 wk apart to ensure washout of short-term learning effects.

For the one-trial learning experiment, before each session, participants practiced elbow extension movements from the initial to the final target without an opposing load. Practice continued until the task was performed without any corrective movements in 7 of 10 practice trials. Subjects then performed elbow extension movements in 16 blocks of 6–10 trials each for a total of 96–160 trials/session. In alternate blocks, a load-opposing elbow extension was introduced or removed. For trials with load, the load was zero when the arm was in the initial position and increased linearly as a function of elbow position after the initial 5° of movement. To compare results between and within groups and to ensure that movements were not affected by weakness in elbow extensors, the load was chosen as a percentage (30%) of the participant's maximal voluntary contraction (MVC; see Table 1) with the arm held in the target position for each session. The MVC torque (Nm) was calculated as the best of three trials using a handheld dynamometer (model 01163; Manual Muscle Testing System, Lafayette, IN) in kilograms multiplied by the forearm length (m) between the dynamometer placement and the elbow axis of rotation. The stiffness constant of the load ranged from 0.10 to 0.15 Nm/° corresponding to 5.0–7.5 Nm at the target position for the stroke group, and from 0.12 to 0.15 Nm/° corresponding to 6.0–7.5 Nm at the target position for the control group.

Participants were instructed to make rapid movement and to correct errors as fast as possible resulting from the unexpected introduction or removal of the load. The random number of trials per block combined with the delayed appearance of the load ensured that subjects did not anticipate the load condition in the forthcoming trial. Short rest periods (1–2 min) between blocks and longer breaks (5 min) after completion of four to five blocks were provided with more frequent and longer rest periods on request.

Elbow position (°) and velocity (°/s) were recorded with separate transducers (high-precision hybrid electromagnetic resolvers aligned with the torque motor shaft) and sampled at 100 Hz from 0.2 s before movement onset to the end of the trial.

Data Analysis

Trials in each block were classified according to the following criteria as 1) the test trial (T), first trial of the block in which the load condition changed; 2) trials P1 and P2, the second and third trials in

Table 1. *Demographic characteristics, clinical scores, tonic stretch reflex threshold (TSRT) values and initial angular positions for TSRT measurement and elbow extension trials of individual stroke subjects*

Subject	Age, yr/Sex, M/F	Time Since Stroke, mo	Lesion Location	CMSA Arm Subscale (/7)	FMA-UL (/66)	CSI (/16)	sWMFT FAS (/5)	Spasticity Zone TSRT: Initial Position, °	Active Control Range TSRT: Initial Position, °	MVC/Load, Nm
S1	54/M	32	L ischemic MCA: posterior limb of internal capsule	3	23	14	1.33	110:75	120:70	15.5/4.7
S2	58/M	19	L ischemic MCA: posterior limb of internal capsule	3	24	13	2.25	140:100	135:80	15.2/4.7
S3	46/M	28	L hemorrhagic: putamen	3	31	10	2.67	140:100	145:95	24.9/7.5
S4	67/M	275	L subarachnoid hemorrhage: frontal regions and central gray matter	3	32	12	1.83	159:115	146:85	15.6/4.7
S5	73/M	8	L hemorrhagic: central gray matter	3	33	11	2.50	130:95	135:75	14.5/4.4
S6	71/F	58	L ischemic PCA: occipital lobe	3	36	7	3.30	125:90	135:90	7.9/2.4
S7	58/M	60	L ischemic MCA: corona radiata	5	36	7	3.83	126:85	130:75	39.4/11.8
S8	49/M	39	L hemorrhagic: Sylvian fissure	5	43	6	2.83	140:95	135:90	19.4/5.8
S9	69/M	39	L ischemic MCA	4	46	8	4.00	140:100	148:95	24.4/7.3
S10	52/F	54	L ischemic MCA: basal ganglia	3	51	11	3.50	152:110	150:90	16.6/5.0
S11	44/M	10	L hemorrhagic: basal ganglia and external capsule	3	53	9	2.83	110:65	115:60	23.7/7.1
S12	61/M	35	L ischemic MCA	6	62	6	5.00	145:100	145:90	22.6/6.8

M, male; F, female; CMSA, Chedoke McMaster Stroke Assessment; FMA-UL, Fugl-Meyer Assessment, upper limb section; CSI, Composite Spasticity Index; MCA, middle cerebral artery; PCA, posterior cerebral artery; sWMFT FAS, streamlined Wolf Motor Function Test-Functional Assessment Scale; MVC, maximal voluntary contraction (Nm), defined as MVC (kg) × forearm length (m).

the changed load condition; and *3*) C trials, all other trials in the same load condition.

Individual trials were aligned at the onset of elbow movement, defined as the time at which movement velocity exceeded and remained above 5°/s for a period of 50 ms. For each trial, the initial and final positions of the primary movement, as well as the final position of the corrective movement, if present, were identified in T, P1, P2, and C trials on an interactive display using angle-time and velocity-time curves. The combination of position, velocity, and time at which a correction of the primary movement was initiated was identified from inflection points in the velocity-angle (phase) diagrams. Specifically, phase diagrams of uncorrected movements had smooth shapes with relatively small terminal loops within the target window when the movement speed approached zero. In contrast, movement corrections occurred when the phase diagrams approached zero velocity outside the target window with subsequent secondary motions terminating within the target window (see arrows in Figs. 6 and 7). The positions associated with the inflection points were used to evaluate movement errors.

Movement errors were defined as an undershoot error when the primary movement ended before reaching the target window or an overshoot error when it ended beyond the target window. The final angular position in each trial was expressed as a percentage of the total movement distance for each subject. Error correction behaviors were classified into four distinct patterns (Fig. 5): *pattern 1* (Fig. 5*A*), an error occurred in the T trial that was corrected in the subsequent trial (P1), and movements were made without errors in remaining trials of the block (P2 and C trials); *pattern 2* (Fig. 5*B*), errors occurred in T and P1 trials that were corrected in P2, and movements in C trials were made without errors; *pattern 3* (Fig. 5*C*), an error occurred in the T trial that was corrected within the next one to two trials, but more than one error occurred in the subsequent C trials (inconsistent behavior); and *pattern 4* (Fig. 5*D*), no clear error correction pattern was identified.

Error correction patterns in individual subjects were further dichotomized into two categories: *category 1*, which included *patterns 1* and *2*, and *category 2*, which included *patterns 3* and *4* (Dancause et al. 2002). The primary outcome was the proportion of blocks in which the error was corrected in one to two trials. Secondary outcomes included elbow angular velocities and the extent of undershoot and overshoot errors (i.e., absolute difference from the final target position).

Statistical Analysis

Descriptive statistics highlighted main demographic characteristics. Data were verified for normality of distributions with the Kolmogorov-Smirnov test, and homogeneity of variance assumptions using Levene's tests. Final positions of the T, P1, P2, and C trials were analyzed with mixed-model ANOVA with repeated measures with one between-subject factor (group: healthy, stroke) and/or two within-subject factors (type of trial, load). Models comparing values in the stroke group between sessions included two (type of trial, load) or three within-subject factors (session, type of trial and load). Preplanned post hoc testing was done with Holm-Bonferroni corrections for multiple comparisons (Holm 1979).

Because of the ordinal nature of the data, error correction patterns were compared between groups and sessions (in the stroke group) using Wilcoxon signed-rank tests and Mann-Whitney *U*-tests, respectively. Significance was set at $P < 0.05$. Spearman rank-order correlation coefficients (2-tailed) estimated the strength of associations between the primary outcome and clinical assessments of motor impairment and activity limitations in the stroke group, adjusted for multiple comparisons using Holm-Bonferroni corrections.

RESULTS

Twenty participants (control: $n = 8$, age 63.5 ± 9.1 yr; stroke: $n = 12$, age 57.8 ± 9.6 yr; Table 1) completed all clinical and experimental sessions. Participants with stroke had moderate UL motor impairment (FMA-UL: 38.8, 95% CI: 31.2–46.3; CSI: 9.4, 95% CI: 7.6–11.2) and activity deficits (sWMFT-FAS: 3.0; 95% CI: 2.3–3.6). Only participant (S2) could not complete all of the blocks in *session 1*. For this subject, data collection was terminated in *session 2* after 9 blocks, similar to the number of blocks completed in *session 1*. The maximal load torques were 19.98 ± 7.93 Nm for the stroke group and 21.01 ± 4.48 Nm for the controls. The load values corresponding to 30% MVC were 5.99 ± 2.38 and 6.30 ± 1.34 Nm, respectively (Table 1).

Control Group

Velocity/angle phase plots for one block of six trials when the condition changed from nonloaded to loaded trials and from loaded to nonloaded trials for one control participant are shown in Figs. 6 and 7. respectively. In healthy subjects, movements in the nonloaded and loaded C trials (Figs. 6*A* and 7*A*) were made without corrections. The hand reached the final position after a smooth transition, a transient overshoot, and small terminal oscillation due to the natural underdamping dynamics of the system. The average velocities of the elbow movements in the C trials were 340.4 ± 62.8°/s for nonloaded trials and 342.6 ± 65.6°/s for loaded trials.

End positions before corrections across four blocks of trials for the same participant are shown in Fig. 8*A*. The change in
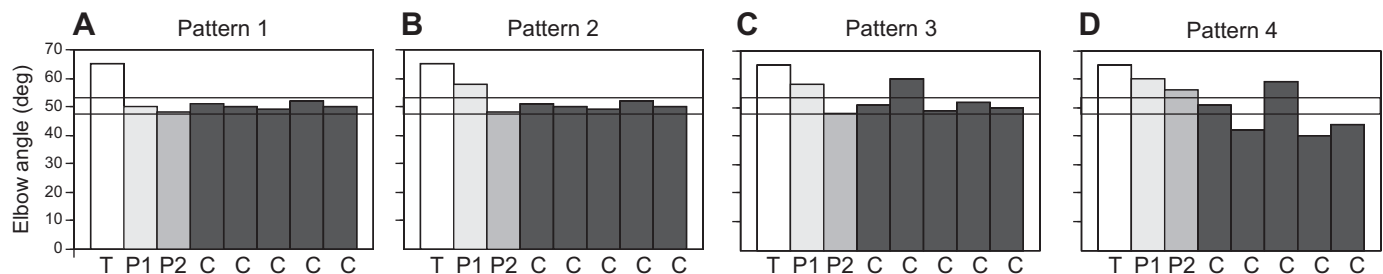

Fig. 5. Classification of error correction patterns. Shown are patterns of overshoot error corrections when conditions changed from load to no load (similar patterns for undershoot error corrections when changing from nonloaded to loaded conditions). *A*: *pattern 1*. The error in the test (T) trial (white bar) is eliminated in the next trial (P1; light gray bar), and behavior is consistent in all subsequent control (C) trials (black bars) within the same block. *B*: *pattern 2*. The error in the T trial is partially corrected in the first posttest trial (P1) and eliminated in the next trial (P2; dark gray bar). Behavior is consistent in subsequent C trials within the same block. *C*: *pattern 3*. The error in the T trial is completely corrected by the second posttest trial (P2), but the behavior is inconsistent in subsequent trials (more than one error in C trials). *D*: *pattern 4*. No stable error correction behavior occurs in a block of trials. In *A–D*, the horizontal lines show the limits of the 6° wide target window.
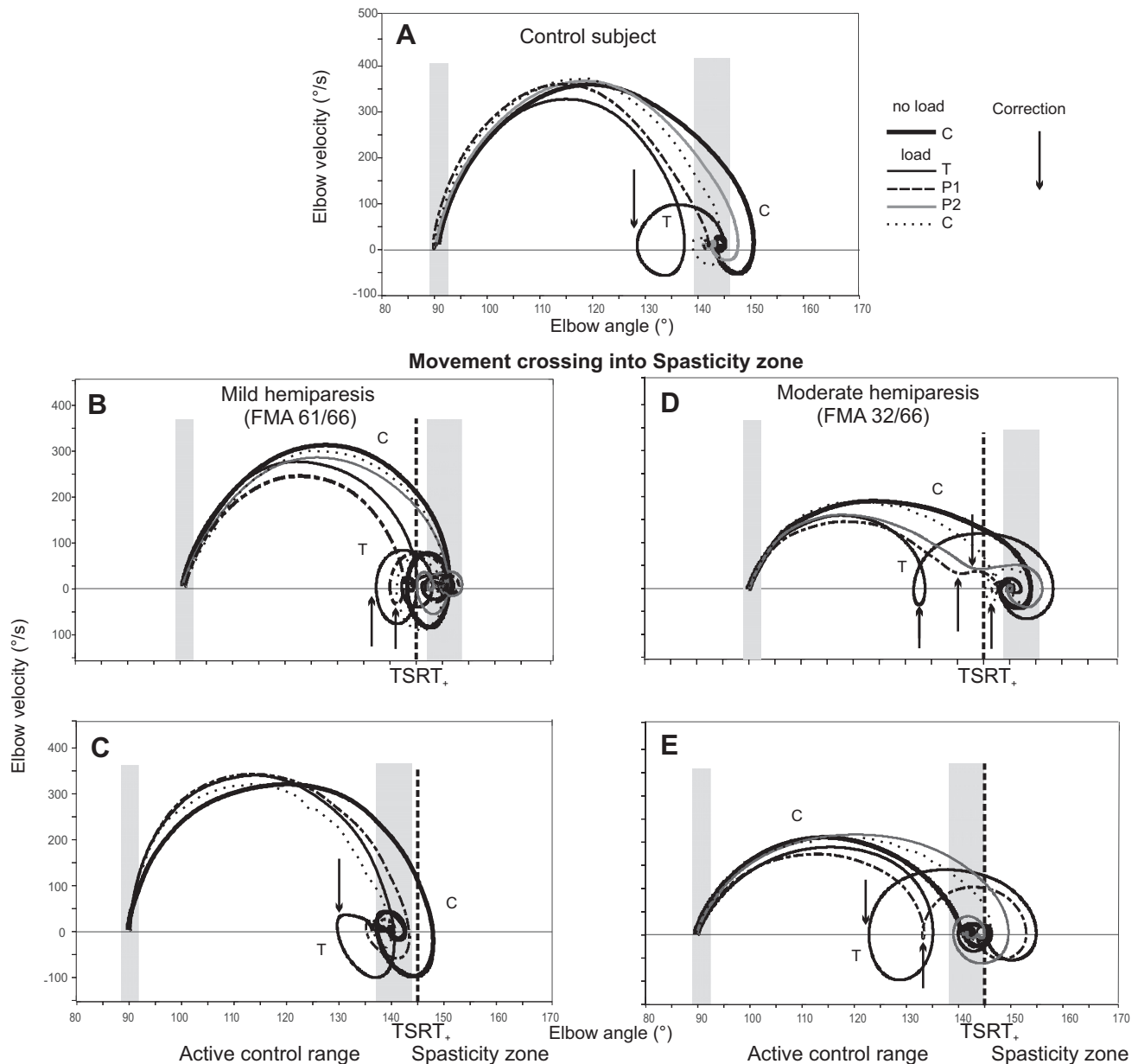
Fig. 6. Representative phase (velocity vs. angle) diagrams for 1 block of trials in which the no-load condition suddenly changed to the load condition in a control subject (*A*) and in subjects with mild (*B*, *C*) and moderate (*D*, *E*) upper limb hemiparesis when the movements ended in the spasticity zone (*B*, *D*) or were restricted to the active control range (*C*, *E*). The *left* and *right* vertical bars indicate the initial (3°) and final (6°) targets, respectively. The vertical dashed line indicates the position of the TSRT located within (*B*, *D*) or beyond (*C*, *E*) the active control range. C (thick curve) is the last trial in a block of trials in the no-load condition. T, P1, P2, and C (dashed curve) are sequential trajectories in the subsequent block of trials in the load condition. Inflection points indicating corrective movements are marked by vertical arrows.

load condition from nonloaded (open bars) to loaded (shaded bars) trials and vice versa resulted in undershoot and overshoot errors, respectively, in T trials, which were corrected in P1 or P2 trials (*patterns 1* or *2*) in the same condition across the majority of blocks.

The angular distance from the initial to the final window was chosen according to the available active control range, individually for each subject. These distances ranged from 40° to 50°. For group comparisons, the elbow angles at which corrections occurred were normalized and expressed as a percentage of the inter-window angular distance. For the whole group, when the condition changed from nonloaded to loaded trials, the mean values of the elbow positions at which the corrections were initiated in T, P1, P2, and C trials were $76.1 \pm 6.7\%$ (undershoot), $93.8 \pm 2.6\%$, $97.8 \pm 1.8\%$, and $98.0 \pm 1.1\%$, respectively. For the opposite condition (change from loaded to nonloaded trials), mean values were $116.3 \pm 8.7\%$ (overshoot), $102.8 \pm 4.6\%$, $100.0 \pm 1.7\%$, and $99.3 \pm 1.8\%$, respectively. Final positions differed significantly for both load conditions (load $\times$ trial interaction; $F_{3,56} = 65.512$, $P < 0.001$). For trials with opposing loads, T trial positions differed from P1 ($t_7 = -6.275$, $P < 0.001$), P2 ($t_7 = -9.329$, $P < 0.001$), and C trials ($t_7 = -9.064$, $P < 0.001$). For trials with no load, T trial positions differed from P1 ($t_7 = 6.995$, $P < 0.001$), P2 ($t_7 = 5.821$, $P = 0.001$), and C trials ($t_7 = 5.361$, $P = 0.001$).
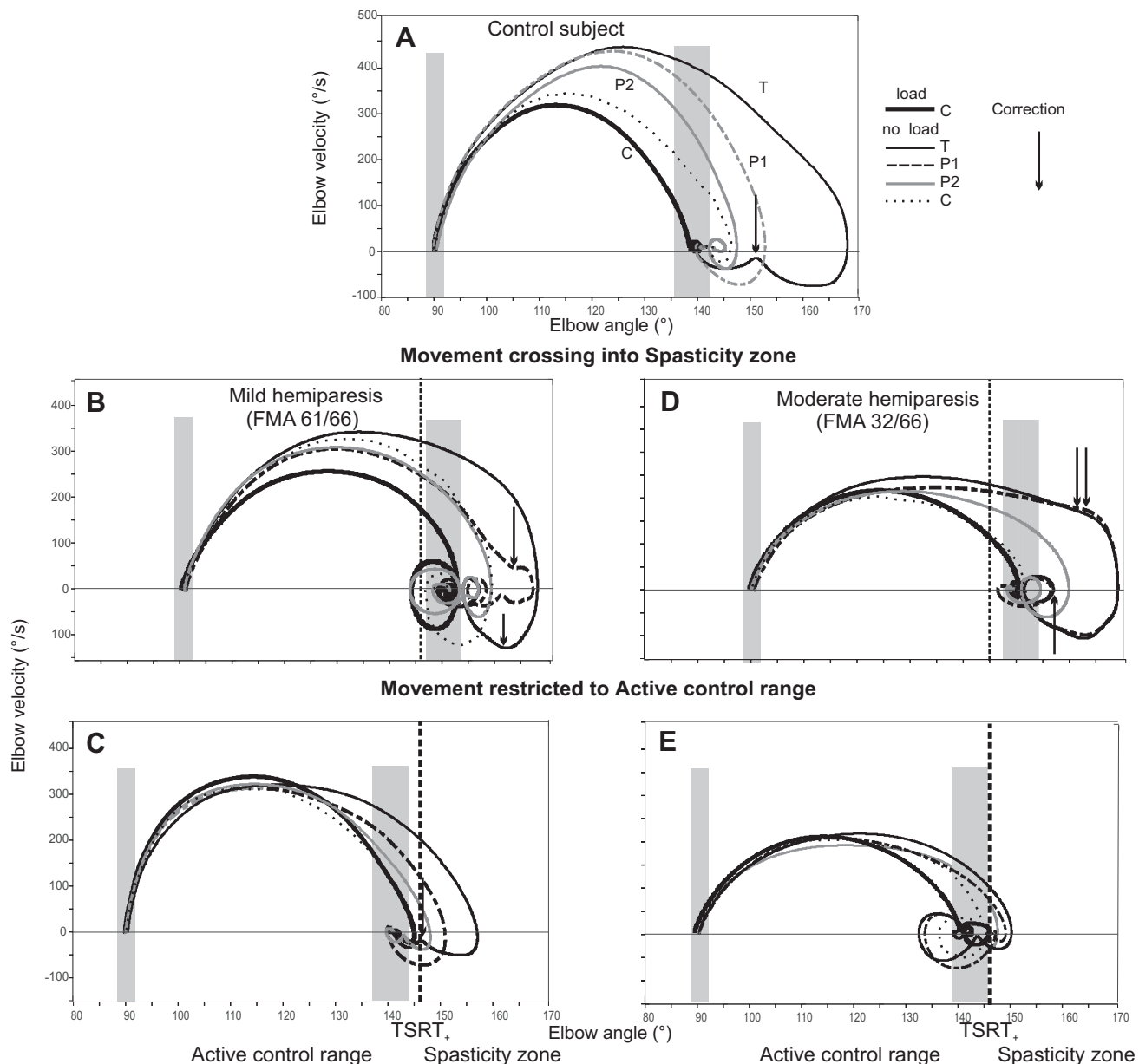
Fig. 7. Representative phase diagrams for 1 block of trials in which the load condition suddenly changed to the no-load condition in a control subject (*A*) and in subjects with mild (*B*, *C*) and moderate (*D*, *E*) upper limb hemiparesis when the movements ended in the spasticity zone (*B*, *C*) or when they were restricted to the active control range (*C*, *E*). C (thick curve) is the last trial in a block of trials in load conditions. T, P1, P2, and C (dashed curve) are sequential trajectories in the subsequent block of trials in the no-load condition. Other labels are as defined in Fig. 6.

All control participants corrected movement errors in one to two trials (*patterns 1* and *2*) in the majority of the blocks (76.7%), with only a small proportion of *patterns 3* and *4* (23.3%; Fig. 9, *A* and *D*, Table 2). The average velocities of the elbow movements in T, P1, and P2 trials for each load condition were similar to the velocities of the C trials.

Stroke Group

Initial elbow angles for extension movements made into the spasticity zone and the active control range, TSRTs as well as magnitudes of opposing loads for each participant are listed in Table 1. Velocity distributions for movements made in the active control range and spasticity zone were similar (active

control: $209.6 \pm 56.2°/s$, 95% CI: 173.9–245.4; spasticity zone: $207.0 \pm 74.4°/s$, 95% CI: 159.7–254.2).

Movement in the Range Affected by Spasticity

Figure 6, *B* and *D*, shows phase plots of elbow extension movements made by two stroke subjects with mild and moderate impairment in trials in which the load was applied after movements were previously adapted to no load. Figure 7, *B* and *D*, shows phase plots in trials in which the load was removed after adaptation to loaded trials. In these examples, movements were not restricted to the active control range, and the final target (*left* shaded area) was located beyond the TSRT (dashed vertical line) such that movements ended in the spasticity zone. The final positions before correction across four
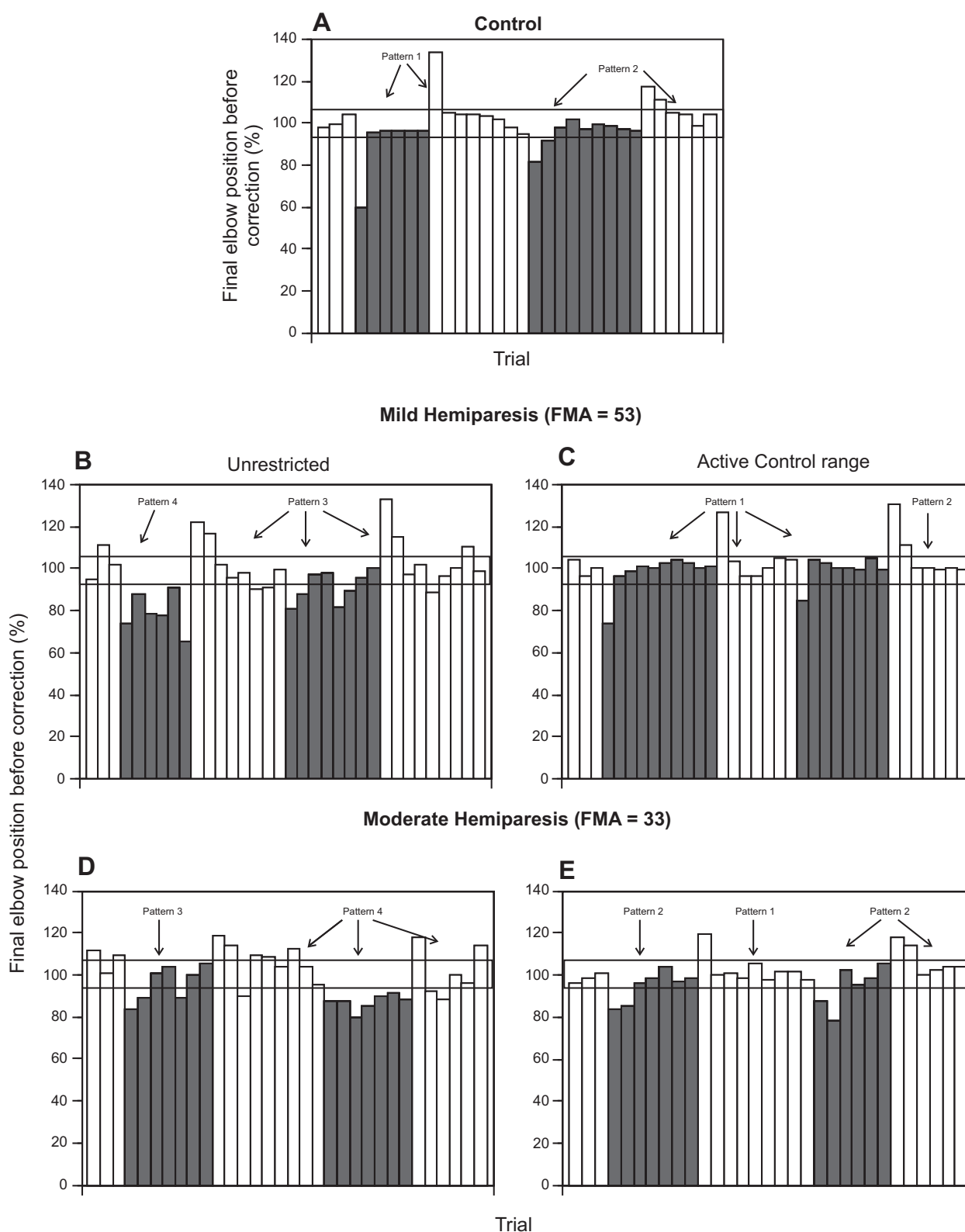
Fig. 8. Sequence of error correction patterns. Final normalized positions of the primary movement before correction are shown for 4 blocks of trials in a control subject (*A*) and in subjects with mild (*B*, *C*) and moderate (*D*, *E*) upper limb hemiparesis when the movements ended in the spasticity zone (*B*, *D*) or the active control range (*C*, *E*). Open and shaded bars represent trials without or with an opposing load, respectively. The error correction patterns are identified with arrows.

blocks of trials for the same two participants are shown in Fig. 8, *B* and *D*, respectively. Similar to healthy subjects, the change from the nonloaded to the loaded condition resulted in an undershoot error in all participants with stroke. The change from loaded to nonloaded conditions resulted in an overshoot error except in one subject (S1) in whom only undershoot
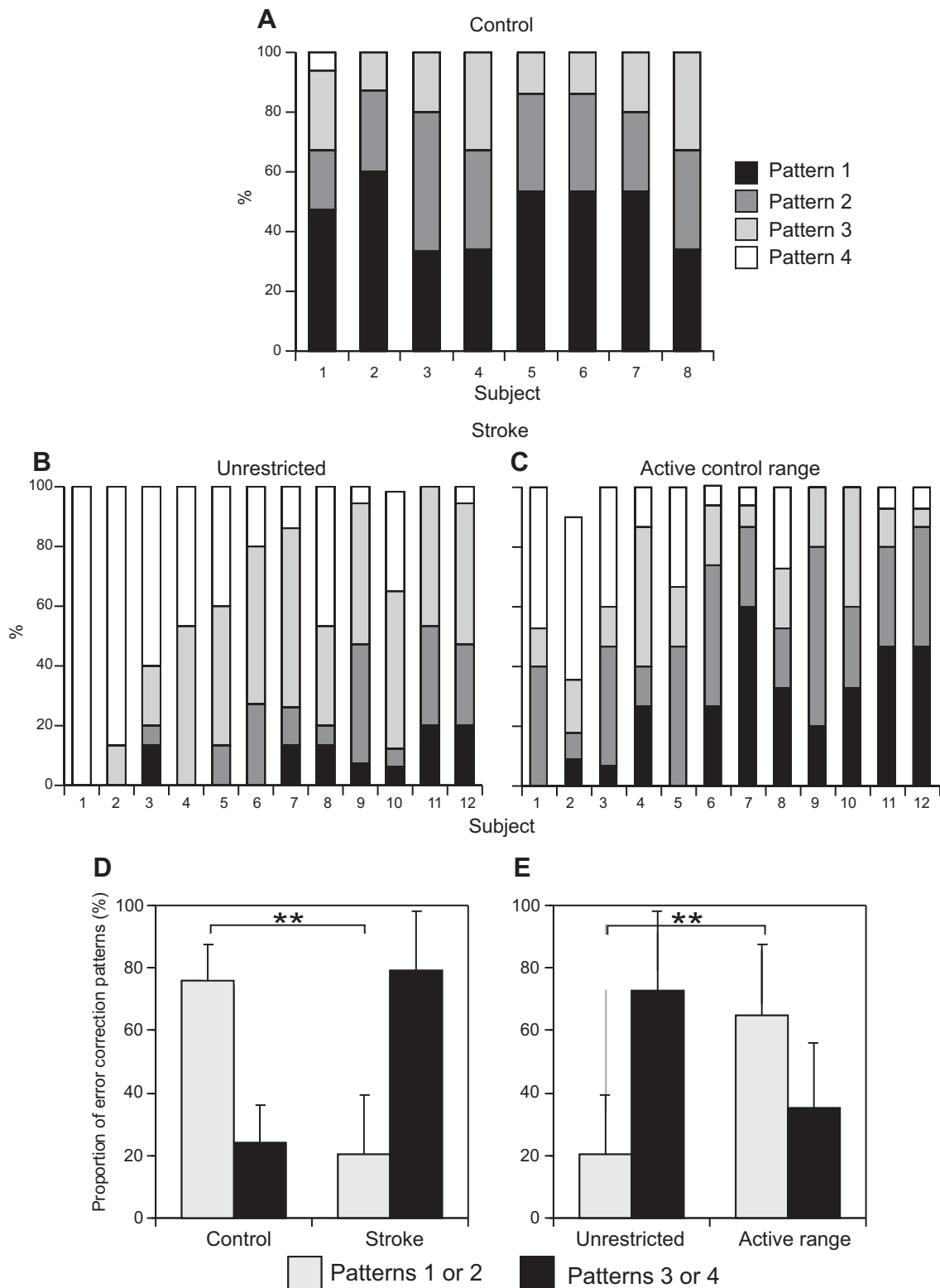
Fig. 9. Group error correction patterns. The frequency of occurrence of error correction patterns is shown in individual control subjects (*A*) and in stroke subjects when the movement included the spasticity zone (*B*) or was restricted to the active control range (*C*). The between-group and between-session (for the stroke group) differences in the combined mean proportions of *patterns 1* and *2* (light gray bars) compared with *patterns 3* and *4* (black bars) are shown in *D* and *E*, respectively. Error bars represent SD. **$P < 0.01$.

errors occurred when load conditions changed regardless of the type of load. Compared with controls, elbow movements were slower in the stroke group for the C trials (loaded: $206.3 \pm$ 77.2°/s, $Z = -3.086$, $P = 0.001$; nonloaded: 208.3 ± 75.1°/s, $Z = -3.009$, $P = 0.002$). Velocities did not differ in subsequent trials in the same load condition.

Error correction strategy. Participants with stroke were unable to correct errors in one smooth movement. After a primary error, subjects could make several corrective submovements in T, P1, and P2 trials, but these were small and difficult to objectively quantify (Figs. 6 and 7, *B* and *D*). When the condition changed from nonloaded to loaded trials, the mean normalized values of T, P1, P2, and C trials were 77.4 ± 6.9% (undershoot), 88.2 ± 9.9%, 89.7 ± 11.3%, and 92.3 ± 10.4%, respectively. For transitions from the loaded to the nonloaded condition, mean values were 113.5 ± 17.6% (overshoot), 102.3 ± 15.7%, 97.9 ± 13.9%, and 98.5 ± 12.7%, respectively. There was a significant load × trial interaction ($F_{3,88} = 6.964$, $P < 0.001$) such that for the loaded trials, the final positions of the T trials were significantly less than those of the P1 ($t_{11} = -4.196$, $P = 0.001$), P2 ($t_{11} = -3.946$, $P = 0.002$), and C trials ($t_{11} = -5.439$, $P < 0.001$). Similarly, the final positions of the nonloaded T trials, were significantly greater than those of the P1 ($t_{11} = 4.959$, $P < 0.001$), P2 ($t_{11} = 6.630$, $P < 0.001$), and C trials ($t_{10} = 6.786$, $P < 0.001$).

Error correction patterns. The final positions of the T, P1, P2, and C trials differed between groups depending on the load condition (load × trial interaction; $F_{1,144} = 23.062$, $P < 0.001$). Overall, the overshoot or undershoot error was corrected in control subjects by the P2 trial, whereas there was still a significant difference in the final position change values of P2 trials in the stroke group, especially in trial blocks with an opposing load (post hoc test; $t_{18} = 2.412$, $P = 0.026$). Participants with stroke were unable to consistently correct movement errors in P2 and C trials (Fig. 8, *B* and *D*) and had a lower percentage of blocks in which movement errors were corrected in one to two trials (mean: 20.5%, 95% CI: 8.5–32.6%; Fig. 9*B*) compared with controls (mean: 76.7%, 95% CI: 67.2–86.1%, $Z = -3.724$, $P < 0.001$; Fig. 9*D*).

Movement in the Active Control Range

Phase plots of elbow extension movements made entirely within the active control range by stroke subjects were qualitatively smoother and similar to those made by control subjects (Figs. 6 and 7, *C* and *E*). Examples of movements made by two stroke subjects with mild and moderate impairment are shown when the condition changed from nonloaded to loaded trials (Fig. 6, *C* and *E*) and from loaded to nonloaded trials (Fig. 7, *C* and *E*), respectively. The final positions before correction across four blocks of trials for the same two participants are shown in Fig. 8, *C* and *E*, respectively. The average velocity of movements in the C trials was 198.4 ± 52.4°/s for loaded trials and 203.5 ± 51.1°/s for nonloaded trials, which did not differ from velocities of movements made into the spasticity zone for the loaded ($Z = -0.235$, $P = 0.814$) or non-loaded trials ($Z = -0.235$, $P = 0.814$).

Error correction strategy. Movement errors in the active control range were generally corrected in one smooth movement. When the condition changed from nonloaded to loaded trials, mean normalized values for the final positions of T, P1, P2, and C trials were 76.2 ± 6.4% (undershoot), 90.3 ± 10.1%, 95.7 ± 7.4%, and 95.5 ± 7.5%, respectively. For the change in condition from loaded to nonloaded trials, mean values were 114.6 ± 7.8% (overshoot), 101.2 ± 9.7%, 100.5 ± 7.4%, and 99.9 ± 7.3%, respectively. Within this condition,

there was a significant load × trial type interaction ($F_{3,88} = 24.217$; $P < 0.001$). Final positions of the T trials significantly differed from those of P1 ($t_{11} = -5.747$, $P < 0.001$), P2 ($t_{11} = -8.706$, $P < 0.001$), and C trials ($t_{11} = -9.859$ $P < 0.001$) for the blocks with an opposing load. Similarly, for blocks with no load, the final positions of the T trials were significantly different from those of P1 ($t_{11} = 10.095$, $P < 0.001$), P2 ($t_{11} = 10.519$, $P < 0.001$), and C trials ($t_{11} = 10.154$, $P < 0.001$).

When the final positions of the T, P1, P2, and C trials were compared between sessions, a significant load × trial type interaction was present ($F_{3,175} = 21.46$, $P < 0.001$). The undershoot error in the P2 trials was significantly less (4.2%) in P2 trials for movements in the active control range compared with the spasticity zone (10.3%; post hoc test, $t_{11} = -2.540$, $P = 0.027$). The final positions of T trials for movements restricted to the active control range did not differ from those made in the spasticity zone for both loaded ($t_{11} = 0.557$, $P > 0.05$) and nonloaded conditions ($t_{11} = -0.295$ $P > 0.05$).

Error correction patterns. Error correction patterns immediately improved when movement was restricted to the active control range (Tables 2 and 3). In the majority of blocks, the error in the T trial was successfully corrected in one smooth movement by the P2 trial (25.8% by the P1 trial and 39.1% by the P2 trial), as shown in the representative phase plots in Figs. 6 and 7, *C* and *E*, and across a series of blocks in Fig. 8, *C* and *E*. Compared with movements made into the spasticity zone, the proportion of blocks in which movement errors were corrected in one to two trials was greater (Fig. 9*C*) when movement was restricted to the active control range (active control range: 64.9%, 95% CI: 51.6–78.1; spasticity zone: 20.5%, 95% CI: 8.5–32.6; $Z = -3.068$, $P = 0.002$), with a lower proportion of trials with incomplete error corrections (spasticity zone: 79.5%; active control range: 35.1%; Fig. 8*E*).

Relationship Between Error Correction Behavior and Clinical Measures

Regardless of the zone in which movements were made, levels of motor impairment and activity, but not extensor muscle strength, were correlated with movement correction ability. The proportion of blocks in which overshoot or undershoot errors were corrected in one to two trials was highly correlated ($P < 0.01$) with impairment [spasticity/active: FMA: $\rho(10) = 0.87/0.79$], resistance to stretch [CSI: $\rho(10) = -0.71/-0.87$], and activity levels [sWMFT-FAS: $\rho(10) = 0.72/0.83$].

Table 2. *Error correction patterns in the control group*

	Error Correction Pattern, %			
Subject	*Pattern 1*	*Pattern 2*	*Pattern 3*	*Pattern 4*
1	47	20	27	6
2	60	27	13	
3	33	47	20	
4	34	33	33	
5	53	33	14	
6	53	33	14	
7	53	27	20	
8	34	33	33	

Data are proportions of error correction *patterns 1–4* used by each healthy subject in the control group.

Table 3. *Error correction patterns in the stroke group*

| | Error Correction Pattern, % | | | | | | | |
| | Movement Crossing into Spasticity Zone | | | | Movement in Active Control Range | | | |
Subject	*Pattern 1*	*Pattern 2*	*Pattern 3*	*Pattern 4*	*Pattern 1*	*Pattern 2*	*Pattern 3*	*Pattern 4*
1	0	0	0	100	0	40	13	47
2	0	0	13	87	9	9	18	64
3	13	7	20	60	7	40	13	40
4	0	0	53	47	27	47	33	13
5	0	13	47	40	0	47	20	33
6	0	27	53	20	27	47	20	7
7	13	13	60	14	60	27	7	6
8	13	7	33	47	33	20	20	27
9	7	40	47	6	20	60	20	0
10	6	6	53	33	33	27	40	0
11	20	33	47	0	47	33	13	7
12	20	27	47	6	47	40	6	7

Data are proportions of error correction *patterns 1–4* used by each of the participants with stroke for movements that crossed into the spasticity zone and in the active control range.

DISCUSSION

We studied the ability of individuals with chronic stroke to perform a one-trial learning paradigm involving elbow extension of their paretic arm when movements were restricted to articular zones in which spasticity was present or absent. Two articular ranges, the active control range and the spasticity zone, were demarcated by the angle of the stretch reflex threshold measured at rest. Healthy subjects could rapidly adapt movements to changed load conditions and correct errors by the first or second trial. However, error correction ability was impaired in subjects with stroke when elbow movements extended into the spasticity zone (i.e., beyond the TSRT) such that more trials were needed to correct errors and terminal positions were less consistent. In the same stroke subjects, when the movement range was limited to the active control range, error correction ability was significantly better such that errors were corrected within two trials and end-point positions were less variable in the remaining trials in the same load condition. This improvement occurred immediately, without training. Longer term learning was not evaluated.

The experiment was set up so that the task could be accomplished by specifying one of two sets of control variables (CVs; R and C commands) learned during practice. One set of CVs was used to move the arm to the target position without a load, and the second set moved the arm to the target with the load. When the load was suddenly changed, if the subject specified the same CVs as those used in the previous trial, an error would occur. For example, if they issued CVs for a nonloaded trial when the subsequent trial was loaded, an undershoot error would occur. To correct the error in the next like-loaded trial, the subject would have to issue the CVs for a loaded trial. The finding that participants with stroke corrected errors after fewer trials when movements were restricted to the active control range suggests that the ability to learn, store, recall, and use the appropriate CVs according to the load condition was preserved. In contrast, if stroke affected the ability to issue appropriate CVs, deficits in error correction would have occurred irrespective of where in task space the movements were executed. This suggests that subjects had no deficit in motor learning but that movements made into the spasticity zone were likely influenced by stretch reflex resistance complemented by possible changes in intrinsic viscoelastic properties of spastic muscles (Lieber et al. 2003; Sinkjaer and Magnussen 1994) affecting the ability of CVs to execute the desired movement patterns and effect corrections.

Motor Learning and Speed-Accuracy Trade-Off

Studies of motor learning in individuals with stroke have used a variety of tasks such as reaching (Park et al. 2016), serial voluntary isometric force production (Hardwick et al. 2017), continuous tracking, and serial reaction time (Boyd et al. 2007). End-point accuracy differs for movements made at different speeds and over different distances (speed-accuracy trade-off; Fitts 1954). Investigators have made conclusions about the capacity of healthy (Reis et al. 2009) and stroke subjects (Hardwick et al. 2017) to acquire new motor skills by designing tasks matched for task difficulty in terms of distance, time, and accuracy constraints. According to this approach, in our study, distance and speed were similar for movements made in either articular zone in an attempt to match task difficulty, despite that fact that the difficulty related to the motor impairment when movements were made in the spasticity zone would be greater than that in the active zone.

Effect of Voluntary Movement on Spasticity Threshold

It is likely that the TSRT angle measured at rest was modulated during active movement. Our results of the correlational analysis do not provide clues to this relationship, because the clinical measures do not take spatial spasticity zones into account in the determination of levels of muscle resistance to stretch (CSI), arm motor impairment (FMA-UL), or activity limitations (sWMFT). Although the exact relationship between spasticity and voluntary movement is unknown, Turpin et al. (2017) showed that TSRTs of elbow flexors in patients with stroke increased by ~20° compared with those measured at rest when the same flexor muscles were stretched by voluntary activation of the antagonist muscles (extensors), thus decreasing the flexor spasticity zone. On the other hand, the actual (dynamic) threshold angle decreases with increasing velocity of stretch (see *Eq. 2*). Because the elbow extension movements were made at moderate to high speeds in the

present study, the increase in the actual TSRTs of flexors was likely offset by the decrease in angle due to the increase in movement velocity.

Motor Learning and Spasticity

Our paradigm allowed us to identify the one-trial learning capacity in patients with different levels of UL impairment while controlling for the confounding influence of spasticity. Individuals with greater UL impairment had lower proportions of *patterns 1* and *2* for movements made in both zones. However, error correction ability improved immediately only when movements were restricted to the joint space unaffected by spasticity. The improvement in error correction ability was not due to training, because the session order was randomized and a 1-wk period between testing sessions provided adequate washout. The paradigm was also specifically designed to minimize any learning effect by randomizing the number of trials within each trial block, so that subjects could not anticipate when the load conditions would change, by randomizing the number of trials in each of the 16 blocks between 6 and 10 and ensuring that the same movement velocity was produced on each trial by providing feedback after every trial. There was no evidence that improvement in error correction ability occurred over the trial block or carried over to the second session. Indeed, within- and between-sessions, movements were made with similar velocities and the magnitudes of overshoot and undershoot errors.

The influence of spasticity on movement production and motor learning has been highly controversial. Views of spasticity range from it being considered integral to disordered motor function (Bobath 1978; Lance 1980) to being completely independent of it (O'Dwyer et al. 1996) as a "positive" manifestation of central nervous system damage (Burke 1988). Traditional rehabilitation approaches that focus on reducing spasticity as a prerequisite for improving motor function (Bobath 1978; Vaughan-Graham et al. 2015) have been criticized in light of evidence suggesting that suppression of spasticity has not led to improvements in function in adults with stroke (McLellan 1977). Findings that deficits in muscle activation are not primarily due to hyperactive reflexes in spastic antagonist muscles but to a decreased ability to contract agonist muscles (Sahrmann and Norton 1977) support this argument. However, previous studies have not used methodologies that allow interpretations of physiological results within a theoretical motor control framework. For example, O'Dwyer et al. (1996) found no correlations between resistance in elbow flexors to 10° quasi-sinusoidal stretching when the elbow was positioned at 90° and 170° extension, voluntary flexor isometric force at 90°, and slow and fast sinusoidal tracking errors. This conclusion is not surprising, because in the absence of identification of spasticity zones vs. active control ranges in individual patients, it is likely that some patients produced actions in active control ranges and that other patients' movements included spasticity zones, making clear correlations impossible.

In contrast, when controlling for the location of the TSRT angle, Levin et al. (2000) showed that elbow flexion and extension movements could be made with typical muscle activation patterns when movements were restricted to the individual's active control range. However, muscle activation patterns were abnormal (i.e., excessive coactivation) when movements were attempted in joint ranges that traversed the threshold angle for spasticity. In particular, each subject had a unique profile of active control and spasticity zones for flexors and extensors throughout the elbow joint range. Similar results were reported for the spasticity zones described for double-joint shoulder/elbow movements in patients with chronic stroke (Musampa et al. 2007).

Limitations and Possible Alternative Explanations

We presumed that the improvement in the precision and correction strategies in the active control range was associated with the ability to produce normal reciprocal muscle activation patterns. A limitation of the study is the lack of EMG data to support this assumption, although we have previously shown that movements made in active control ranges and spasticity zones are characterized by normal and disrupted muscle activation patterns, respectively (see Levin et al. 2000).

Only effects of movements made within the active control range in one session were assessed. Thus, whether improvements in error correction ability persist when elbow extension movements are repeated within the active control range (short-term learning) and whether task practice limited to the active control range results in better motor learning outcomes remain to be investigated. In addition, we used an error correction task involving only one joint. Whether short-term motor learning of an UL task involving double-joint movements (Foisy and Feldman 2006) is influenced by the presence of spasticity zones remains to be determined. Finally, generalization of results is limited by the small sample size.

One can consider the possibility of alternative explanations of results of the present study based on the conventional hypothesis that motor control relies on direct preprogramming of motion in terms of kinematic and kinetic variables describing the motor outcome. In particular, it is assumed that the nervous system preplans a trajectory of the effector in reaching the movement target; then, using a neuronal emulator ("internal model") of mechanical laws, it computes and specifies forces required to drive the effector along the desired trajectory.

Deficits in motor control then could be caused by problems in planning and/or execution of motion resulting from brain lesions. However, this approach does not provide an explanation of the decreased range of regulation of spatial stretch reflex thresholds and, as a consequence, the occurrence of spasticity zones. The hypothesis of direct preprogramming of motion also conflicts with the finding of threshold position control of muscle activation and reflexes in intact systems, initially demonstrated by Matthews (1959) and Asatryan and Feldman (1965; see their Fig. 1). This finding implies that kinematic and kinetic aspects of movements emerge, without preprogramming, in response to threshold shifts.

Concluding Comments

This study provides evidence that motor learning processes may be preserved in patients with stroke but masked by the presence of spasticity in specific joint ranges. Taken together, previous results and those of the present study suggest that movements made to study motor learning in patients with stroke should be done in consideration of the angular zone in

which the task is performed in order not to confound impairments in motor execution (weakness, spasticity, and abnormal motor synergies) with abnormalities of motor learning or performance. Accounting for the location of angular spasticity zones may also be relevant to the design of more effective interventions based on individualized motor impairment.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

A.G.F. and M.F.L. conceived and designed research; S.K.S., A.G.F., and M.F.L. performed experiments; S.K.S., A.G.F., and M.F.L. analyzed data; S.K.S., A.G.F., and M.F.L. interpreted results of experiments; S.K.S., A.G.F., and M.F.L. prepared figures; S.K.S., A.G.F., and M.F.L. drafted manuscript; S.K.S., A.G.F., and M.F.L. edited and revised manuscript; S.K.S., A.G.F., and M.F.L. approved final version of manuscript.

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