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Upper Limb Motor Impairment Post Stroke

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Synopsis

Understanding upper limb impairment after stroke is essential to planning therapeutic efforts to restore function. However determining which upper limb impairment to treat and how is complex for two reasons: 1) the impairments are not static, i.e. as motor recovery proceeds, the type and nature of the impairments may change; therefore the treatment needs to evolve to target the impairment contributing to dysfunction at a given point in time. 2) multiple impairments may be present simultaneously, i.e., a patient may present with weakness of the arm and hand immediately after a stroke, which may not have resolved when spasticity sets in a few weeks or months later; hence there may be a layering of impairments over time making it difficult to decide what to treat first. The most useful way to understand how impairments contribute to upper limb dysfunction may be to examine them from the perspective of their functional consequences. There are three main functional consequences of impairments on upper limb function are: (1) learned nonuse, (2) learned bad-use, and (3) forgetting as determined by behavioral analysis of tasks. The impairments that contribute to each of these functional limitations are described.

Keywords

Stroke; Arm; Weakness; Hemiparesis; Motor Control

The nature of upper limb motor impairment

According to the International Classification of Functioning, Disability and Health model (ICF) (Geyh, Cieza et al. 2004), impairments may be described as (1) impairments of body function such as a significant deviation or loss in neuromusculoskeletal and movement related function related to joint mobility, muscle power, muscle tone and/or involuntary movements, or (2) impairment of body structures such as a significant deviation in structure of the nervous system or structures related to movement, for example the arm and/or hand. A stroke may lead to both types of impairments. Upper limb impairments after stroke are the cause of functional limitations with regard to use of the affected upper limb after stroke, so a clear understanding of the underlying impairments is necessary to provide appropriate

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treatment. However understanding upper limb impairments in any given patient is complex for two reasons: 1) the impairments are not static, i.e. as motor recovery proceeds, the type and nature of the impairments may change; therefore the treatment needs to evolve to target the impairment contributing to dysfunction at a given point in time. 2) multiple impairments may be present simultaneously, i.e., a patient may present with weakness of the arm and hand immediately after a stroke, which may not have resolved when spasticity sets in a few weeks or months later; hence there may be a layering of impairments over time making it difficult to decide what to treat first. It is useful to review the progression of motor recovery as described by Twitchell (Twitchell 1951) and Brunnstrom (Brunnstom 1956) to understand how impairments may be layered over time (Figure 1).

Understanding motor impairment from a functional perspective

The most useful way to understand how impairments contribute to upper limb dysfunction may be to examine them from the perspective of their functional consequences. There are three main functional consequences of stroke on the upper limb: (1) learned nonuse, (2) learned bad-use, and (3) forgetting as determined by behavioral analysis of a task such as reaching for a food pellet and bringing it to the mouth in animal models of stroke (Whishaw, Alaverdashvili et al. 2008). These are equally valid for human behavior. Each of the functional consequences and the underlying impairments are elaborated below.

Learned nonuse

Initially after a stroke, individuals may not use their affected upper limb suggesting learned nonuse. Nonuse can result from several impairments described below. Initially nonuse may occur due to weakness/ paralysis or sensory loss. However as time progresses, nonuse may become habitual and the limb may not be incorporated into functional activities even though the individual can move it. Now it becomes a learned behavior and is referred to as “learned nonuse”.

Weakness or paralysis is the predominant impairment that contributes to dysfunction after stroke (Canning, Ada et al. 2004; Wagner, Lang et al. 2007). It is a direct consequence of the lack of signal transmission from the motor cortex, which generates the movement impulse, to the spinal cord which executes the movement via signals to muscles. This results in delayed initiation and termination of muscle contraction (Chae, Yang et al. 2002), and slowness in developing forces (Canning, Ada et al. 1999), manifested as an inability to move or move quickly with negative functional consequences. Abnormally increased EMG-force slopes are seen on the affected side compared to the contralateral side as well as compared to neurologically intact subjects, suggesting that greater EMG activity is necessary to generate a given force in patients with stroke (Suresh, Zhou et al. 2008). This is thought to result from a combination of abnormal firing rate patterns and changes in motor unit control. Weakness may affect all muscle groups of the upper limb, or may be selective, affecting some muscle groups more than others. Large inter-subject differences exist in the pattern of muscle weakness across muscle groups, but research has shown that no consistent proximal-to-distal gradient or greater extensor relative to flexor weakness exists (Mercier and Bourbonnais 2004; Tyson, Chillala et al. 2006). While the absolute strength in any particular muscle

group has not shown to predict function, the rate of change of force development in wrist extensor and handgrip strength (Renner, Bungert-Kahl et al. 2009) are good predictors of upper limb function. Isolated motor deficits in a single arm can occur after a stroke without other cranial or sensory dysfunction; these are rare and easily misdiagnosed requiring a high index of suspicion and assessment of risk factors for stroke (Castaldo, Rodgers et al. 2003; Hiraga 2011). In cases where trauma accompanies the vascular lesion, the pattern of weakness may need to be examined carefully to rule out spinal cord injury or peripheral nerve injury. Weakness leads to immobility, which can initiate a cascade of problems that can further contribute to motor impairment (Figure 2), as described below.

Sensory loss across tactile, proprioceptive and/or higher-order sensory modalities such as deficits in two-point discrimination, stereognosis and graphesthesia are common after stroke, and may be associated with the degree of weakness and the degree of stroke severity, as well as mobility, independence in activities of daily living, and recovery (Tyson, Hanley et al. 2008). Sensory impairments without motor weakness may also occur from specific lesions in the parietal cortex (Bassetti, Bogousslavsky et al. 1993). In studies that compared the ability of hemiparetic and healthy subjects to produce symmetrical forces with both upper limbs, it was found that joint maximum voluntary forces and proprioceptive impairments in the affected limb predicted the errors in force matching (Mercier, Bertrand et al. 2004). These results suggest that sensory impairments may lead to inaccurate motor output even though motor capacities were adequate to perform the task. In fact the term learned nonuse was coined from observations in deafferented monkeys who could move but did not do so voluntarily (Taub, Heitmann et al. 1977). Chronic loss of sensation may contribute to motor impairment due to inaccurate internal representations of the task and/or inability to control the motor output appropriately due to lack of feedback about the consequences of the motor action.

Weakness leads to *immobility*, which can be considered a functional impairment according to the ICF classification (Geyh, Cieza et al. 2004). Immobility in turn can begin a vicious circle of problems including peripheral soft-tissue changes that reduce tissue compliance, potentiation of reflex mechanisms and spasticity, eventually leading to muscle fibrosis and contributing to abnormal limb posturing, pain and decreased function (Figure 2) (Stecco, Stecco et al. 2014). Spasticity is now thought to arise as a consequence of contractures rather than being a cause of contractures (Ward 2012). Hence early interventions to reduce immobility and preserve range of motion either passively or actively despite paresis, and prevent contractures may be critical to prevent spasticity and its ensuing complications. Passive tissue restraint and agonist weakness, rather than antagonist restraint, have been shown to be the most common contributors to decreased active range of motion (Reinkensmeyer, Schmit et al. 1999). Immobility also leads to changes in bone mineral density with increased risk of developing osteoporosis on the paralyzed side and particularly in the upper limbs (Hamdy, Krishnaswamy et al. 1993; Hamdy, Moore et al. 1995). In fact, fractures are common on the paretic side after stroke (Ramnemark, Nyberg et al. 1998). Practitioners need to pay more attention to changes in bone mineral density post stroke and take active measures to prevent problems arising from immobility.

Motor and sensory impairments and immobility are associated with an increased risk for ***stroke-related pain*** (Lundstrom, Terent et al. 2008). Stiffness in the connective tissue of the immobilized limb may stimulate free nerve endings and proprioceptors, such as Pacini and Rufini corpuscles (Yahia, Rhalmi et al. 1992; Stecco, Gagey et al. 2007; Tesarz, Hoheisel et al. 2011), in the tissue producing pain (Bell and Holmes 1992; Stecco, Meneghini et al. 2014). Shoulder pain on the paretic side is common after stroke and is strongly associated with abnormal shoulder joint examination, ipsilateral sensory abnormalities and arm weakness (Gamble, Barberan et al. 2002). Deafferentation and sensory loss may also lead to the development of neuronal hypersensitivity and eventually chronic central pain (Boivie, Leijon et al. 1989; Rausell, Cusick et al. 1992; Klit, Finnerup et al. 2009), which is often difficult to treat. Pain can lead to learned nonuse which may persist even after the pain has resolved.

Learned bad-use

When the paretic limb is forced to move, weakness, sensory impairments, and pain can prevent “normal” movement; instead compensatory strategies are used to complete the task(s) (McCrea, Eng et al. 2005). Furthermore, stiffness and contractures resulting from immobility and the development of spasticity and abnormal motor synergies can contribute to compensatory movements. The use of compensatory strategies has been well described for human reaching and grasping after stroke (Levin, Kleim et al. 2009). Patients with stroke use trunk flexion rather than elbow extension to reach for objects (Cirstea and Levin 2000), forearm pronation and wrist flexion rather than neutral forearm position and wrist extension to orient the hand for grasping, and metacarpophalangeal (MCP) joint flexion rather than proximal interphalangeal (PIP) joint flexion to grasp objects (Raghavan, Santello et al. 2010). While the use of compensatory strategies may lead to initial success in completing a task, over time success is reduced due to poor accuracy, which increases the probability of failure. Reinforcement of the abnormal strategy by occasional successes can lead to it becoming a bad habit over time (Skinner 1938), and performance will decline despite extended training because the abnormal behavior is repeated and reinforced at the cost of the correct pattern of behavior (Dickinson 1985). Thus in the absence of appropriate feedback and correction of the abnormal motor behavior “learned bad-use” develops. When the focus of training is to reduce compensatory behaviors, for example, when the trunk is restrained during reach practice, the typical use of a more normal pattern of reaching by extending the elbow is restored along with a reduction in overall impairment (Michaelsen, Luta et al. 2001; Woodbury, Howland et al. 2009). Spasticity and in-coordination due to abnormal motor synergies can lead to the development of learned bad-use.

Spasticity is defined as a motor disorder characterized by a velocity-dependent increase in muscle tone with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex, as one component of the upper motor neuron syndrome (Lance 1980). The prevalence of spasticity increases with time since stroke (Watkins, Leathley et al. 2002), and is related to the secondary effects of weakness and immobility on skeletal muscles (Hufschmidt and Mauritz 1985; Dietz and Berger 1995; Lundstrom, Terent et al. 2008). Initially spasticity is considered a positive development as it suggests that the nervous system is beginning to initiate repair mechanisms to restore muscle tone and movement. Indeed patients who

demonstrate spasticity are further along in their recovery than individuals who are more flaccid (Brunnstrom 1966; Brunnstrom 1970). However, when the threshold for reflex activity continues to reduce due to progressive re-organization of the supraspinal descending drive to the spinal cord, peripheral structures of the muscle, muscle spindles and fascia are further shortened and spasticity evolves into stretch-sensitive forms such as spastic-co-contraction (Gracies 2005). Spastic co-contraction refers to inappropriate antagonist recruitment triggered by volitional command (Gracies 2005). Clinically, spastic co-contraction leads to involuntary movement in the opposite direction of the intended voluntary movement and contributes to impairment in active function. The degree of spastic co-contraction has been shown to be positively related to the Fugl-Meyer Score (Chae, Yang et al. 2002; Aluru, Lu et al. 2014), suggesting that individuals with spastic co-contraction, although significantly impaired, are further along in their recovery process. Spasticity and spastic co-contraction may however lead to learned bad-use. Reduction in spasticity with Botulinum toxin injections has been shown to improve kinematic parameters such as velocity and smoothness, without significant changes in clinical outcomes such as hand function (Bensmail, Robertson et al. 2010). It is possible that weakness and atrophy produced by the injections negates the benefit from improvement in the form of the movement, or that functional improvement requires aggressive concurrent therapy (Canning 2009).

Abnormal motor synergies have been well described post stroke (Brunnstrom 1970). For example, during reaching, the shoulder must flex while the elbow extends. However in patients with stroke attempts at voluntary forward reaching often result in shoulder abduction and elbow flexion due to the constraining effect of abnormal descending motor commands (Beer, Ellis et al. 2007). The abnormal muscle synergies were not related to proximal weakness or abnormalities in the elbow flexor-extensor strength balance. More recently it has been found that the common drive to muscles that are functionally coupled during reaching in healthy individuals, for example, the anterior deltoid and triceps brachii, is weakened after stroke likely due to interruption of information flow in the corticospinal pathway (Kisiel-Sajewicz, Fang et al. 2011). These abnormal muscle synergies have been shown to be independent of weakness, slowness of muscle activation, excessive co-contraction and spasticity and reflects a loss of skill in generating spatial and temporal muscle activation patterns which conform with environmental demands (Canning, Ada et al. 2000). Training strategies that promote movement outside of the abnormal motor patterns may be needed to retrain more normal movement patterns.

Forgetting

Once a motor skill is attained through training, there is an expectation that it will be retained forever, despite intervals of no training (in the same way that one never forgets how to ride a bicycle). However, rats with motor cortex injury show a decline in performance during intervals of no training, and additional training is required to get performance back to pre-training levels (Whishaw, Alaverdashvili et al. 2008). Breaks in rehabilitation similarly lead to forgetting of upper extremity motor skills in humans post stroke (Takahashi and Reinkensmeyer 2003; Krakauer 2006). Thus new skills, while reasonably stable in healthy individuals, are more transient post stroke. Skill learning requires that at least three

independent processes occur across multiple time scales (Huang, Haith et al. 2011). First precise task-specific sensory-motor mappings occur through trial-and-error adaptation during practice with appropriate error sensing. Adaptation is a fast learning process (Joiner and Smith 2008), which leads to a rapid reduction in movement error, and typically takes only a few trials (Gordon, Westling et al. 1993); however it is easily forgotten (Benson, Anguera et al. 2011; Schweighofer, Lee et al. 2011). The second process is repetition, which alters movement biases depending on what is repeated. It leads to a slow tuning of directional biases towards the repeated movement (Galea and Celnik. 2009). A task can be repeated with or without adaptation to error, and does not require error sensing. The third process is reinforcement whereby movements are rewarded intrinsically or extrinsically and reward leads to faster re-learning or savings on subsequent attempts (Haith, Huberdeau et al. 2015). Although these three processes occur independently, it has been shown that learning is most successful when sensorimotor adaptation is combined effectively with repetition (Huang, Haith et al. 2011) (Figure 3). For instance, appropriate sensory-motor mappings learned through adaptation must be repeated over time for sustained and appropriate changes in skill to occur (Bastian 2008). Impaired sensorimotor adaptation and lack of opportunities for long-term practice can lead to unlearning or forgetting after stroke (Kitago, Ryan et al. 2013).

Studies from several laboratories have shown that ***adaptation of reach and grasp*** are impaired post stroke despite reasonable amounts of repetition with the affected hand (Hermsdorfer, Hagl et al. 2003; Nowak, Hermsdorfer et al. 2003; Raghavan, Krakauer et al. 2006; Raghavan, Santello et al. 2010). This suggests that patients may be unable to effectively sense the error with their affected hand and/or subsequently update their motor behavior. Adaptation requires specific sensory inputs: kinesthetic sense from muscle forces used to lift objects is required to produce fingertip load forces appropriate for object weight (Johansson and Westling 1988); tactile sensation from touch receptors is required to produce grip forces appropriate for object texture, with higher grip forces needed to hold smoother objects (Johansson and Westling 1984); and visual input about object contours determines how the hand is shaped during reach (Sakata, Taira et al. 1997; Santello and Soechting 1998; Marino, Stucchi et al. 2010). In reaching experiments, both vision and proprioception provide information about arm configuration, but faulty integration of visual and proprioceptive signals may introduce errors in motor planning (Gordon, Forssberg et al. 1991; Scheidt, Conditt et al. 2005; Sarlegna, Przybyla et al. 2009); this might explain why we close our eyes when we want to enhance feeling. Thus, although multiple sensory contexts may collaborate to maintain task performance (Holmes and Spence 2005), they can also compete and interfere with the acquisition of accurate sensorimotor associations (Gordon, Forssberg et al. 1991; van Beers, Baraduc et al. 2002; Cole 2008). In the presence of sensory deficits after a stroke, however, one sensory context may substitute for another to improve the accuracy of sensory-motor maps (Quaney, He et al. 2010). Information about how and when sensory substitution should be utilized is key to the development of effective rehabilitation protocols for the recovery of motor skill. Even mild sensory and/or motor deficits can impair error-sensing and affect adaptation of movements and forces with the affected hand post stroke (Raghavan, Krakauer et al. 2006; Raghavan, Santello et al. 2010). Thus, the first step in overcoming learned bad-use and forgetting is to facilitate the

formation of sensory-motor mappings or adaptation, which can then be repeated and reinforced for faster re-learning during subsequent encounters.

Therapeutic Considerations

A key consideration to determine treatment may be to first examine which impairment(s) are contributing to the present functional status of the patient. If weakness and immobility are predominant and leading to nonuse, then interventions that potentiate excitatory plasticity (Figure 1) may be warranted. On the other hand, if spasticity, spastic co-contraction and abnormal motor synergies are predominant and lead to the use of abnormal compensatory strategies to accomplish the task, one might consider interventions that potentiate inhibitory plasticity (Aluru, Lu et al. 2014). Since patients with paresis will likely evolve to develop some degree of spasticity, the intervention(s) may need to evolve with the stage of recovery.

Furthermore, in chronic patients with stroke, due to the layering of impairments, it may be important to bear in mind that treatment of one of the impairments might unmask other underlying impairments. For example, spasticity and weakness often co-exist. Therefore treatment of spasticity may unmask underlying weakness which might now need specific intervention. It may be necessary to work on several underlying impairments simultaneously for the best results, and the treatment regimen may need to be individualized for each patient.

Clinical Outcomes

Simple self-report measures used to characterize weakness in the upper limb after stroke, such as the NIH Stroke Scale, and the Stroke Impact Scale provide information about degree of impairment particularly in severely affected individuals, but are not sensitive to mild or moderate weakness of the upper limb after stroke (Bohannon 2004).

The Fugl-Meyer scale is based on the observation of sequential recovery of motor function by Twitchell and Brunnstrom (Twitchell 1951; Brunnstrom 1966; Brunnstrom 1970). It is the most widely-used quantitative measure of motor recovery post stroke (van Wijck, Pandyan et al. 2001; Gladstone, Danells et al. 2002), the scores have been shown to correlate with the extent of corticospinal tract damage (Zhu, Lindenberg et al. 2010). The minimal detectable change on the upper extremity component of the Fugl-Meyer scale is found to be approximately 8% of the maximum score of 66 (5.28 points), supporting its utility in clinical settings (Rabadi and Rabadi 2006; Lin, Hsu et al. 2009). However the Fugl-Meyer Scale was constructed on the assumptions that recovery proceeds in a proximal-to-distal fashion and from synergistic-to-isolated movements (Fugl-Meyer, Jaasko et al. 1975; Gladstone, Danells et al. 2002); however, both these assumptions have been contested recently (Woodbury, Velozo et al. 2007; Beebe and Lang 2008; Crow and Harmeling-van der Wel 2008). Furthermore, the Fugl-Meyer scale may show ceiling effects for fine motor skills in higher functioning patients (Thompson-Butel, Lin et al. 2014).

Grip strength has been found to be a useful objective measure of motor impairment, particularly the rate of increase in grip forces (Renner, Bungert-Kahl et al. 2009). Task-based kinematic measures such as speed and extent of isolated joint range of motion

(Raghavan, Santello et al. 2010; Aluru, Lu et al. 2014), might be useful direct, objective and reliable measures of movement ability. In fact it has been shown that active range of motion early on predicts function at later time points (Beebe and Lang 2009). However, longitudinal measurements of active range of motion may not show a linear improvement profile as the Fugl-Meyer Scale does, especially when spasticity and spastic co-contraction set in. Hence more than one type of measurement and frequent assessment of motor impairment to inform a change in strategy to target the critical impairment may be warranted in the clinical setting.

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Key Points

- Weakness or paresis is the key impairment early on that leads to learned nonuse. Sensory impairment, immobility and chronic pain may further contribute to learned nonuse.
- Spasticity, spastic co-contraction and abnormal motor synergies occur as recovery proceeds and may lead to abnormal compensatory movements, which if repeated and reinforced will lead to learned bad-use.
- Impairment in sensorimotor adaptation can lead to transient retention of new skills despite extensive practice; this is referred to as forgetting.

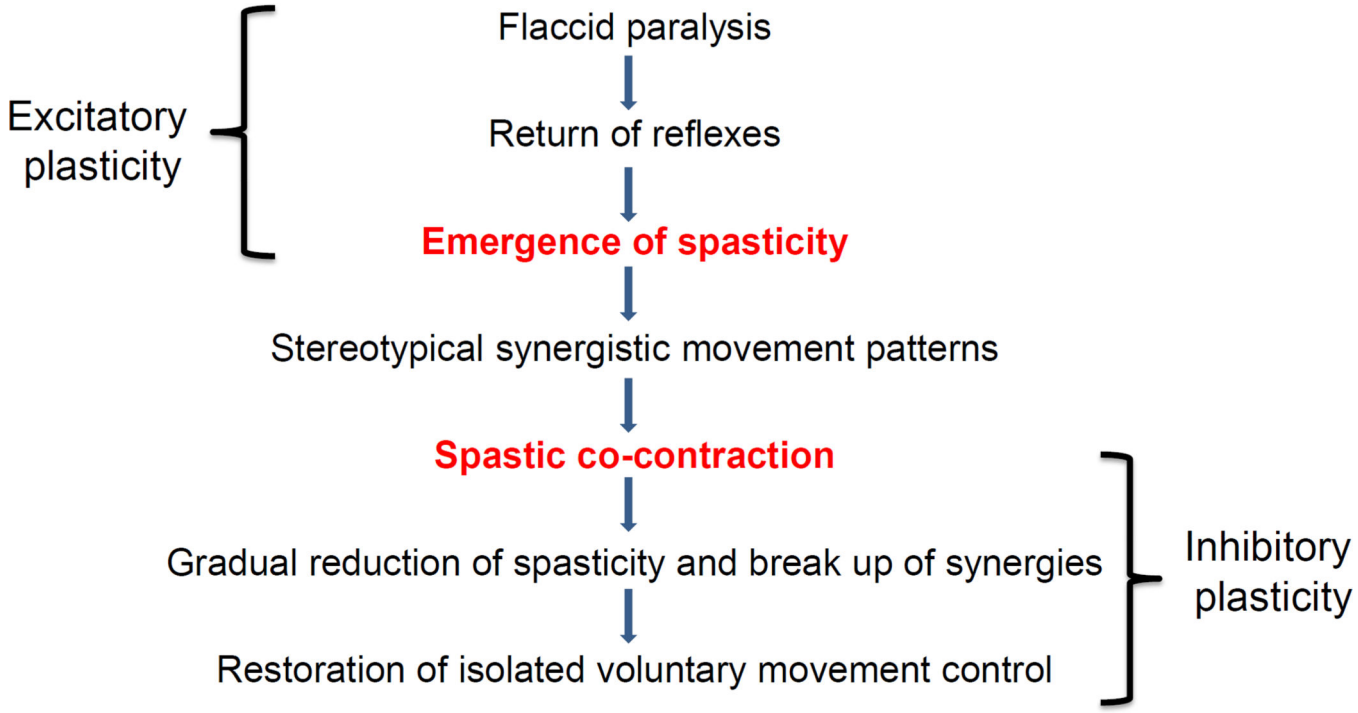

Figure 1.

Sequential progression of motor recovery as described by Twitchell and Brunstrumm. Note that while recovery is proceeding from one stage to the next, residual impairment from preceding stages may still be present leading to the layering of impairment. Also note the underlying physiological processes that may account for progression from one stage to the next.

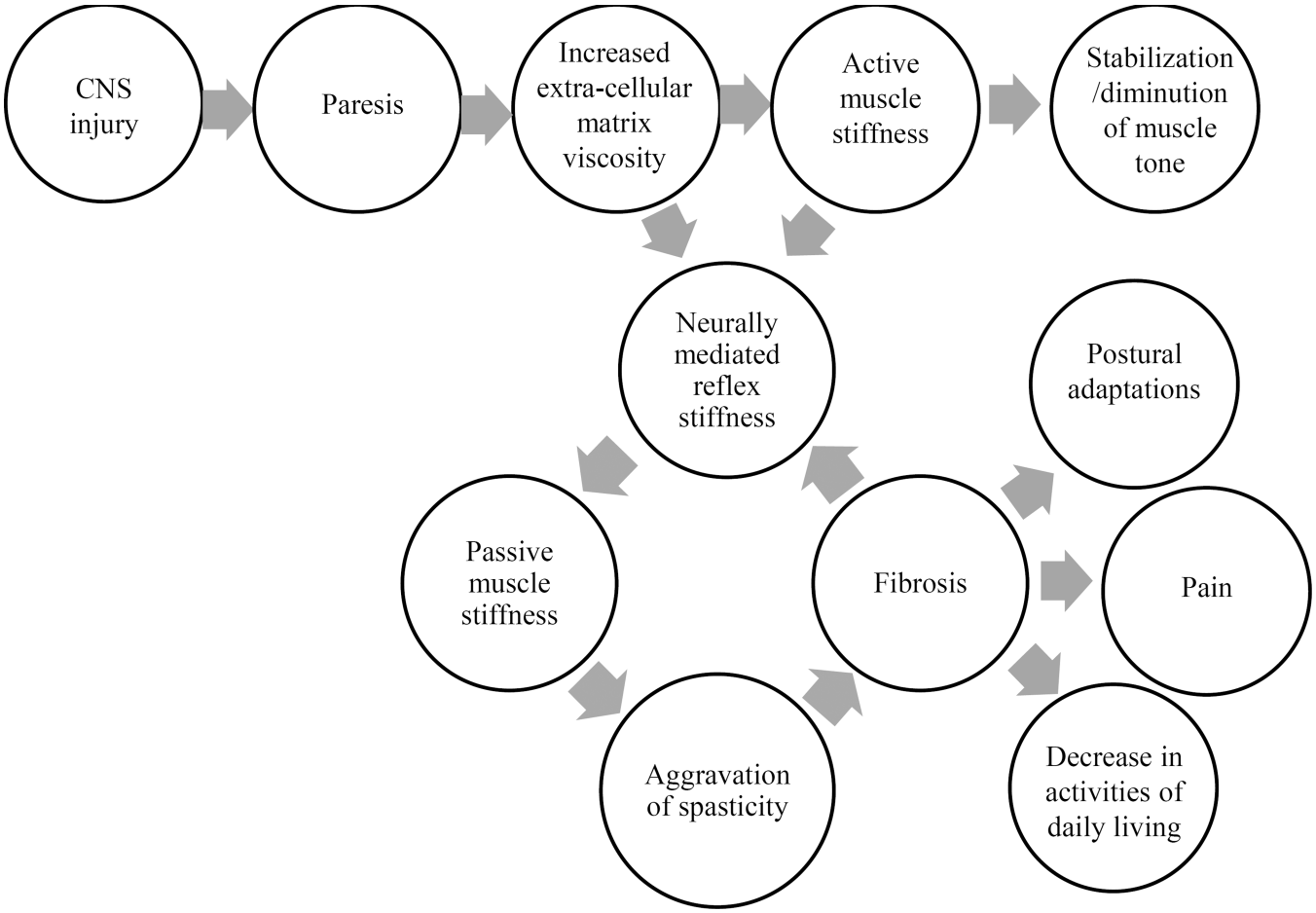

Figure 2.
Model of the contribution of paresis and immobility to the evolution of spasticity. (Adapted from Stecco et al., 2014, *Current Physical Medicine and Rehabilitation Reports*)

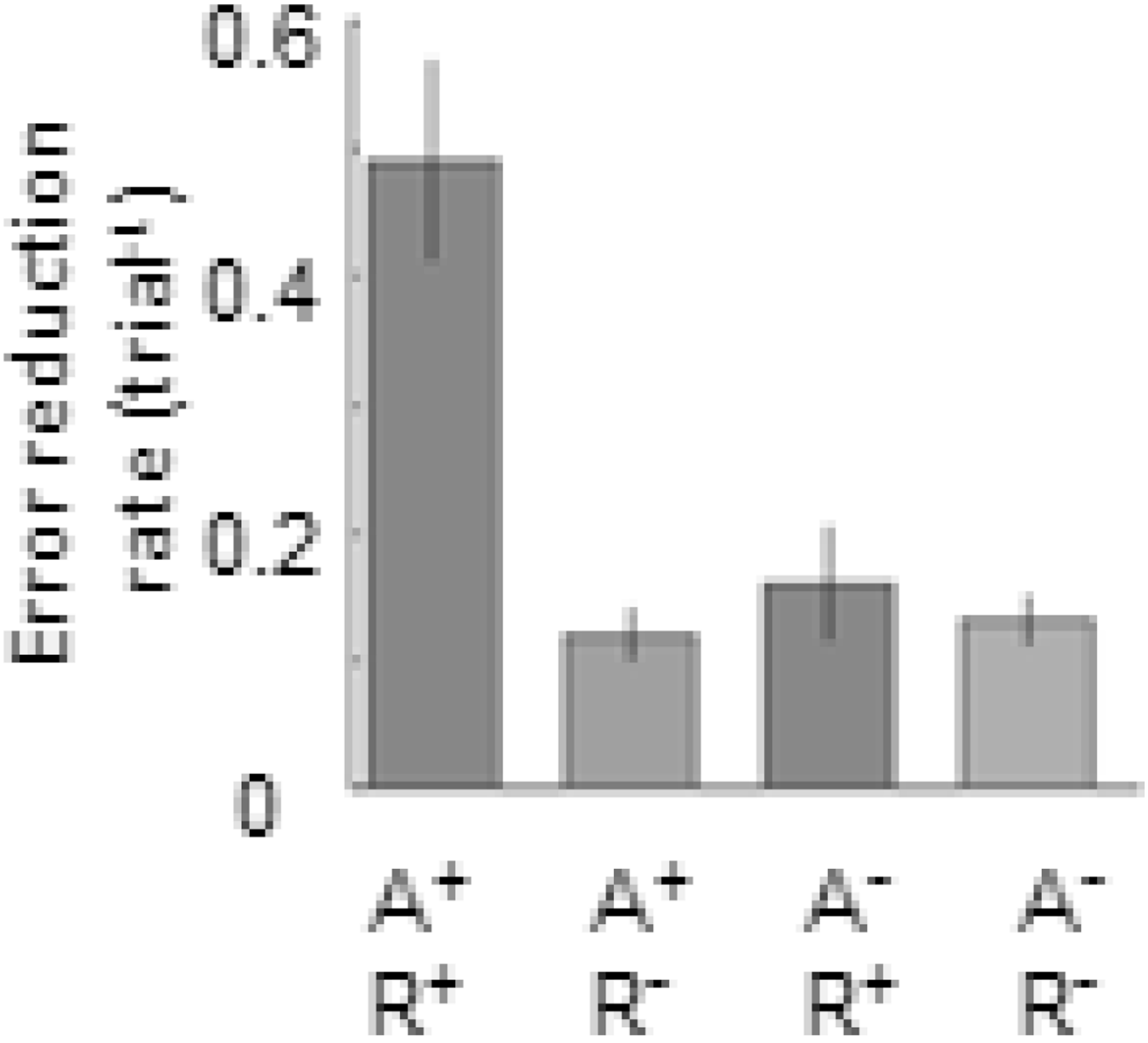

Figure 3.
Error reduction rates, reflecting learning, are greatest when adaptation (A) and repetition (R) combine. (Adapted from Huang et al., 2011, *Nature Neuroscience*)