Chapter 14
Deficits in Spatial Threshold Control of Muscle Activation as a Window for Rehabilitation After Brain Injury

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14.1 Background

14.1.1 The Problem of Spasticity and Disordered Motor Control

Spasticity is associated with an upper motor neuron lesion affecting recovery of motor function (Kim and Park 2011; Sunnerhagen et al. 2013). It is estimated that spasticity affects over 12 million patients worldwide who have had a stroke, spinal cord injury, head trauma, multiple sclerosis, cerebral palsy (CP) or other disorder of the central nervous system (CNS; Burke 1988; www.aans.org). At the root of the motor deficit after stroke is hemiparesis characterized by a diminished capacity to recruit agonist muscles (Hammond et al. 1988; Gemperline et al. 1995; Chang et al. 2013), or the use of abnormal recruitment patterns (Bourbonnais et al. 1989; Dewald et al. 1995; Kamper and Rymer 2001; but see Gowland et al. 1992; Fellows et al. 1994a, 1994a) and weakness (Kamper et al. 2006; Chang et al. 2013). Agonist–antagonist coactivation may be responsible for a reduced ability to selectively activate arm and hand muscles (Lang and Schieber 2004). This disability can also be associated with abnormal timing of agonist and synergist muscle activation, and failure to deactivate antagonist muscles (Hoffman and Strick 1995).

Spasticity, weakness, and motor impairments have traditionally been considered as separate phenomena (Fig. 14.1). However, the idea that they are interrelated was alluded to in the definition suggested by Lance in 1980. This definition, now frequently cited in the literature, describes spasticity as "a *motor disorder* characterized by a velocity-dependent increase in stretch reflexes (muscle tone with exaggerated tendon jerks) as one component of the upper motor neuron syndrome." Lance's definition suggests that spasticity and motor deficits are related to problems in stretch

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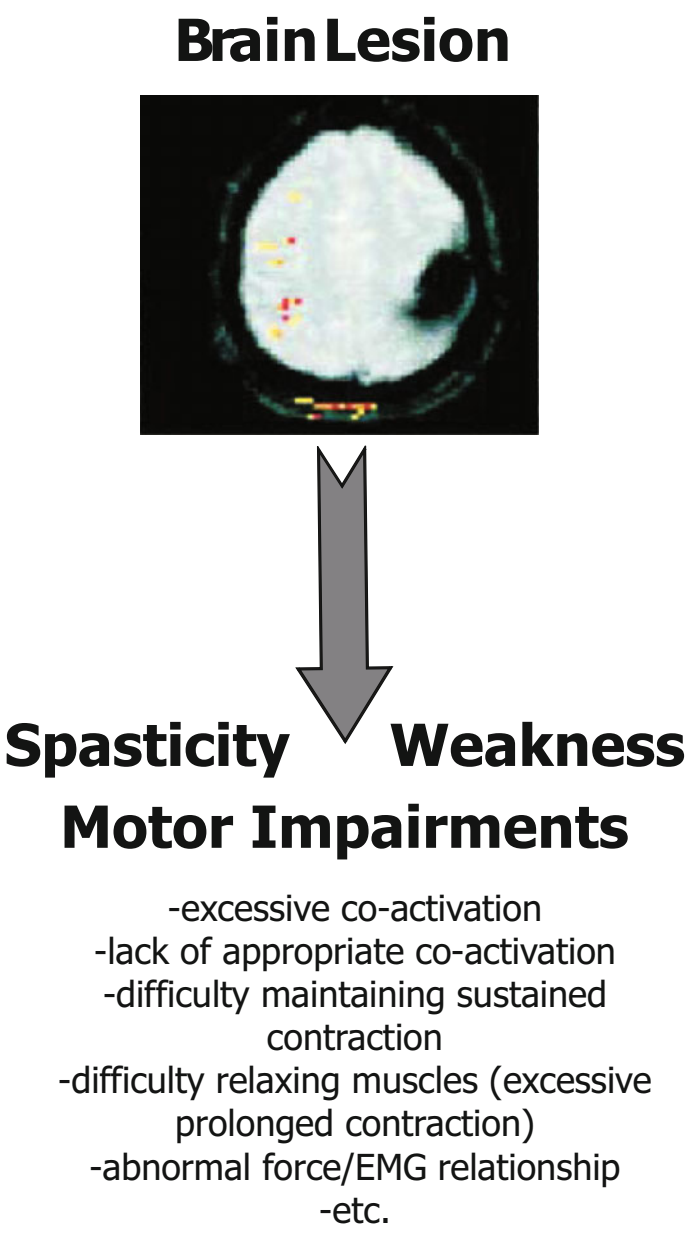

Fig. 14.1 Schematic diagram of the interrelationship between spasticity, weakness, and disordered motor function after a brain lesion

reflex excitability. However, the precise relationship between spasticity, hyperactive stretch reflexes, and the abnormalities in the production of voluntary movement remains unclear.

Studies attempting to determine the degree to which altered muscle tone and stretch reflex hyperexcitability may influence voluntary motor control have yielded equivocal results. Corcos et al. (1986) studied ballistic ankle dorsiflexion movements in subjects with spasticity of mixed etiology and showed that deficits in voluntary calf muscle activation were related to hyperactive stretch reflexes. However, other studies of upper limb hemiparesis reported that the disorder in voluntary motor control may not be related to spasticity or hyperactive stretch reflex activity (Sahrmann and Norton 1977; O'Dwyer et al. 1996; Burne et al. 2005; Chang et al. 2013). The controversy is associated with a lack of a coherent view of the relationship between spasticity and disordered voluntary muscle activation.

A major problem in the understanding of this relationship is the method used to quantify spasticity. Although the presence or absence of spasticity can be identified using current clinical scales (Malhotra et al. 2009), such clinical scales are insufficient to determine the relationship between spasticity, deficits of voluntary movements, and functional ability (for reviews, see Elovic et al. 2004; Wood et al. 2005; Calota and Levin 2009). There is general agreement that current scales of spasticity, even if objective, measure biomechanical variables (e.g., the resistance to imposed muscle stretch) that are *effects*, rather than *causes* of spasticity (Malhotra et al. 2009), and that various physiological measures (e.g., inhibition/facilitation mediated by cutaneous and muscular afferents) do not provide a comprehensive understanding of the nature of spasticity and disordered movement (Malhotra et al. 2008, but see Levin et al. 2000, Krakauer 2005; Musampa et al. 2007; Mullick et al. 2013).

Different components of spasticity have been identified using physiological measures (e.g., disorders of reflex pathways, Mazevet et al. 2003), biomechanical measures (e.g., deficits in interactive joint torque control, Dewald et al. 1995; Beer et al. 2000), or a combination of both (e.g., reflex and nonreflex components of ankle spasticity, Zhang et al. 2013); based on the assumption that these components are controlled separately. However, there is increasing evidence to suggest that not only spasticity and movement impairments but also muscle weakness and loss of dexterity (skilled muscle actions) are all consequences of the same underlying control deficit. It is proposed that this common control deficit is the specification and regulation of spatial thresholds (STs) of the stretch and other proprioceptive reflexes (Levin and Feldman 1994; Feldman and Levin 1995; Musampa et al. 2007; Feldman 2011).

14.1.2 ST Regulation in the Healthy Nervous System

The ST corresponds to the muscle length or joint angle (R) at which muscle activity begins. This expresses the muscle activation threshold in the spatial (angular) rather than the temporal (e.g., in terms of latencies) domain. The ST is the position of a body segment at which postural reflexes begin to act—such that postural reflexes are

"centered" at the ST (Levin and Feldman 1994; Feldman 2011). Associated with the ST is a torque/angle characteristic representing the dependency of muscle reflex force on muscle length for a given threshold. The angular range through which the ST and its associated torque/angle relationship can be regulated is shown as a horizontal band in Fig. 14.2a. In the healthy nervous system, ST can be regulated throughout and beyond the biomechanical range of the joint (shown as the physical limits of joint flexion and extension lying between θ_- and θ_+, which correspond to the minimal and maximal biomechanical muscle length, respectively). The limits of ST regulation are shown by R_- and R_+. By shifting ST, the brain resets ("re-addresses") posture-stabilizing mechanisms to a new limb or body position. When the ST is shifted entirely to the left as shown in Fig. 14.2a (the lower limit, R_-), the muscle is activated even at very short muscle lengths. When it is shifted beyond the biomechanical range to the upper limit (R_+), the muscle cannot be activated and is fully relaxed. By regulating ST and its associated torque/angle characteristic (diagonal line in Fig. 14.2a) within these limits, the CNS can produce any physiologically possible combination of muscle activity, torque, and position without the need to specify these variables directly (Levin et al. 2000; Feldman 2011). Resetting of ST is also associated with the conversion of movement-resisting to movement-producing forces, providing a solution to the fundamental posture-movement problem; originally formulated by von Holst (1954; see also Ilmane et al. 2013 and Feldman, this volume).

ST regulation is an important mechanism explaining motor control in the healthy nervous system (Feldman and Levin 1995; Feldman 2009, 2011). ST control of stretch reflexes (in cats and humans) and intentional movements (in humans) is well established (Matthews 1959; Asatryan and Feldman 1965; Feldman and Orlovsky 1972; Nichols and Steeves 1986; Capaday 1995; Raptis et al. 2010; Sangani et al. 2011; Ilmane et al. 2013). The ST of a given muscle is controlled by descending inputs directly or indirectly influencing the membrane potential or electrical threshold of α-motoneurons (pre and postsynaptically via interneurons or γ-motoneurons; Matthews 1959; Feldman and Orlovsky 1972; Nichols and Steeves 1986; Hultborn and Kiehn 1992; Capaday 1995; McClelland et al. 2001). Changes in ST can be mediated by cutaneous afferents or those responsible for reflex intermuscular interactions, including reciprocal inhibition of agonist–antagonist muscles (Matthews 1959; Feldman and Orlovsky 1972). Segmental mechanisms including presynaptic and reciprocal inhibition are themselves modulated by biomechanical factors leading to the recruitment of different subsets of motoneurons (Ter Haar Romeny et al. 1984; van Zuylen et al. 1988) as well as by task-related descending influences mediated by neuromodulators, such as serotonin and norepinephrine, which change motoneuronal intrinsic properties, thus causing depolarization, plateau potentials, and shifts in their electrical thresholds (Lundberg 1967; Hultborn et al. 1987; Hultborn and Kiehn 1992; Meunier and Pierrot-Deseilligny 1998; McPherson et al. 2008). These mechanisms combine to regulate STs in a multi-muscle system according to the configuration of the system and specific task demands (Nichols and Steeves 1986; Feldman and Levin 1995; McClelland et al. 2001).

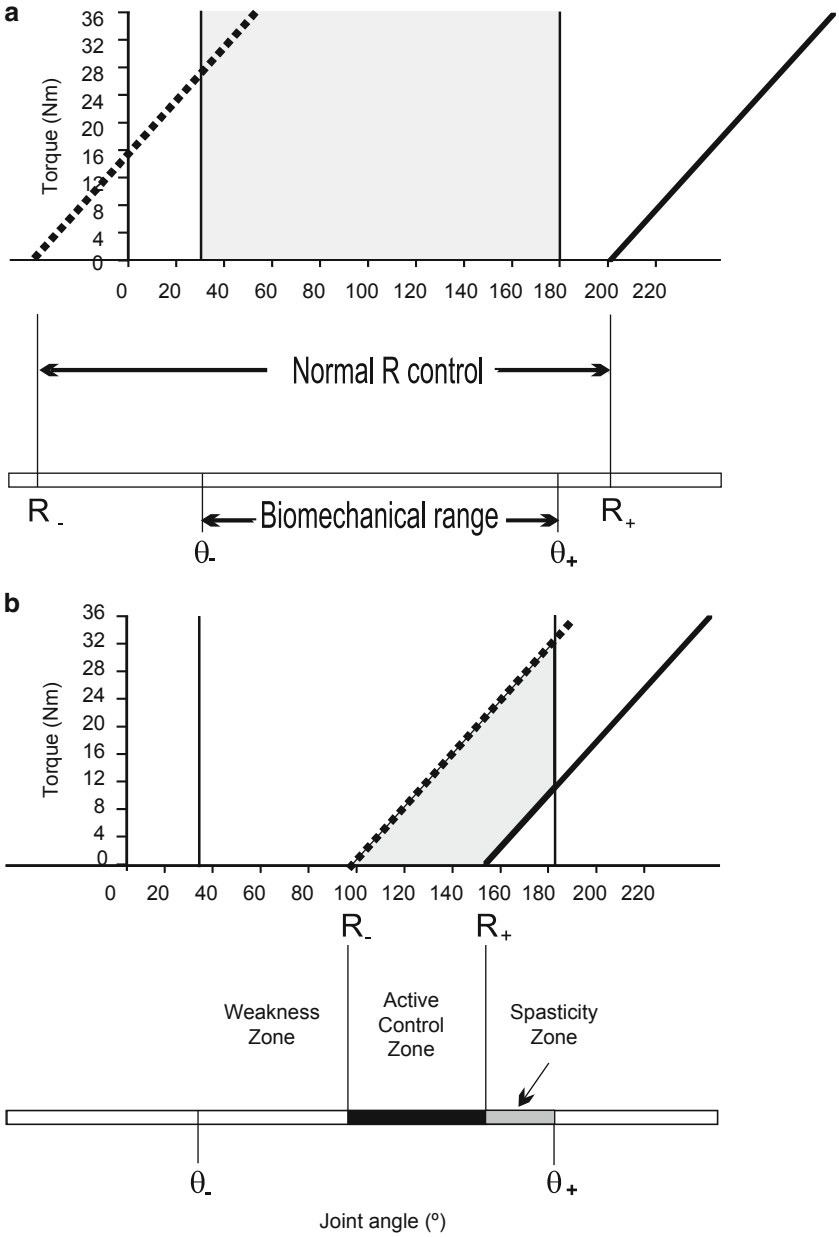

Fig. 14.2 Schematic diagram of central control of spatial thresholds (*STs*) of proprioceptive reflexes and motor deficits resulting from impairments of ST control. In this scheme, it is assumed that flexor muscles lengthen and extensor muscles shorten with increasing joint angle. **a** Normally, the range (R_-, R_+) of threshold regulation exceeds the biomechanical range of the joint (θ_-, θ_+), thus enabling the system to relax (when R_+ is shifted to the right beyond θ_+) or to activate muscles at any angle (when R_+ is shifted anywhere between θ_+ and R_-) to generate motion within the

14.1.3 ST Regulation After CNS Lesions

As reviewed above, in healthy subjects normal muscle activation is associated with an ability to regulate the ST in a range that exceeds the biomechanical joint range. Consequently, a decrease in the range of ST regulation following CNS injury or disease would result in motor control deficits (Fig. 14.2b and 14.2c). Deficits in descending and spinal mechanisms resulting from CNS injury, possibly together with changes in intrinsic motoneuronal properties such as in chloride reversal potentials and properties of serotonin receptors, in combination and isolation may contribute to limitations in ST regulation and the appearance of spasticity (Nielsen et al. 2007).

Empirical support for a common deficit in ST regulation underlying spasticity and motor control problems in single- and double-joint systems has been described in subjects with stroke (Lee et al. 1987; Powers et al. 1989; Levin and Dimov 1997; Levin et al. 2000; Mihaltchev et al. 2005; Musampa et al. 2007) and in children with hemiplegic CP (Jobin and Levin 2000). In particular, poststroke subjects with spasticity have deficits in the regulation of STs within the physiological joint range (Fig. 14.2b). Figure 14.2b shows what would occur if the lower and upper physiological limits of ST regulation (R_-, R_+) were reduced so that they lie within the biomechanical range of the joint. This would result in a narrowing of the range in which movement can be produced using "typical" reciprocal muscle innervation patterns. This range is called the "active control zone" since it is characterized by muscle activation patterns observed in people without neurological injury or disease. In the joint range between R_+ and the end of the joint excursion, muscles would be unable to relax at rest and the amount of muscle activation would be related to the velocity of stretch. This spatial joint range is called the "spasticity zone," which is not usually observed in people without neurological injury. Conversely, when the lower ST limit (R_-) is increased (i.e., shifted to the right of θ_-), the spatial zone between θ_- and R_- would be characterized by muscle weakness ("weakness zone") due to inadequate muscle activation (Levin et al. 2000). A combination of changes in the regulation of the upper and lower limits of R can result in different deficits occurring in different joint ranges (zones) accounting for abnormal muscle activation patterns observed when patients attempt voluntary movement (Levin et al. 2000; Lang and Schieber 2004).

Patterns of spasticity, weakness, and active control zones in flexor and extensor muscles around the elbow joint from 12 subjects with chronic stroke (> 0.8–7.1 years post stroke) and different levels of arm paresis ranging from mild to severe

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biomechanical range. **b** Following a brain lesion, the upper limit (R_+) of threshold regulation can abnormally fall within the biomechanical range, resulting in an inability to relax flexor muscles at joint angles exceeding the position of R_+. Clinically, this deficit in poststroke subjects is identified as spasticity. **c** Following a brain lesion, the lower limit (R_-) can also fall within the biomechanical range, resulting in an inability to activate flexor muscles if the joint angle is less than the R_- (weakness zone)

(Fugl-Meyer Arm Assessment Score 15–65 out of 66; Fugl-Meyer et al. 1975) are shown in Fig. 14.3 (Levin et al. 2000). Zones were determined by stretching passive muscles at different velocities to identify STs of elbow flexors R_{f+} and elbow extensors R_{e+} and then by identifying patterns of agonist and antagonist muscle coactivation during slow full-range voluntary elbow extension and flexion. Each subject had a unique pattern of active control (blue), spasticity (black/yellow), and coactivation (black) zones within the biomechanical joint range. The range of movement in which active movement was possible is indicated from M_- to M_+. The range of M could extend beyond the active control zone, but movements made in this angular zone would be characterized by agonist/antagonist muscle coactivation. The figure shows that different patients had different angular ranges in which active movement was accomplished using reciprocal muscle activation (blue zones) and further movement was possible only with muscle coactivation (black zones). In all cases, the borders between reciprocal and coactivation zones coincided with STs identified in flexor (R_{f+}) and extensor (R_{e+}) muscles, shown for the first four subjects in Fig. 14.3.

14.1.4 Methodology Used to Identify the Range of Regulation of STs

In a series of studies, we sought to determine the upper limit of ST regulation for flexor and extensor muscles of the elbow in patients with spasticity. However, by definition, the tonic ST is the value of the threshold when the system is at rest (e.g., at zero velocity of stretch) and thus cannot be measured directly. Therefore, we developed a method to determine the velocity-dependent or *dynamic* stretch reflex thresholds (DSRTs), and then to extrapolate the value of the tonic stretch reflex threshold (TSRT) from these values. Each DSRT is the joint angle and velocity corresponding to the onset of the EMG in the stretched muscles at each stretch velocity. A regression line is plotted through the set of DSRTs and the intercept of this regression line with zero velocity is the TSRT (Fig. 14.4b and 14.4c; Mullick et al. 2013). The TSRT thus corresponds to the ST for this muscle. In nondisabled individuals, DSRTs can usually only be evoked in noncontracting muscles if the stretch is performed at very high velocities ($> 300°$/s; Thilmann et al. 1991; Levin et al. 2000). However, in adults with stroke and children with CP, muscle stretches at speeds as low as 8°/s, applied to the elbow joint, can generate DSRTs.

Previous studies in patients with spasticity due to stroke or CP have shown that (1) the TSRT lies within the physiological range of motion of the elbow; (2) the TSRT value may or may not be correlated with the degree of resistance produced by the same muscles when they are stretched; (3) the TSRT may be a better measure of spasticity than muscle resistance measures (Jobin and Levin 2000; Levin et al. 2000).

Jobin and Levin (2000) measured TSRTs in elbow flexor muscles of children with CP and tested the reliability of this value as an estimate of clinical spasticity. Fourteen children with CP and eight typically developing children participated in the

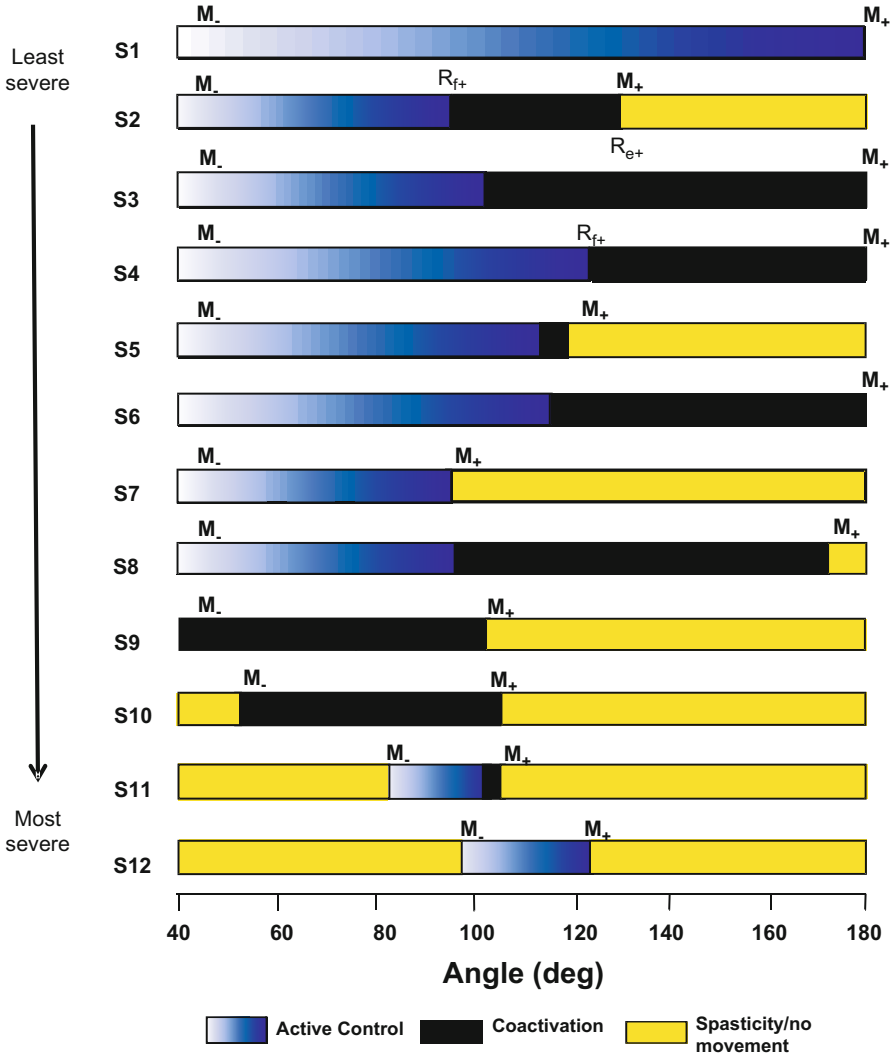

Fig. 14.3 The profiles of active control (*blue*), spasticity + coactivation (*black* + *yellow*) and spasticity + weakness (*yellow*) zones in 12 patients with chronic stroke (> 0.8–7.1 years post stroke) and different levels of arm paresis ranging from mild to severe (Fugl-Meyer Arm Assessment Score 15–65 out of 66). Each subject had a unique pattern of disability. Subject 1 (*S1*) had full range of movement with typical muscle innervation patterns. All other subjects had limited active control zones, while *S9* and *S10* had no active control zones throughout the entire joint range. Movements were still possible in these two subjects, but these were accomplished with muscle coactivation (*black zones*). (Adapted with permission from Levin et al. 2000)

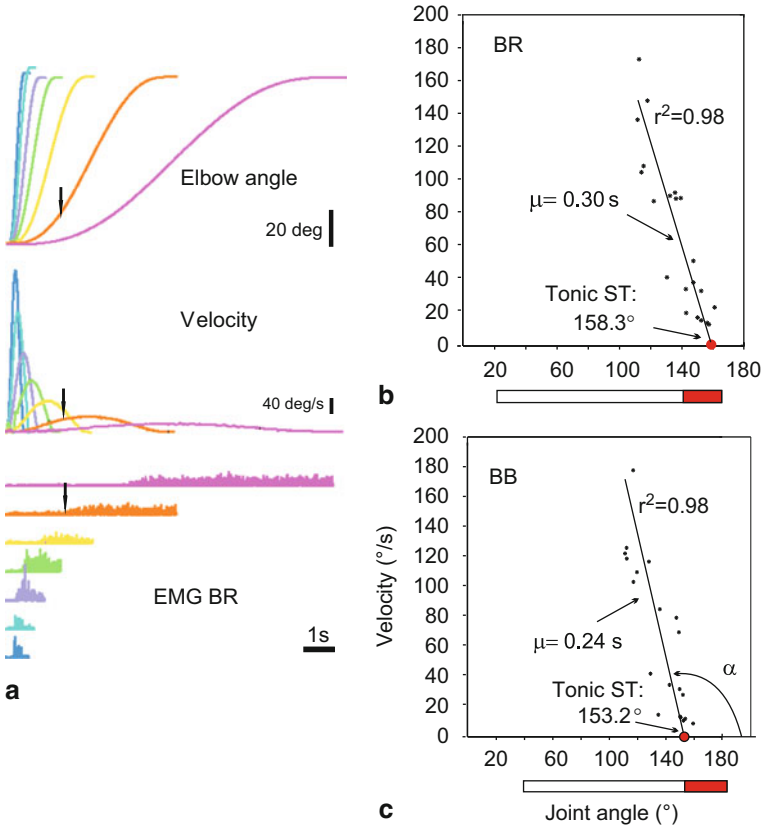

Fig. 14.4 Spasticity in a poststroke subject is associated with the presence of velocity-dependent threshold angles beyond which the muscle cannot be relaxed. **a** Single traces of elbow displacements (*top*) through a 90° range of extension at seven different velocities (*middle*) ranging from 8°/s (purple) to 160°/s (*dark blue*). Stretches were applied by a torque motor. EMG activity from Biceps Brachii (*BB*) evoked by each stretch is shown in bottom traces. Dynamic stretch reflex thresholds (*DSRTs*) are defined as the angle and velocity values at which EMG activation in the stretched muscle begins (arrows). **b, c** Example of dependencies of reflex spatial thresholds (*STs*) on velocity for two elbow flexors (*BR*, Brachioradialis; *BB*) in stroke subjects. Each point on the graphs represents the *DSRTs* for different velocities of stretch. The intercept and slope of the regression line through the *DSRTs* are the tonic stretch reflex threshold (*TSRT*) and dynamic sensitivity (μ) of the system, respectively. *Horizontal strips* below each graph show the flexor spasticity zone (*red*) and active control zone (*white*). (Adapted with permission from Mullick et al. 2013)

trial. DSRTs were evaluated by performing eight sets of stretches at seven, randomly selected velocities between 8 and 160°/s using a torque motor. For the elbow flexors, TSRT angles occurred later (closer to full elbow extension) in children with less-severe spasticity and TSRTs were only weakly correlated with clinically measured resistance to stretch of the passive muscle ($r = 0.39$). The test–retest reliability of the computed TSRTs was estimated as good (ICC = 0.73, $p < 0.001$).

14.1.5 TSRTs in Multi-Joint Muscles and Double-Joint Systems

Although the range of regulation of TSRTs can predict the location of ranges of spasticity and normal and abnormal movement patterns in a single joint, everyday gestures involve movements of more than one joint. Indeed, the TSRT value in a given muscle can be modulated by reflex interactions between muscles crossing adjacent joints (Matthews 1993; Bonasera and Nichols 1994; Ginanneschi et al. 2006; Musampa et al. 2007; Roberts et al. 2008). Interjoint interactions include autogenic facilitation; recurrent inhibition; presynaptic inhibition; reciprocal inhibition and facilitation; and heteronymous inhibition and facilitation (Cavallari and Katz 1989; Gracies et al. 1991; Katz et al. 1991; Cavallari et al. 1992; Créange et al. 1992). Interjoint reflex interactions have been identified in arm muscles in healthy subjects (Marchand-Pauvert et al. 2000; Archambault et al. 2005) and may lead to changes in net motoneuronal excitability of the stretched muscle from the convergence of information from afferents of heteronymous muscles via spinal, propriospinal, and/or transcortical pathways. In the intact nervous system, intermuscular reflex interactions have been reported for double-joint elbow muscles (Biceps Brachii and Triceps Brachii) when muscles were preactivated and placed in three different positions (McClelland et al. 2001).

In patients with poststroke spastic hemiparesis, Musampa et al. (2007) evaluated the influence of intermuscular interactions on TSRTs in uni and biarticular muscles of the double-joint elbow–shoulder system using a similar methodology as described above. They characterized the relationship between muscle length, TSRTs, and voluntary muscle activation patterns in elbow flexors and extensors and identified spasticity zones in the space of elbow–shoulder configurations from three initial positions of the shoulder (Fig. 14.5a). They also investigated how the presence of spasticity zones influenced voluntary arm movement. Similar to the previous identification of spasticity zones in a single joint (elbow; Levin et al. 2000), the limitations in the regulation of TSRTs in the double-joint arm system were shown to result in a subdivision of all-possible arm configurations into spatial spasticity zones and no-spasticity zones (Fig. 14.5b). All patients had ranges of shoulder–elbow arm configurations in which muscles could not be relaxed (spatial spasticity zones), which in some patients covered a substantial part of the biomechanically defined range of all possible arm configurations. It was also observed that these zones were practically the same for synergistic muscles and could overlap for antagonist muscle groups, such that there could be a zone in which both flexor and extensor muscles would show spasticity.

When patients attempted to voluntarily extend or flex their arm into the spasticity zones, significant abnormal agonist–antagonist muscle coactivation patterns occurred. Of note is that the angular value of the TSRTs evoked by stretching of passive muscles and the borders of the spasticity zone identified by active movement were highly correlated ($r = 0.64$–0.86), indicating a relationship between spasticity and disordered movement confined to specific areas in joint space. Thus, during active elbow extension, when the elbow reached the specific joint angle associated with the elbow flexor TSRT, elbow flexors became active and coactivation occurred. This relationship was less marked in the extensors during active flexion.

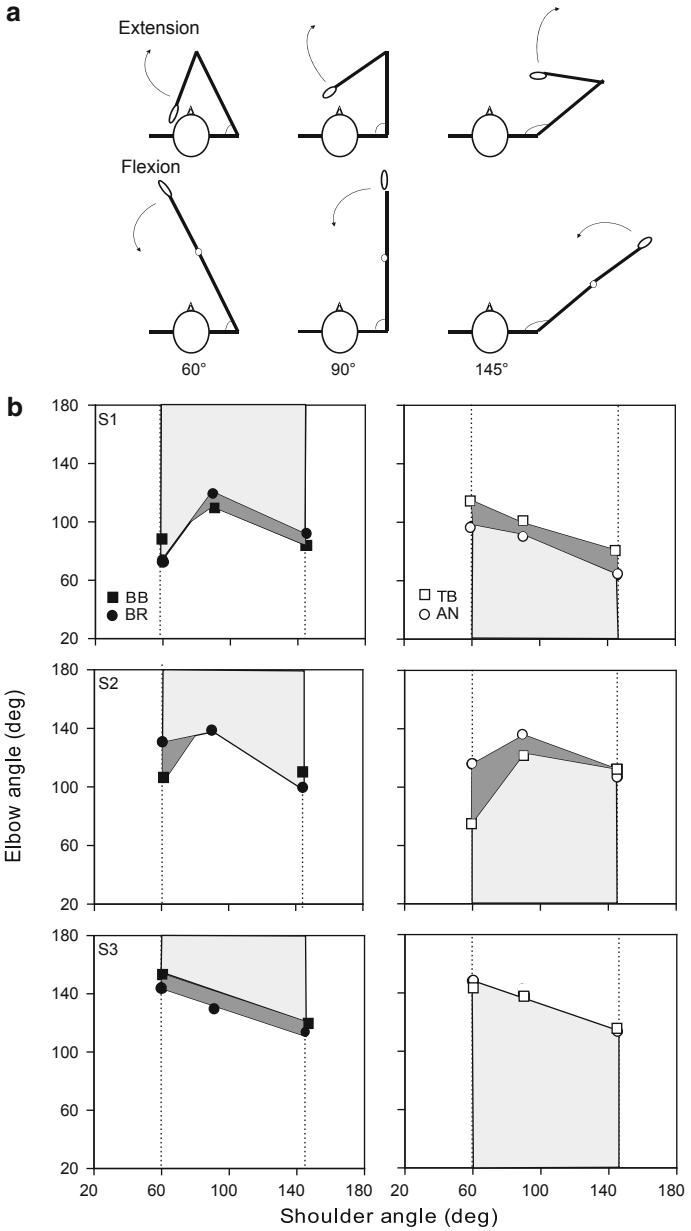

Fig. 14.5 **a** Methodology used to stretch single- and double-joint elbow muscles in order to identify the influence of initial muscle length (changes in initial shoulder position) on evoked responses. Initial position of the shoulder was varied from 60° to 145° before stretching elbow flexors (*top row*) and elbow extensors (*bottom row*). **b** Spatial thresholds (*STs*) evoked in four elbow muscles (Biceps Brachii, *BB*, *black squares*; Brachioradialis, *BR*, *black circles*; Triceps Brachii, *TB*, *white squares*; Anconeus, *AN*, *white circles*). Spatial spasticity zones are shown separately for the flexor muscles (*left panels*) and extensor muscles (*right panels*). Data from three patients with stroke-related upper

In summary, our studies have shown that limitations in the ranges of regulation of the TSRT correspond to the appearance of abnormal muscle activation patterns, such as excessive coactivation, when patients attempt to make voluntary movements (Levin et al. 2000; Musampa et al. 2007). In addition, ranges in which typical patterns of muscle activation can occur, such as reciprocal activation, have also been described using the TSRT approach. The use of this fundamental concept of threshold control based on equilibrium-point theory has led to a new and more in-depth understanding of the mechanisms underlying motor control deficits in patients with CNS lesions. Considering these findings, it may be inappropriate to view spasticity independently from the motor control deficit. Indeed, the concept of threshold control allows us to account for the spatial structure of deficits in the regulation of muscle activation and reflexes by indicating where in the biomechanical range, descending control of movement is mostly affected. Thus, it describes the control deficit in a functional context. This spatial structure of motor deficits is usually not taken into account in clinical evaluations and research on spasticity and disordered motor control, which may explain the heretofore elusive explanation of the relationship between them. Accounting for the spatial structure of motor deficits may benefit both researchers and clinicians by advancing the understanding of the mechanisms underlying unimpaired and impaired motor control. Based on this understanding, clinicians may more accurately measure these phenomena and establish more effective medical and physiotherapeutic interventions for their management.

14.2 Corticospinal Origin of Spasticity and Disordered Motor Control

In the healthy nervous system, cortical descending fibers mainly cross at the pyramidal decussation and project to muscles on the opposite side of the body (Kuypers 1985). Uncrossed fibers from the ipsilateral corticospinal tract mainly innervate axial and proximal muscles (Chen et al. 1997; Harris-Love et al. 2007). Previous studies using transcranial magnetic stimulation (TMS) in healthy subjects have shown that the corticospinal system exerts control over STs (Raptis et al. 2010; Sangani et al. 2011; Ilmane et al. 2013). Other crossed and uncrossed descending systems, such as the reticulospinal, rubrospinal, and tectospinal tracts, have also been shown to regulate STs in animal models (e.g., Feldman and Orlovsky 1972; Nichols and Steeves 1986). Of particular interest is that the crossed corticospinal tract is involved not only in the production of intentional movements but also in muscle relaxation in the entire biomechanical joint range (Raptis et al. 2010) and in anticipatory preparation of muscles to perturbations (Petersen et al. 2009; Sangani et al. 2011).

limb hemiparesis are shown (*S1*, *S2*, *S3*). The *STs* are points on the border in shoulder–elbow joint space beyond which muscles are activated abnormally at rest. *Dark shaded areas* indicate joint space where flexors or extensors are activated. *Light shaded areas* indicated joint spaces in which only one of the two flexor or extensor muscles is active. (Adapted with permission from Musampa et al. 2007)

Stroke-related damage to cortical and subcortical systems results in an imbalance of excitatory and inhibitory activity at different levels of the CNS (Ward 2011). The presence of a tonic stretch reflex response within the biomechanical range (ST) at rest in spastic muscles implies that the resting membrane potential of α-motoneurons innervating the muscle is higher than normal (> -70mV; i.e., closer to threshold). This is supported by findings that TSRT excitation and inhibition are altered in motoneuronal pools due to interruption of the inhibitory cortico-reticulospinal pathway (Lance 1980; Powers et al. 1989) and due to changes in other descending pathways from the brainstem (e.g., Fedirchuk and Dai 2004).

With respect to voluntary movement, deficits in muscle activation result from damage to the same descending pathways mentioned above. Indeed, transmission in cortical (Shimizu et al. 2002; Bütefisch et al. 2003) and spinal (Zehr et al. 2012) neuronal circuits is impaired after stroke. The uncrossed corticospinal, rubrospinal, and reticulospinal tracts all could provide alternate routes for motor cortical output to reach the contralateral spinal cord and may result in the appearance of motor compensations (Lawrence and Kuypers 1968a, 1968a; Woolsey et al. 1972; Kuypers 1985; Fisher 1992; Fries et al. 1993; Cao et al. 1998; Belhaj-Saif and Cheney 2000). The motor cortex also sends uncrossed corticospinal projections to the spinal cord (Colebatch and Gandevia 1989; Kuypers and Brinkman 1970; Nirkko et al. 2001). Alterations in rubrospinal and reticulospinal pathways influence predominantly the proximal musculature (Nathan and Smith 1982; Nathan et al. 1996). In addition to spinal and muscle mechanisms, damage to the corticospinal system leads to an imbalance in afferent influences ascending to different brain areas, including sensory and motor cortices in ipsi and contralateral hemispheres (Lindberg et al. 2009). After stroke, greater ipsilateral corticospinal tract involvement is associated with greater compensations and poorer recovery (Perez and Cohen 2009) and an imbalance between projections from both hemispheres due to altered interhemispheric inhibition (Misawa et al. 2008).

14.2.1 Functional Consequences of Limitation in Regulation of STs

Motor deficits and abnormal movement synergies after stroke are traditionally thought to be expressions of excessive reflex activity because of the loss of descending modulatory control (Burke 1988). However, these physiological mechanisms by themselves do not explain how movement deficits arise. The concept of ST regulation allows us to describe how excessive reflex activity may lead to the appearance of abnormal movement synergies and motor compensations. In other words, it provides a means to describe how the deficits can be expressed in a spatial frame of reference, or within the context of movements made by the body. Figure 14.6 is a schematic diagram of a model of disordered motor control giving rise to abnormal synergy patterns. Each joint range, shown by horizontal bands, is characterized by spatial ranges in which active control is possible and where spasticity is present in either

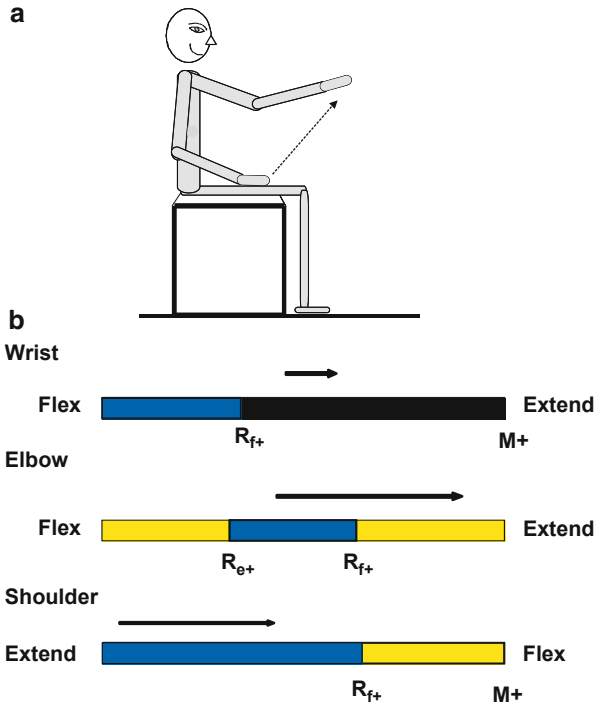

Fig. 14.6 Hypothetical model of how disordered control of spatial thresholds (*STs*) may give rise to abnormal muscle synergies. When a patient attempts to make a reaching movement **a**, the ability to activate muscles depends on the location of the *ST* within the joint range of each degree of freedom involved in the movement **b**. *STs* are indicated in each joint range as R_{f+} for flexor muscles and R_{e+} for extensor muscles and mark the angles at which spasticity begins. Ranges of movement required at each joint are indicated by horizontal arrows. For the movement shown in **a**, shoulder flexor activation is normal throughout the movement (first horizontal band—*black* range), while elbow extensors are unopposed initially (second horizontal band, *blue* zone) but coactivation of flexors occurs when R_{f+} is reached (second horizontal band—right *yellow* zone) and wrist muscles are coactivated throughout the movement (third horizontal band, *blue* zone). Abnormal interjoint couplings and restricted ranges of regulation of *STs* may explain limitations in the kinematic redundancy of the affected limbs

agonist or antagonist muscles. Active control zones are limited in each of the joints so that when movements are attempted that require one or several joints to move into spasticity zones, abnormal couplings between joints or abnormal synergies may arise. These abnormal interjoint couplings and restricted ranges of regulation of STs may explain limitations in the kinematic redundancy of the affected limbs. An example is shown in Fig. 14.5 by overlapping spasticity zones in elbow–shoulder joint space (Musampa et al. 2007). Thus, individuals who have deficits in multiple ST regulation have a limited number of possible joint combinations with which to produce different actions. If one extends this concept to more than two joints in the seven degree of freedom upper limb, then one would have multiple areas within the

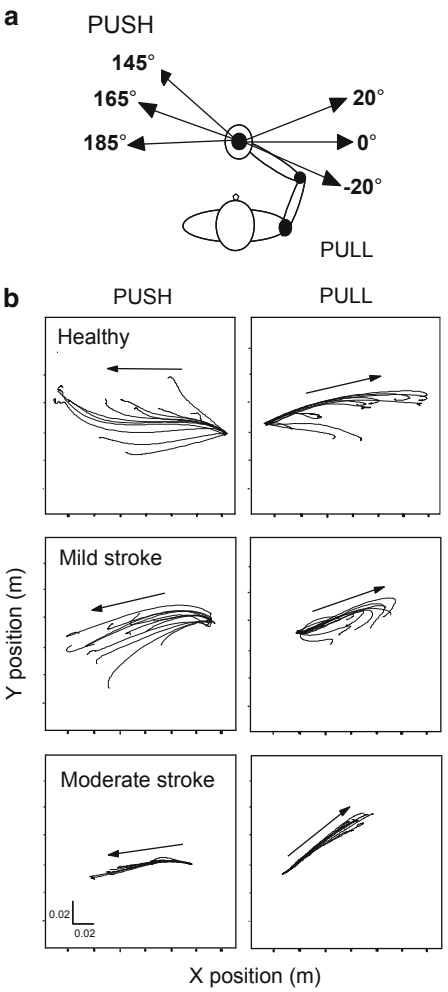

Fig. 14.7 Example of limited redundancy in upper limb movements in patients with stroke. **a** Subjects isometrically pushed or pulled a handle against a load in three different directions. The initial load of 30 % MVC was fully or partially removed in six steps for each load direction. **b** Hand trajectories after unloading for one healthy subject (*top panels*) and for two patients with stroke (*middle, lower panels*). Spatial dispersions of hand trajectories were smaller and more restricted in stroke compared to healthy subjects. (Adapted with permission from Mihaltchev et al. 2005)

3D arm workspace in which there would be restrictions in the number of available combinations of joint movements.

Another example of limited redundancy in stroke is shown for an upper limb pushing/pulling task. The subject was asked to resist a load while either pushing (Fig. 14.7, left panels) or pulling (Fig. 14.7, right panels) against it in three different directions (see Mihaltchev et al. 2005 for full details). The initial load of 30 % of their maximal voluntary contraction (MVC) was fully or partially removed in six steps for each load direction. Figure 14.7 also shows the hand trajectories after unloading for one healthy subject (Fig. 14.7b, top panels) and for two patients with stroke (Fig. 14.7b, middle and bottom panels). Compared to the healthy subject, the spatial dispersions of the hand trajectories were smaller and more restricted in terms of their variability in most participants with stroke. In this example and others (Reisman and

Scholz 2003; van Kordelaar et al. 2012), arm movements made by subjects with stroke are characterized by limitations in the variability of joint rotations, leading to stereotypical endpoint trajectories, as seen here, which are more marked in people with more severe sensorimotor impairment.

The presence of stereotypical movement patterns is related to the severity of the hemiparesis. Brunnstrom (1970) described five stages of recovery from stroke in which severe pathology is characterized by the presence of hyperactive segmental reflexes, spasticity and, when present, movement of the limbs only within defined flexor and/or extensor synergistic patterns, called "abnormal synergy patterns." In the upper limb, the abnormal flexor synergy consists of shoulder retraction, upper arm flexion, abduction and external rotation coupled with elbow flexion, forearm supination and finger flexion. The extensor synergy includes shoulder extension, adduction, and internal rotation coupled with elbow extension, forearm pronation and finger extension. Patients at various levels of recovery demonstrate all or some of the elements of abnormal synergies when they attempt to make voluntary movements. Movements are often also characterized by compensatory movement patterns such as excessive shoulder elevation and/or shoulder protraction when attempting to flex the upper arm (Merdler et al. 2013; Niessen et al., 2008) and excessive trunk displacement when attempting to move the hand away from the body (Cirstea and Levin 2000; Levin et al. 2002). Thus, there is evidence for both limited redundancy in the joints affected by reduced ST regulation leading to a restricted range of movement with limited variability and a preserved redundancy in joints not affected by such limitations leading to the appearance of motor compensations.

Indeed, recovery of upper limb movement is often evidenced by the ability of system to express more appropriate redundancy: The appearance of fractionated movements and movements out of pathological synergies due to a decrease in synergistic coupling between joints and/or by the use of fewer compensatory motor strategies (Lang and Schieber 2004; Michaelsen et al. 2006).

14.3 Summary

Despite detailed knowledge of the anatomo-physiological changes in descending, ascending, and spinal pathways after stroke, the relationship between spasticity and disordered motor control remains elusive. Part of the problem is that these have been considered to be separate phenomena. However, the role of the corticospinal tract in regulating STs provides a mechanism by which these motor impairments can be considered together. An important consideration is that motor deficits are present when subjects with stroke move or intend to move paretic limbs while most physiological examinations of spasticity are conducted at rest. In addition, previous studies have not considered the spatial nature of the impairments when measuring corticospinal output.

The extent to which the corticospinal tract is involved in ST regulation merits further investigation. By analyzing motor deficits within the experimentally based

context of ST control, it is possible to go beyond the usual characterization of motor impairments after CNS injury as separate phenomena. Consideration of the motor impairment in spasticity as being derived from a common deficit in the descending regulation of STs at which neuromuscular elements, including reflexes, begin to act, provides a way to understand the relationships between them and possibly provide an explanation for the reduced kinematic redundancy in the motor system typically seen in patients after CNS lesions. Identification of spasticity zones can also be used to monitor improvements in patient status due to pharmacological, medical, and physical treatment interventions.

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