Muscle Activation Patterns and Postural Control Following Stroke

S. Jayne Garland, Vicki L. Gray, and Svetlana Knorr

Many stroke survivors have residual sensorimotor deficits that impact negatively on balance and quality of life. The purpose of this review is to provide an overview of the impairments in motor control following stroke and the impact of those impairments on muscle activation patterns during postural control in stroke. Motor control impairments following stroke result in force production that is slow, weak and lacking in precision making it difficult to produce a fast rate of force development with sufficient magnitude to be effective for postural responses. Whether postural perturbations require feedback or feedforward responses, there is impairment to the timing, magnitude and sequencing of muscle activation following stroke. The impairment in muscle activation is dependent on the extent of the motor control impairments and strategies used by the individuals following stroke to compensate for the impairments. The central nervous system uses a variety of mechanisms to improve the muscle activation patterns needed for the recovery of postural responses following stroke.

Keywords: electromyography, force, balance, movement

Many stroke survivors have residual sensorimotor deficits that impact negatively on balance and quality of life. Indeed, it has been reported that 83% of patients 2–4 weeks poststroke had a balance disability and practicing balance and walking activities comprised 34% of physiotherapy treatment (Tyson & Selley, 2006). The impact of standing balance on activities of daily living can be significant, in that standing balance is associated with ambulatory ability and independence in gross motor function (Berg, Wood-Dauphinee, & Williams, 1995; Patterson et al., 2007; Piotrowski & Cole, 1994; Tyson, Hanley, Chillala, Selley, & Tallis, 2007). The ability to stand and walk influences hospital length of stay (Wee, Bagg, & Palepu, 1999) and discharge disposition (Wee & Hopman, 2005). Thus, regaining independent standing balance and ambulation is of paramount importance for patients recovering from stroke. The purpose of this review is to provide an overview of the impairments in motor control following stroke and the impact of those motor control impairments on muscle activation patterns during postural control in stroke.

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Motor Control Impairments

There are several motor control impairments that accompany stroke that are particularly relevant for postural control, including slow movements, weakness, fatigue, and incoordination. These impairments in the motor system are associated with a reduced number of motor units (Hara, Akaboshi, Masakado, & Chino, 2000; McComas, Sica, Upton, & Aguilera, 1973; Young & Mayer, 1982) putatively caused by reduced trophic inputs to the motor unit from the descending inputs (Dattola et al., 1993; Lindberg et al., 2007; McComas et al., 1973). The number of motor units has been shown to decrease as early as 9 days after stroke and continue over the course of a year (Hara, Masakado, & Chino, 2004). Evidence suggestive of an early denervation process followed by a reinnervation process supports the notion of a structural rearrangement of motor units (Dattola et al., 1993). Fast twitch motor units may be more susceptible to death or atrophy because they are harder to excite (Henneman, Somjen, & Carpenter, 1965), leaving a muscle comprised of a higher proportion of slow twitch motor units as seen in human gastrocnemius (Dattola et al., 1993) and flexor hallucis longus muscle (Hachisuka, Umezu, Ogata, 1997). In quadriceps, however, atrophy of type II fibers was evident without a concomitant hypertrophy of type I fibers (Hachisuka, Umezu, Ogata, 1997). In the presence of muscle atrophy, individuals following stroke would need to increase the number of motor units activated to achieve any given force, e.g., less force is generated per unit of electromyographic (EMG) activity (Tang & Rymer, 1981), which may also increase the sense of effort (Enoka & Stuart, 1992) experienced poststroke. The loss of fast twitch motor units and/or atrophy of remaining type II muscle fibers could reduce the speed of contractions contributing to slow movements and muscle weakness.

Motor units in the first dorsal interosseus muscle in individuals with long-term hemiparesis (mean, 26 weeks) were found to produce larger forces with slower contraction times in both fast and slow motor units (Young & Mayer, 1982), which could be explained by collateral sprouting. These authors also found an increase in the fatigability of motor units and the emergence of motor units that they coined as “slow-twitch fatiguable” (Young & Mayer, 1982). Despite this interesting finding over 20 years ago, there are relatively few studies that have investigated neuromuscular fatigue poststroke. Riley & Bilodeau (2002) found that individuals following stroke were unable to maximally activate paretic muscles, which got progressively worse during a sustained maximal contraction. Lower levels of fatigue were found on the paretic versus nonparetic side; this has been attributed to the lower forces being produced by the paretic muscle which might not have been sufficient to induce muscle fatigue or muscle fiber rearrangement with an increased proportion of type I fibers (Toffola, Sparpaglione, Pistorio, & Buonocore, 2001).

The coordination of movement requires varying the levels of activation of the agonist and antagonist, depending on the task requirements. Reciprocal inhibition and recurrent inhibition are two mechanisms that modulate the amount of antagonist and agonist activation, respectively. Reciprocal inhibition during a movement promotes the reduction of antagonist muscle activation; in that activation of the agonist muscle causes inhibition of the antagonist muscle via the Ia inhibitory interneurons (Jankowska & Roberts, 1972). The strength of reciprocal inhibition
is modulated by descending supraspinal excitation or inhibition of the Ia inhibitory interneurons (Crone, Hultborn, Jespersen, & Nielsen, 1987); indeed, presynaptic inhibition of the antagonist can be seen before the onset of voluntary movement (Pierrot-Desilligny & Lacert, 1973). In subjects with marked extensor spasticity in the lower limb, reciprocal inhibition at the onset of ankle dorsiflexion onto the ankle plantarflexors was weak (Crone et al., 1987; Morita, Crone, Christenhuis, Petersen, & Nielsen, 2001; Yanagisawa & Tanaka, 1978) and presynaptic inhibition of Ia afferents in the ankle plantarflexors was diminished at the onset of ankle dorsiflexion (Morita et al., 2001), making the production of ankle dorsiflexion force more difficult.

Recurrent inhibition of the agonist muscle via the Renshaw cells is modulated by supraspinal control (Smith, 1981). An increase in recurrent inhibition was observed in healthy subjects performing low intensity voluntary contractions, whereas in strong contractions the increase in recurrent inhibition was less to support larger agonist activation (Katz, Pierrot-Deseilligny & Hultborn, 1982). Following upper motor neurone lesions, the resting levels of recurrent inhibition were higher and subjects displayed less ability to modulate the level of recurrent inhibition during voluntary muscle contraction (Katz et al., 1982). This higher level of recurrent inhibition may be related to the lower firing rates of motor units during contraction evident following stroke (Hu, Tong, & Hung, 2006; Zhou, Suresh, & Rymer, 2007). Thus, impairment in the supraspinal modulation of spinal circuitry may influence the quality of motor control following stroke.

Cocontraction, the concurrent activation of agonist and antagonist muscles, can be inefficient for joint movement or agonist force production because of the opposing force generated by the antagonist. However, it may be beneficial in maintaining joint stability under heavy loads (Baratta et al., 1988), enhancing postural stability of the limb (Levin & Dimov, 1997), or improving the accuracy of multijoint movements (Gribble, Mullin, Cothros & Mattar, 2003). Cocontraction is commonly seen in novel tasks but as the new task is learned, cocontraction decreases (Gribble et al. 2003; Thoroughman & Shadmehr, 1999). There is a commonly-held belief that, following stroke, high levels of antagonist cocontraction across a joint make the activation of a focal muscle group appear weak, based in part on the aforementioned impairments in reciprocal inhibition. For instance, alcohol block of the spastic plantarflexors has been shown to result in increased strength in the ankle dorsiflexor muscles (Yanagisawa & Tanaka, 1978). This improvement only occurred in the 4 of 11 subjects who had marked extensor spasticity. Spasticity has been associated with a reduction in the threshold for muscle activation in response to passive stretch after stroke (Chung et al. 2008; Levin & Feldman 1994). However, Morita et al. (2001) found that the impairment in reciprocal inhibition and presynaptic inhibition in subjects with spasticity did not influence maximal force production as almost all their subjects were able to produce maximal contractions within normal values. Hammond et al. (1988) also advocated that cocontraction blocked movement; however careful inspection of the data reveals that inadequate activation of the agonist musculature could have been more of a factor in the weakness than the concomitant elevated antagonist muscle activity. Kamper & Rymer (2001) concurred that both cocontraction of the finger flexor and extensor muscles and decreased voluntary excitation of the extensors limited finger extension movements poststroke. Levin and colleagues (2000)
found that, in mid ranges of active movement, individuals with hemiparesis following stroke were able to produce reciprocal activation of the elbow flexor and extensor muscles, but cocontraction became more prominent in outer or inner ranges of movement. Inadequate agonist activation during upper extremity movement has been documented in other studies (Gowland, deBruin, Basmajian, Plews, & Burcea, 1992; Sahrmann & Norton, 1977). Many studies have not found cocontraction (Garland & Hayes, 1987; Gowland et al., 1992) or spasticity in the antagonist muscle (Nadeau, Gravel, Arsenault, Bourbonnais, & Goyette, 1997; Sharp & Brouwer, 1997) to be related to weak contractions. Indeed, there are also reports that, although the strong reflex response to stretch that is a hallmark feature of spasticity occurred in a passive muscle, under active conditions there was little difference in the reflex response to stretch between paretic and nonparetic limbs (Ada, Vattanasilp, O’Dwyer, & Crosbie, 1998; Ibrahim, Berger, Trippel, & Dietz, 1993). Thus, the influence of spasticity and antagonist muscle activity on torque development may be less important in active conditions and in mid ranges of movement.

There is a large body of knowledge demonstrating that the production of a fast movement incorporates a triphasic (or three burst) muscle activation pattern (for example, Cooke & Brown, 1990; Gottlieb, Song, Hong, & Corcos, 1996; Hallett, Shahani, & Young, 1975; Marsden, Obeso, & Rothwell, 1983; Mustard & Lee, 1987). The triphasic muscle activation pattern is characterized by an initial agonist EMG burst to set the limb in motion, then an antagonist muscle burst to provide a braking action, followed by a second agonist burst to fine-tune the movement as the desired distance is approached. Although studied most commonly in the upper extremity, triphasic pattern of muscle activation was evident with rapid single joint movements of the knee and ankle (Aruin, 2001). The presence of a triphasic pattern is dependent on the velocity of the movement (Mustard & Lee, 1987). That is, as movement velocity increases, there is an increase in the size of the first agonist and antagonist muscle bursts (Marsden et al., 1983). Individuals following stroke have been shown to lack this coordinated muscle activation pattern (Fagioli, Berardelli, Hallett, Accornero, & Manfredi, 1988; el Abd, Ibrahim, & Dietz, 1993; McCrea, Eng, & Hodgson, 2005). For example, individuals with upper motor neurone lesions have been shown to have difficulty turning off the initial agonist burst (Mizrahi & Angel, 1979; Sahrmann & Norton, 1977), making it difficult to produce reciprocating movements. With insufficient central drive to the muscle after stroke, the first agonist burst would be small resulting in slower movements and little need for the subsequent bursts that control the limb deceleration. Impaired coordination of muscle activation and torque generation across joints in the upper extremity has been shown in isometric and movement conditions, especially abnormal linkages between shoulder abduction and elbow flexion and shoulder adduction and elbow extension (Beer, Dewald, Dawson, & Rymer, 2004; Ellis, Acosta, Yao, & Dewald, 2007; Dewald & Beer, 2001). Thus, the impaired coordination of muscle activation results in a reduction of net force production needed for movement.

Impairments in motor control also affect the speed of movement in functional movements. Functional movements, such as squatting and stepping movements, require the coordination of muscle activity in several lower limb and trunk muscles (Cheron, Bengoetxea, Pozzo, Bourgeois, & Draye, 1997; Hase, Sako, Ushiba,
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& Chino, 2004; MacKinnon et al., 2007; Rocchi, Mancini, Chiari, & Cappello, 2006). The findings from our laboratory demonstrate that individuals following stroke who have significant motor impairment have difficulty coordinating the muscle activity across muscles bilaterally during a fast squatting task, which in turn was associated with slow performance of the task in comparison with healthy subjects (Figure 1). Figure 1 depicts the EMG recordings during a single squat in a subject one month poststroke (right panel) and a healthy age- and sex-matched control (left panel). The healthy subject initiates the squat with activation of tibialis anterior and controls the deceleration of the squat with activation of quadriceps and hamstrings. In the subject poststroke, the moderate level hemiparesis of the foot and ankle resulted in a compensatory increase in EMG activity in the nonparetic leg and an asymmetrical squat, with the nonparetic leg starting over 100 ms before the paretic leg. It is not clear whether individuals who compensate with increased use of the nonparetic leg have sensorimotor impairments in the paretic limb that cannot be overcome or whether there is a pattern of learned nonuse of the paretic limb (Mark & Taub, 2004).

Although the impairments in motor control following stroke are well documented, there is also evidence of neural plasticity with rehabilitation or practice (for recent reviews, see Forrester, Wheaton & Luft, 2008; Dobkin, 2008; Cramer & Riley, 2008). Nudo et al. (1996) demonstrated that alterations in the primary motor cortex of adult squirrel monkeys poststroke could be induced through functional use of the limb (described as rehabilitation by the authors). Constraint-induced movement therapy, which forces an increased use of the paretic limb by immobilizing the nonparetic limb, was associated with increased motor cortex activation (Wittenberg et al., 2003). Indeed, even short sessions of skilled target tracking practice resulted in increased motor cortex activation (Carey et al., 2002, 2004; Liepert, Graef, Uhde, Leidner, & Weiller, 2000) and improved movement kinematics (Cirstea, Ptito, & Levin, 2003). Specific to postural control, Marigold et al. (2005) found that exercise focusing on agility (fast-paced dynamic movements) resulted in faster step reaction times and earlier muscle onset latencies to a force platform translation perturbation than stretching and weight shifting exercises. This suggests that the deficits in muscle activation may be associated with the type of exercise or habitual activity engaged in after stroke.

In summary, a constellation of motor control impairments following stroke result in force production that is slow, weak and lacking in precision. Stroke induced impairments (Figure 2), such as increased recurrent inhibition of spinal motoneurones and impaired descending inputs to muscles contribute to slow motor unit firing rates. Remodeling of the hemiparetic muscle leads to slower contractile properties and weakness. Impaired coordination of muscle activity reduces the efficiency of force production in movements and functional tasks. In terms of postural control, these impairments make it difficult to produce force in postural muscles with sufficient speed and magnitude to be effective for postural responses to perturbations.

Postural Control

Standing balance requires that an individual has the ability to stand erect and also the ability to withstand external and internal perturbations to stance (Alexander,
**Figure 1** — EMG recordings during a single squat in a subject 1 month poststroke (right) and a healthy age- and sex-matched control (left). The traces top to bottom are acceleration (ACC) of the right knee, right quadriceps (QUAD), hamstrings (HAM), tibialis anterior (TIB), soleus (SOL), ACC of the left knee, left QUAD, HAM, TIB and SOL. The vertical line at the onset of the knee acceleration denotes the onset of the squat. Note the symmetric pattern of acceleration bilaterally in the healthy subject versus the individual poststroke. In the healthy subject, the squat is initiated with activation of tibialis anterior (denoted with arrows) and deceleration is controlled with activation of the quadriceps and hamstrings (shaded region). In the subject poststroke, the nonparetic leg moves in advance of the paretic leg with substantially higher EMG. The paretic leg has a moderate level of hemiparesis (Chedoke McMaster Stroke Assessment score of 3/7 and 5/7 in the foot and leg, respectively) resulting in a paucity of EMG activation to initiate the movement.
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1994; Horak, 1987; Patla, Frank, & Winter, 1992). External perturbations, which result from unexpected forces being applied to the body, incorporate “feedback” control of posture in which muscles are activated after the disruption starts in an attempt to keep the center of mass (COM) within the base of support. Postural disruptions that result from voluntary movement of the body are examples of internal perturbations. This type of perturbation involves “feedforward” or anticipatory control of posture in which muscles are activated in advance of the focal movement to minimize the displacement of the individual’s COM with respect to the base of support.

There are three global requirements for balance. First, sensation (e.g., visual, vestibular, proprioceptive/somatosensory) is required to detect or anticipate a postural disturbance. Second, neural processing is required to select appropriate feedback and feedforward postural control mechanisms. The third requirement is an effective motor output. The muscles must produce the required forces in the proper timing to maintain standing balance. The relative roles of sensation, neural processing, and motor output in postural control impairments following stroke remain to be determined. First, there is a pivotal role for somatosensation in maintaining upright stance (Fitzpatrick, Rogers, & McCloskey, 1994) and withstanding perturbations (Stapley, Ting, Hulliger, & Macpherson, 2002). Although strong coupling has been found between tactile inputs from the sole of the foot and motor neurones innervating the ankle plantarflexors in healthy subjects (Fallon, Bent, McNulty, & Macefield, 2005), tactile sensation of the plantar aspect of the foot was not related to the incidence of falls (Marigold, Eng, Tokuno, & Donnelly, 2004) and proprioceptive deficits were not linked with weight bearing asymmetry or postural instability in standing (Roerdink, Geurts, De Haart, & Beek, 2009) in individuals after stroke. Second, the neural processing of sensory information was affected with stroke, as evidenced when vision and somatosensory information were impaired experimentally with the Sensory Organization Test (Marigold et al., 2004; Smania, Picelli, Gandolfi, Fiaschi, & Tinazzi, 2008). Misperception of

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**Figure 2** — Schematic of motor impairments after stroke.
vertical orientation in standing has also been demonstrated following stroke and shown to impact negatively on balance recovery (Bonan et al. 2006, 2007). Third, the motor output from a structurally-rearranged hemiparetic muscle, as a result of disuse or motor unit remodeling (Lukacs, 2005), could lose the ability to contract rapidly or selectively in postural responses.

This section of the review will provide an overview of the muscle activation patterns during external and internal perturbations of postural control in stroke subjects and healthy controls. Although the literature on postural control is expansive, we will emphasize specific studies with EMG findings that highlight the muscle activation patterns used in standing balance. Muscle activation, as measured by an EMG burst, can be characterized by parameters of slope, peak amplitude and area, and latency of the burst. The initial slope of the EMG burst increases as the velocity of movement increases (Corcos, Gottlieb, & Agarwal, 1989), suggesting that a higher rate of rise of the EMG is required to produce faster muscle contractions needed for movement or postural responses. The peak amplitude and area of the EMG burst increase with an increase in load (Gottlieb et al., 1996), suggesting that the magnitude of the force production is captured in the magnitude of the EMG burst, at least in isometric or near isometric contractions. The latency of the burst with respect to the onset of an internal perturbation reveals the extent to which preprogrammed postural responses are evoked.

**External Perturbations.** When the body is unexpectedly perturbed by a translation of a supporting surface or an imposed change in body position, the COM will shift and muscles are activated to correct the disturbance and regain stability. Researchers have frequently used movements of the supporting surface to examine the response of muscles to external perturbations. The magnitude of the muscular response is shaped by the size and direction of the perturbation (Henry, Fung, & Horak, 1998; MacPherson, 1988). In healthy subjects, backward translations of the supporting surface are characterized by gastrocnemius activation about 100 ms after onset of forward sway to return the body to a vertical orientation (Nashner, 1976); forward translations created similar activation patterns but in the tibialis anterior during backward sway. There is a “distal to proximal” sequence of muscle activation in the leg and trunk in response to horizontal translations of modest amplitude and speed that form a centrally preprogrammed pattern (Nashner, 1977; Nashner, Woollacott, & Tuma, 1979). Modest perturbations can be accommodated using an inverted pendulum model of balance control (Winter, Patla, Prince, Ishac, & Gielo-Perczak, 1998). In this inverted pendulum model, the central nervous system adjusts the center of pressure (COP), which is the neurophysiological response to changes in the body’s COM, to maintain upright stance (Winter, 1990). Larger or faster perturbations would require stronger and earlier activation of the trunk and thigh musculature (Horak & Nashner, 1986) or stepping responses to increase the base of support (Maki & McIlroy, 1997). After stroke, Geurts and colleagues (2004) have suggested that there may be a greater reliance on compensatory stepping behaviors to avoid a fall. These “change of support” strategies are common in healthy individuals but are impaired in older adults (Maki & McIlroy, 1997). The threshold for stepping may be lower because of an altered perception of stability margins, however, further research is required to determine this possible connection.
In individuals following stroke, postural adjustments to external perturbations have been characterized by a slow onset of EMG response and low amplitude of the EMG bursts. A delay in the activation of the paretic gastrocnemius, tibialis anterior, hamstrings, quadriceps (Di Fabio, Badke, & Duncan, 1986) and paretic gluteus medius muscles (Kirker, Simpson, Jenner, & Wing, 2000) has been reported in postural responses to external perturbation. The delay in the onset of the EMG bursts has been linked with an increased incidence of falls, defined as a loss of balance that required support from a safety harness to prevent them from falling to the floor (Marigold & Eng, 2006). In that study, unexpected platform translations of unknown direction were used to observe the postural responses in 44 subjects with hemiparesis at least 1-year poststroke. Subjects that fell during the study demonstrated delayed paretic tibialis anterior, and paretic and nonparetic rectus femoris onset latencies in response to forward translations. In addition, 11 subjects fell with forward platform translations while only 3 fell in response to the backward translations. The authors suggested that this might stem from the biomechanics of the foot as the COP under the foot has less opportunity for backward excursion as compared with forward excursion.

Berger et al. (1988) examined different magnitudes of short duration (90 ms) treadmill displacements on ankle muscle activation of 11 subjects with spastic hemiparesis, on average 6 years post onset of symptoms. They found a delayed onset of muscle activity in the paretic gastrocnemius and tibialis anterior in response to backward and forward perturbations, respectively. The EMG amplitude was smaller on the paretic side compared with the nonparetic side. Subjects were able to modulate the EMG activity to changing acceleration magnitude, with peak burst EMG amplitude increasing as acceleration increased, with no change in onset latency.

Marigold et al. (2004) examined the effect of weight bearing on muscle activation in response to perturbations of unexpected onset and direction in 10 individuals with hemiparesis at least 1 year poststroke. Subjects experienced relatively slow translations (3 m/s²) over 8 cm in three different conditions; equal weight bearing, 70% weight bearing on the paretic leg, and 70% weight bearing on the nonparetic leg. The postural responses in the hemiparetic subjects revealed delayed muscle activation in the paretic side when compared with the nonparetic side and healthy controls. The timing of the onset of the burst was consistent across different weight bearing conditions in individuals following stroke, whereas the onset latencies in gastrocnemius shortened with increased weight bearing in control subjects. Although stroke patients were unable to modulate the timing of the muscle activation with weight bearing status, they were able to increase the magnitude of the EMG burst with greater weight bearing suggesting that an increase in weight bearing on the paretic leg promotes muscle activity.

The sequencing or pattern of muscle activation across muscles is impaired following stroke. Badke et al. (1987) reported differences in the sequencing of distal and proximal muscle activation to external perturbation. In healthy subjects, the timing difference between distal and proximal muscles was 10–45 ms with the distal muscle being activated first. Subjects with hemiparesis demonstrated a delay in the activation of the paretic leg muscles, in voluntary backward sway and unexpected perturbations, with the proximal muscles being recruited before distal muscles, suggestive of a hip strategy. Impaired lower limb muscle activation
patterns have been reported in other external perturbation studies (Di Fabio et al., 1986; 1987). In an earlier study by Badke and Duncan (1983), subjects with hemiplegia were reported to have more variation in muscle response patterns from trial to trial than healthy subjects, although the most frequent muscle response involved cocontraction of the distal muscles (gastrocnemius and tibialis anterior) and proximal muscles (hamstrings and vastus medialis) on the paretic and nonparietic sides. Subjects with moderate impairment of the lower extremity (50–75% of maximum lower extremity performance score on the Fugl-Meyer, Table 1) had more difficulty organizing and eliciting rapid postural responses than subjects with modest impairment (75–100% of maximum lower extremity performance score on the Fugl-Meyer).

The influence of the predictability of the platform translation perturbation was examined by Badke et al. (1987). They examined 10 subjects, 3 months to 2.5 years poststroke onset with modest lower extremity impairment (85% of maximum Fugl-Meyer score), who were perturbed by either unexpected or expected (after an auditory tone) platform translations. With prior knowledge of platform’s direction of movement, the onset latencies were significantly shorter in the paretic leg than when the perturbation was unexpected. If subjects were given wrong information about the movement direction, onset latencies were prolonged in the paretic leg. Interestingly, subjects had a larger delay in the onset of the EMG burst in voluntary sway (after an auditory tone) with respect to controls suggesting a motor processing problem which is more pronounced during voluntary than automatic reactions. In contrast, Hocherman et al. (1988) studied the postural responses to predictable oscillating horizontal platform translations with persons 1–3 months following stroke. Whereas healthy elderly subjects produced anticipatory sequences of reciprocal muscle activation of gastrocnemius and tibialis anterior, the individuals following stroke tolerated only 10–50% of the platform excursion of healthy elderly controls and did not anticipate the platform movement but rather used tonic cocontraction of the ankle muscles. These subjects were older (mean 68 years versus 56 years) and more acute than those in Badke et al. (1987) and may have been less able to use the information about the perturbation to facilitate the development of an internal postural response pattern to improve the timing and coordination of muscle activation.

It is possible for stroke subjects to learn the appropriate activation patterns after practice from external perturbations as shown by Kirker et al. (2000) in 13 patients ranging from 2 to 31 weeks poststroke. When healthy subjects experienced a sideways push at the hips to the right, there was increased activation of the right gluteus medius muscle followed by right adductor muscle activity; this was often associated with inhibition of the left gluteus medius muscle. Three subjects poststroke were unable to recruit hip muscles on either side regardless of the side of the perturbation. Other subjects who were able to recruit paretic muscles compensated with increased nonparietic muscle activity. Interestingly, the 3 subjects demonstrating no EMG activity in the hip muscles had cognitive impairments. After 30 sideways pushes, the majority of subjects changed their pattern of hip activation toward a more normal activation pattern. Consistent with this observation of short-term effects, Marigold et al. (2005) found that a 10 week exercise program focusing on agility resulted in earlier muscle onset latencies to a force platform translation perturbation in individuals with chronic stroke. Both studies
### Table 1  Clinical Scales

<table>
<thead>
<tr>
<th>Clinical scale</th>
<th>Purpose</th>
<th>Scoring</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fugl-Meyer Assessment of Sensorimotor Recovery After Stroke</td>
<td>Evaluation of:</td>
<td>Items scored on 3-point scale (0–2)</td>
<td>Total motor score:</td>
</tr>
<tr>
<td></td>
<td>• motor recovery</td>
<td>Total motor score = 100</td>
<td>&lt;55 = severe</td>
</tr>
<tr>
<td></td>
<td>• balance</td>
<td>Reliability of lower limb subscale = ICC 0.92</td>
<td>56–79 = moderate</td>
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<tr>
<td></td>
<td>• sensation</td>
<td></td>
<td>&gt;79 = mild</td>
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<tr>
<td></td>
<td>• joint motion</td>
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<td></td>
<td>• pain</td>
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<td></td>
<td>Evaluation of:</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>• severity of shoulder pain</td>
<td>Items scored on 7 point scale (1–7)</td>
<td>&lt;3 = minimal to no movement</td>
</tr>
<tr>
<td></td>
<td>• postural control</td>
<td>Reliability of leg and foot subscales = ICC 0.98, 0.94, respectively</td>
<td>3 = movement in synergy patterns</td>
</tr>
<tr>
<td></td>
<td>• arm motor control</td>
<td></td>
<td>4 = some isolated movement</td>
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<tr>
<td></td>
<td>• hand motor control</td>
<td></td>
<td>5 = more selective movement</td>
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<tr>
<td></td>
<td>• leg motor control</td>
<td></td>
<td>&gt;5 = incoordination, slow movement</td>
</tr>
<tr>
<td></td>
<td>• foot motor control</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Evaluation of functional balance:</td>
<td>Items scored on 5 point scale (0–4)</td>
<td>&lt;37 = high risk of falling in older adults</td>
</tr>
<tr>
<td></td>
<td>• maintenance of upright posture</td>
<td>Total score = 56</td>
<td>32 = mean for orthosis users after stroke</td>
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<tr>
<td></td>
<td>• adjusting balance for voluntary movement</td>
<td>Reliability for clients with stroke = ICC 0.99</td>
<td>42 = mean for non-orthosis users after stroke</td>
</tr>
</tbody>
</table>

*Note. Summarized from Finch, Brooks, Stratford, & Mayo, 2002. Full details on clinical scales can be found in Finch et al., 2002.*
Garland, Gray, and Knorr revealed that muscle activation patterns in response to external perturbations can be developed through practice of dynamic movement.

A schematic summary of how the muscle activation patterns influence postural stability during external perturbations is provided in Figure 3. External perturbations require muscle activation in the legs and trunk to counteract the shift in the COM induced by the perturbation. When the muscle activation is timely, of sufficient amplitude, and coordinated across muscles, the excursion of the recorded COP is large enough to keep the COM well within the base of support (Figure 3A). After stroke, the muscle activation patterns can be 1) delayed in onset because of impaired neural processing; 2) small in amplitude because of low motor unit firing rates or insufficient motor unit recruitment; and/or 3) improperly

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**Figure 3** — Schematic representation of muscle activation (EMG), center of pressure (COP) and center of mass (COM) displacements during external perturbation (horizontal translation of force platform). A. In healthy subjects, the muscle activation is timely, of sufficient amplitude, and coordinated across muscles creating a large excursion of the COP which keeps the COM well within the base of support. B. After stroke, the muscle activations are delayed and low in amplitude producing a smaller COP excursion, which results in a larger shift in the COM potentially approaching the limits of stability. C. By activating the muscle earlier (gray shaded area) or increasing the amplitude of the burst (dashed area), the COP displacement is increased and the shift in the COM is reduced.
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sequenced because of excessive cocontraction or disruption of the centrally programmed pattern of muscle activation. If the muscle activations are delayed, of low amplitude or incoordinated after stroke, the COP excursion will be smaller and the shift in the COM will be larger, potentially approaching the limits of stability (Figure 3B). Figure 3C shows how the shift in the COM can be reduced by activating the muscle earlier or by increasing the amplitude of the burst, even if these two parameters are changed independently of each other. Without a change in the muscle activation pattern to produce a smaller shift in the COM, the individual must compensate by either tolerating smaller external perturbations, increasing the activation of the nonparetic muscles and/or use a stepping strategy to maintain upright posture.

Internal Perturbations. Internal perturbations create a disturbance in balance through limb movement. The postural response to the internal perturbation is a feedforward mode of motor control in which the muscles respond before the self-triggered perturbation. These anticipatory postural adjustments are centrally programmed serving to counteract the effects of a perturbation on postural equilibrium by predicting disturbances and producing a programmed response before the start of the limb movement (Hallett et al., 1975). Triphasic muscle activation in trunk and leg muscles, similar to that seen during limb movement, is used in healthy subjects during postural adjustments associated with fast arm pointing perturbations (van der Fits, Klip, van Eykern, & Hadders-Algra, 1998). Subjects with moderate motor impairment following stroke did not exhibit these sharp bursts of coordinated muscle activation in response to an arm raise perturbation (Garland, Willems, Ivanova, & Miller, 2003; Garland, Ivanova, & Mochizuki, 2007).

Disrupted, reduced, and delayed anticipatory postural responses have been reported in patients poststroke with internal perturbations (Garland et al. 1997; 2003; 2007; Hedman, Rogers, Pai, & Hanke, 1997; Horak, Esselman, Anderson, & Lynch, 1984; Slijper, Latash, Rao, & Aruin, 2002), revealing a failure to coordinate postural muscles to deal with the internal perturbations. Horak et al. (1984) studied 13 patients, ranging from 2 months to 25 years poststroke, in a standing task in which they raised their nonparetic arm to a horizontal position in a variety of conditions, i) fast self-paced, ii) slow self-paced, iii) reaction time, iv) self-paced with weight (0.9 kg). The fast self-paced and the weighted self-paced conditions had a significant delay in the activation of the paretic biceps femoris and paretic paraspinal muscles, respectively, compared with normal subjects. Hedman et al. (1997) used a standing leg flexion perturbation and demonstrated delayed activation of the paretic gluteus medius, as compared with the nonparetic side.

We performed a series of experiments using a unilateral arm raise perturbation in subjects with longstanding hemiparesis following stroke (Figure 4). During a unilateral arm flexion perturbation, healthy individuals demonstrate anticipatory postural adjustments with early activation of the ipsilateral hamstrings muscle followed by the contralateral hamstrings (Figure 4, left). These sharp bursts of muscle activation in the leg muscles were less apparent in age-matched individuals one month poststroke despite performing the unilateral arm raise perturbations with comparable arm acceleration as the healthy subject. The middle panel depicts one subject with marked hemiparesis (Chedoke McMaster Stroke Assessment
score of 2/7 and 3/7 in the foot and leg, respectively; Gowland et al., 1993, Table 1) in which there is little modulation of the paretic muscles and depressed activation of the nonparetic muscles. In the right panel, a subject with a moderate level of hemiparesis shows evidence of nonparetic hamstring activation but high levels of quadriceps activation bilaterally with minimal modulation with the perturbation.

Stevenson and Garland (1996) found that individuals with better functional balance, as measured with the Berg Balance Scale (Berg et al., 1995, Table 1), had anticipatory nonparetic hamstring activation and lower COP excursion speed than subjects with poor functional balance who lacked anticipatory hamstring muscle activation. In a follow-up study, Garland et al. (1997) demonstrated that those subjects with good functional balance, on average 48/56 on the Berg Balance Scale, showed a delay in the onset of the paretic hamstrings activation in comparison with healthy elderly controls. This suggested that individuals with good functional outcomes after stroke still had physiological impairments in muscle activation in response to internal perturbations.

Garland et al. (2003) examined patients poststroke at admission to a rehabilitation program and again 4 weeks later. Overall, there was significant improve-
ment in both functional balance and muscle activation patterns in response to a unilateral arm raise perturbation. There was a significant decrease in the latency of hamstring activation in the nonparetic and paretic leg, indicating an improvement in the feedforward anticipatory response to the upper extremity flexion movement. However, in some subjects the activation of the paretic hamstrings did not improve over the 4 weeks and, instead, subjects increased the feedforward activation of the nonparetic hamstrings. Subjects who used this compensatory strategy had less recovery of functional balance than those who displayed improvement of the onset of muscle activation on the paretic side. The latency of the paretic hamstrings activation was correlated with the stage of motor recovery in the leg and foot, as assessed with the Chedoke McMaster Stroke Assessment, which supports the view that low levels of motor control in the lower limb impact negatively on the effectiveness of postural responses to internal perturbations.

Patients undergoing rehabilitation form a select group of patients with moderately-severe strokes and significant hemiparesis, other impairments, or environmental factors that require comprehensive rehabilitation (Garraway, Akhtar, Smith, & Smith, 1981). Little is known about whether individuals with mild strokes demonstrate physiological impairments in standing balance or whether their postural control changes over time. Garland et al. (2007) investigated changes in functional balance and muscle activation patterns during arm raise perturbations in individuals with mild or moderately-severe strokes at one month and again at three months after stroke. Subjects in the moderately-severe group increased the paretic hamstrings EMG burst area while subjects in the mild group increased the paretic hamstring EMG burst slope at three months poststroke, with neither group demonstrating change in the latency. Thus, the central nervous system appears to increase the effectiveness of muscle activation in a variety of ways over time and can modulate these EMG burst parameters independently of each other.

To illustrate the impairments evident after mild stroke (Garland et al. 2007), Figure 5 depicts a representative 28 year old subject with a mild stroke (right panel, Berg Balance Score of 56/56, Chedoke McMaster Stroke Assessment score of 7/7 in the foot and leg) and an age- and sex-matched healthy subject (left panel) performing the unilateral arm raise task. Despite this high level of functional balance, the subject poststroke is moving with an arm acceleration of approximately 50% of maximal acceleration of the healthy subject. Although the nonparetic hamstrings activation is similar between the two subjects, the paretic hamstring burst has lower amplitude in the individual with a mild stroke. Comparing the 50% acceleration with the 100% acceleration (dotted line) in the healthy subject, the EMG amplitude scales with the magnitude of the perturbation but the timing of the onset of the EMG bursts does not change significantly, as has been shown previously by Lee, Buchanan, & Rogers (1987) and Mochizuki, Ivanova, & Garland (2004). Therefore, subjects with a mild stroke, who score maximally on clinical scales of functional performance, can still demonstrate impairments in the muscle activation patterns associated with postural control.

The arm raise postural perturbation has the disadvantage that the perturbation cannot be standardized. Ariun and Latash (1995; 1996) developed a self-triggered perturbation task in which subjects drop a standard load held in the outstretched arm. Using this task, Slijper et al. (2002) examined 10 subjects, on average 220
weeks poststroke, with the load dropped from the front or the side. They showed a decrease in the magnitude of the anticipatory postural adjustment (APA) on the paretic side in persons with stroke as well as a reduced ability to modulate the APAs. Whereas healthy subjects modified the APA magnitude with the different load drop positions, the subjects following stroke displayed a smaller change in APA magnitude in the paretic rectus femoris, biceps femoris and erector spinae. Interestingly, there was no difference in the COP excursion between controls and subjects with hemiparesis, suggesting that individuals may have developed a compensatory strategy of redistributing their weight to the nonparetic side, as seen in individuals following moderately-severe stroke (de Haart, Geurts, Huidekoper, Fasotti, & van Limbeek, 2004; Eng & Chu, 2002; Garland et al., 2007).

Figure 5 — Responses to a unilateral arm perturbation in a healthy subject (left) and a subject following mild stroke (right). Top to bottom, the traces are right quadriceps (QUAD), hamstrings (HAM), tibialis anterior (TIB), soleus (SOL), left QUAD, HAM, TIB and SOL. The dotted lines represent the healthy subject data when moving as fast as possible, whereas the solid lines are for 50% of maximal acceleration, which approximates the speed of the arm perturbation in the subject poststroke. All traces are averages of 10 trials aligned to the beginning of arm acceleration (vertical line). Note the reduction in amplitude of the EMG bursts on the paretic side.
Figure 6 — Anticipatory response of hamstrings during a load drop task in a healthy subject (left) and a subject following stroke (middle and right) before and after exercise retraining. The healthy subject demonstrates a reduction of activity in both hamstrings (left, gray shaded; right, black line) in advance of the load release to minimize the backward sway. Hamstrings activity for a subject following stroke is represented before (gray shaded) and after (black line) exercise for both nonparetic (middle) and paretic (right) sides. The nonparetic hamstrings demonstrated an anticipatory EMG reduction that was similar to healthy subjects. The anticipatory EMG reduction was diminished in the paretic leg before exercise but was more evident after 30 min of exercise. All traces are averages of 10 trials aligned to the load drop release (vertical line).
We used this load drop task to determine if exercise retraining, that incorporated functional movements (fast squats and steps) designed to promote the speed of muscle activation, would result in improved postural responses. When the load is held in front, release of the load results in a backward sway. Healthy subjects and the nonparetic leg of individuals with stroke demonstrate a reduction of hamstrings activity in advance of the release to minimize the backward sway (Figure 6). Although this anticipatory EMG reduction was diminished in the paretic leg before exercise, subjects were more effective in shutting down unwanted EMG activity after 30 min of exercise (unpublished observations). This exercise protocol also resulted in an increase in the EMG burst slope during an arm raise perturbation task (Gray et al., 2008).

Similar to external perturbations, if the stroke results in muscle activations that are delayed, of low amplitude or incoordinated during internal perturbations tasks, the shift in the COM will be larger, especially if the anticipatory nature of the programmed muscle activation patterns is disrupted. Disruption of the anticipatory muscle activation would require individuals with stroke to rely on increasing the amplitude or slope of the muscle bursts through increased motor unit recruitment and firing rate to increase postural stability. However, modifying the onset of muscle activation through earlier activation of paretic and nonparetic muscles would be a possible strategy for postural recovery for those with reduced numbers of motor units or limits to the firing rate of motor units. The ability of the central nervous system to use a variety of mechanisms to promote postural stability is important given the inherent heterogeneity in stroke impairments across patients.

**Conclusion**

Balance is an essential part of daily living. Whether the perturbations require feedback or feedforward postural adjustments, there is impairment to the timing, magnitude and sequencing of muscle activation following stroke. The muscle activation pattern is dependent on the extent of motor control impairment and the strategies used by the stroke patients to compensate for the impairments. The central nervous system uses a variety of mechanisms to improve muscle activation patterns needed for postural responses following stroke.

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**References**


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