Modulation of Ankle Muscle Postural Reflexes in Stroke: Influence of Weight-bearing Load

Daniel S. Marigold^{*,§}, Janice J. Eng^{*,†,§}, and J. Timothy Inglis^{*,‡}

^{*}Graduate Program in Neuroscience, University of British Columbia

[†]Department of Physical Therapy, University of British Columbia

[‡]School of Human Kinetics, University of British Columbia

[§]Rehab Research Lab, GF Strong Rehab Centre, Vancouver, B.C., Canada

Abstract

Objective—Given the known sensorimotor deficits and asymmetric weight-bearing posture in stroke, the aim of this study was to determine whether stroke affects the modulation of standing postural reflexes with varying weight-bearing load.

Methods—Ten individuals with chronic stroke and 10 healthy older adult controls were exposed to unexpected forward and backward platform translations while standing. Three different stance conditions were imposed: increased weight-bearing load, decreased weight-bearing load, and self-selected stance. Surface EMG from bilateral ankle dorsiflexors (tibialis anterior) and extensors (gastrocnemius) were recorded and the magnitude of background muscle activity (prior to the platform translation) and postural reflex onset latency and magnitude (75 ms following reflex onset) were determined.

Results—Load modulation of ankle extensors was found in controls and individuals with stroke. Although controls demonstrated modulation of ankle dorsiflexors to different loads, individuals with stroke did not show this modulation. Further, load did not change the onset latency of postural reflexes of the individuals with stroke.

Conclusion—The delayed paretic muscle onset latencies in conjunction with impaired modulation of ankle dorsiflexor postural reflexes may contribute to the instability and frequent falls observed among individuals with stroke.

Significance—The results provide some insight into standing postural reflexes following stroke.

Keywords

postural control; reflex; weight-bearing; cerebrovascular accident

1. Introduction

The incidence of falls for community-dwelling individuals with stroke has been reported to be as high as 73% (Forster and Young, 1995). Thus, an understanding of postural control in stroke is of importance to facilitate the reduction of falls in this population. Reactive postural control entails a response, mediated by postural muscle reflexes, to a sudden unexpected perturbation applied to the body and relies on the central nervous system's (CNS) ability to interpret afferent information to ensure that the centre of mass (COM) stays within the base of support. Proprioceptive afferents from extensor muscles in the legs and exteroceptive afferents from mechanoreceptors in the foot are among the available inputs to the CNS to modulate standing or locomotor postural reflexes (Duysens et al., 2000). It has been suggested that these afferent inputs can provide information regarding load, and thus, have been referred to as load receptors and are particularly useful in signalling COM position (Dietz et al., 1992). Afferent input regarding load may interact with vestibular processing for postural control (Marsden et al. 2003) and vestibular input may be important for modulating the magnitude of postural reflexes, particularly for the later recovery responses following surface translations (Inglis et al. 1995).

Load receptor input for the regulation of stance in humans has been demonstrated in studies which found an increase in the magnitude of postural reflexes with increasing body load (via lead vests) in response to a translation of a platform upon which participants were standing under water (Dietz et al., 1989a; Horstmann and Dietz, 1990). Furthermore, greater load applied to the body during standing platform translations on land was associated with an increase in the magnitude of extensor muscle postural reflexes in healthy individuals (Dietz et al., 1992). It is believed that Ib afferents from golgi tendon organs (GTOs) provide load information to the spinal cord (Dietz et al., 1992; Duysens et al., 2000). In addition to Ib afferents from GTOs, cutaneous mechanoreceptors in the sole of the foot are in an optimal location to sense limb load and have been recently shown to be important in standing balance (Kavounoudias et al., 2001).

Stroke results in damage of neural tissues in the brain and can greatly affect supraspinal centres and their pathways responsible for postural control. Studies on postural reflexes in stroke have demonstrated delayed muscle onset latencies and abnormal recovery strategies in response to external perturbations while standing (Berger et al., 1988; Dietz and Berger, 1984; Di Fabio, 1987; Di Fabio et al., 1986). Understanding the postural responses under varying weight-bearing load is of interest since individuals with stroke tend to bear greater weight on the non-paretic limb (Eng and Chu, 2002). Whether load-dependent modulation is present following stroke is unknown. Therefore, the objective of this study was to determine whether stroke affects the modulation of postural reflexes with weight-bearing load. Assessing the contribution of postural reflexes from weight-bearing load will quantify the extent that afferent information from the lower extremities can be utilized for modulation of postural reflexes in stroke and will determine whether the asymmetrical posture in individuals with stroke (i.e. reduced loading on the paretic limb) contributes to these altered reflexes.

2. Method

2.1 Participants

Ten individuals with hemiparesis (4 right and 6 left) due to stroke and ten healthy older adult controls were recruited from the community. Participant characteristics are described in Table 1. Functional balance was assessed using the Berg Balance Scale, which is a 14-item scale involving tasks including turning, stepping, and transferring (Berg et al., 1989). The inclusion criteria for the individuals with stroke were: (1) over 50 years of age, (2) only one stroke, (3) at least one year post-stroke, (4) able to stand independently for at least 5 minutes without an assistive device, and (5) able to follow two-step commands. Persons with musculoskeletal or neurological disorders in addition to their stroke were excluded. Healthy older adults with musculoskeletal or neurological disorders were also excluded. Limb dominance of the healthy older adults was determined by asking which leg they used to kick a soccer ball. Following university and hospital ethics approval, informed consent was received from all participants prior to their participation.

2.2 Protocol

Participants stood on two force plates (Bertec Corp.), one limb on each force plate, embedded in a platform. Participants wore a full-body harness that was attached to a beam in the ceiling via a dynamic rock-climbing rope to prevent the occurrence of a fall and at least one spotter was present. The harness was adjusted so that it did not take any weight of the person unless they fell. Forward or backward translations of the platform were applied during three stance conditions: increased weight-bearing load (Increased Load), decreased weight-bearing load (Decreased Load), and self-selected stance (Neutral). Participants were instructed to maintain their normal standing posture (Neutral) or shift their weight onto either the right or left leg (depending on the condition) such that both limbs were always in contact with the platform, which would increase loading in one limb and decrease load on the adjacent limb. For the Increased or Decreased Load conditions, the experimenter visually monitored the force plate vertical forces (in Newtons) in order to ensure that participants maintained the appropriate amount of weight-bearing load (target of approximately 70% weight-bearing on one limb). The experimenter provided verbal cueing to the participant so that they maintained the appropriate level of load on each limb prior to triggering the platform translation. The weight-bearing load was then determined by averaging the vertical forces from each force plate for one second prior to platform onset for each trial. Subsequently, this value was converted to percent body weight to show the actual loading achieved for each standing condition across participants (see Table 2).

A total of 15 backward and 15 forward platform translations (8 cm displacement, 30 cm/s velocity, and 300 cm/s² acceleration) were induced in three blocks (Increased Load, Decreased Load, and Neutral) of 5 trials separated by 15–30 second intervals. Participants were told that the platform could move at any time prior to triggering the perturbation but the onset and direction of translation were unexpected in nature. The blocks of trials were randomly ordered for both platform directions and weight-bearing load conditions.

Surface electromyography (EMG) (Bortec Electronics) were collected from bilateral tibialis anterior (TA) and the medial head of gastrocnemius (MG) using self-adhesive electrodes (Ag/AgCl) (Kendall Meditrace), which were placed 2 cm apart and longitudinally on the muscle bellies. The signal was band-pass filtered (10–1000 Hz) and sampled at 600 Hz for 6 seconds (2 seconds prior to platform movement and 4 seconds after) along with force plate data. The common mode rejection of the EMG system was 115 db and input impedance was 10 Gohm. The MG muscle was used for analysis in the backward translations and the TA muscle was used in the forward translations due to their role as primary recovery muscles for these movements (Horak and Nashner, 1986). The MG was selected because previous studies have reported that the reflex responses of this muscle are load-dependent in healthy individuals (Dietz et al. 1989, 1992).

EMG was full-wave rectified and low-pass filtered at 100 Hz (zero lag, second-order, Butterworth algorithm). All EMG data processing used a custom written MATLAB program. The mean EMG signal for one second prior to the onset of platform movement was determined along with the standard deviation. Muscle onset latency, representing a postural reflex, was defined as an increase in muscle activity that exceeded + 2 standard deviations for at least 30 ms and was determined by a combination of visual inspection and computer algorithm via an interactive program.

Background muscle activity was calculated as the area under the curve (trapezoid rule) for one second prior to the onset of the platform movement. The magnitude of muscle activity for 75 ms following the onset of a postural reflex was also calculated as the area under the curve (see Figure 1a). For each individual trial, the magnitude was obtained after removal of each muscle's background activity from the EMG signal (on a single trial basis) so that the reflex magnitude was not masked by any changes in background muscle activity. The 75 ms time window was chosen for two reasons. First, the platform begins to decelerate approximately 250 ms after onset and since postural reflex onset latencies were between 100 and 200 ms generally, the 75 ms time period would not be influenced by the deceleration phase. And second, previous studies on platform translations have used this same procedure (Horak et al. 2001).

For each condition in both directions, trials 2–5 were averaged. The first trial in response to a perturbation has been shown to be clearly different from subsequent ones (Marigold and Patla, 2002). Custom MATLAB software was used to calibrate the force plate data and subsequently, the mean vertical forces were calculated one second prior to platform onset to determine the amount of weight-bearing for each limb for each trial.

2.3 Statistical analysis

Data from the dominant and non-dominant limbs of controls were pooled over each load condition because there was no significant difference between them using paired t-tests ($p > 0.05$). Three separate repeated measures analyses of variance determined the effect of weight-bearing load conditions (Decreased Load, Neutral, Increased Load) on the muscle onset latency of the (1) paretic limb of the stroke group, (2) non-paretic limb of the stroke group, and (3) controls and were followed by linear and quadratic contrasts (which measure linear and quadratic trends between factors). These statistical procedures were also

performed for the dependent variables of the background muscle activity and postural reflex magnitude. An alpha level of 0.05 was used for these procedures. For graphical presentation, both background muscle activity and magnitude of postural reflexes were normalized to the Neutral condition (Figure 2 and 3); however, statistical analyses were performed on the non-normalized data.

Muscle onset latencies were not different between load conditions for TA (see Results) and thus, were collapsed in order to perform independent t-tests between the controls and both the paretic and non-paretic limbs of the individuals with stroke. In contrast, MG onset latencies for controls were different between load conditions (see Results) and thus, separate independent t-tests for each load condition were performed between the controls and both the paretic and non-paretic limbs of the individuals with stroke. Paired t-tests compared paretic and non-paretic limb onset latencies for both the TA and MG (no differences were found between load conditions for this muscle for the individuals with stroke and subsequently were collapsed for this test) muscles. For the multiple t-tests a more stringent alpha level of 0.01 was used.

3. Results

Three individuals with stroke fell (i.e. were caught by the rope and harness system or required the assistance of the spotter) during the platform translations, of which all falls occurred during the forward translations (i.e. backward induced sway that required TA activity). No control participants experienced a fall.

In response to both forward and backward translations individuals with stroke utilized a compensatory step approximately 25% of the time (predominantly with the non-paretic limb). Controls stepped approximately 20% of the time in response to backward translations and 50% of the time in response to forward translations (with no preference for either limb).

Table 2 shows the amount of weight-bearing on the paretic and non-dominant limb for the individuals with stroke and controls, respectively. In the Neutral condition the individuals with stroke had approximately 10% greater weight-bearing on their non-paretic limb while controls were nearly symmetrical. Although trunk control is often impaired following stroke (Dickstein et al. 2004; Karatas et al. 2004), we observed no noticeable differences in the way the individuals with stroke shifted their body to adjust limb load compared to our controls.

In the individuals with stroke, the paretic limb TA and MG were significantly delayed up to 15 ms ($p = 0.009$) and 26 ms ($p < 0.0001$), respectively, compared to the non-paretic TA and MG. Only the paretic TA was different than the TA of controls ($P < 0.0001$) and the paretic MG was different than the MG of controls for the Neutral ($p = 0.002$) and Increased Load ($p = 0.01$) conditions (Table 3).

Qualitative observations during testing suggested that both the individuals with stroke and the healthy older adults utilized predominantly an ankle strategy to recovery from the unexpected surface translations.

3.1 The effect of weight-bearing load following forward surface translations

After removal of background activity, the TA postural reflex magnitude was greater as weight-bearing load increased in the control group ($F_{2,32} = 5.25$, $p = 0.01$; linear contrast, $p = 0.005$) (Figure 2A); however, TA changes were small (i.e. compared with the change in the extensor muscles; see Figure 3A). Non-normalized values for the TA postural reflex magnitude for the control group in the Decreased Load, Neutral, and Increased Load conditions were $18.65 \pm 8.46 \mu\text{V}\cdot\text{s}$, $22.99 \pm 5.97 \mu\text{V}\cdot\text{s}$, and $23.60 \pm 8.35 \mu\text{V}\cdot\text{s}$, respectively (Figure 2A). In contrast, there was no difference in magnitude of TA postural reflexes among the three load conditions for both the paretic ($F_{2,18} = 1.51$, $p = 0.25$) and non-paretic ($F_{2,18} = 0.67$, $p = 0.52$) limbs of the individuals with stroke (Figure 2A for normalized data). Non-normalized values for the TA postural reflex magnitude in the Decreased Load, Neutral, and Increased Load conditions for the paretic limb of the individuals with stroke were $29.79 \pm 15.67 \mu\text{V}\cdot\text{s}$, $30.38 \pm 16.54 \mu\text{V}\cdot\text{s}$, and $35.28 \pm 22.40 \mu\text{V}\cdot\text{s}$, respectively, while for the non-paretic limb were $49.32 \pm 21.90 \mu\text{V}\cdot\text{s}$, $38.55 \pm 19.00 \mu\text{V}\cdot\text{s}$, and $42.11 \pm 18.15 \mu\text{V}\cdot\text{s}$, respectively (see Figure 2A for normalized data).

The TA background muscle activity produced similar findings to the magnitude of the postural reflexes (Figure 2B). In controls, background muscle activity increased as weight-bearing load increased ($F_{2,32} = 7.75$, $p = 0.002$; linear contrast, $p = 0.0004$). Non-normalized values for the TA background muscle activity for the control group in the Decreased Load, Neutral, and Increased Load conditions were $6.66 \pm 2.92 \mu\text{V}\cdot\text{s}$, $8.67 \pm 5.10 \mu\text{V}\cdot\text{s}$, and $11.09 \pm 5.96 \mu\text{V}\cdot\text{s}$, respectively. However, there were no differences in background muscle activity between load conditions for the paretic ($F_{2,18} = 1.07$, $p = 0.36$) and non-paretic ($F_{2,18} = 3.14$, $p = 0.07$) limbs of the individuals with stroke (Figure 2B). Non-normalized values for the TA background muscle activity in the Decreased Load, Neutral, and Increased Load conditions for the paretic limb of the individuals with stroke were $14.48 \pm 7.69 \mu\text{V}\cdot\text{s}$, $14.40 \pm 10.47 \mu\text{V}\cdot\text{s}$, and $18.87 \pm 15.48 \mu\text{V}\cdot\text{s}$, respectively, while for the non-paretic limb were $34.55 \pm 19.02 \mu\text{V}\cdot\text{s}$, $15.39 \pm 10.60 \mu\text{V}\cdot\text{s}$, and $23.94 \pm 15.02 \mu\text{V}\cdot\text{s}$, respectively.

There was no difference in onset latency (means less than 5 ms apart) among the three weight-bearing conditions for the TA in the stroke (Paretic limb: $F_{2,18} = 0.15$, $p = 0.87$; non-paretic limb: $F_{2,18} = 0.78$, $p = 0.47$) and control ($F_{2,32} = 0.34$, $p = 0.71$) groups during the forward platform translations (Table 3).

3.2 The effect of weight-bearing load following backward surface translations

In response to the backward platform translations, the magnitude of MG postural reflexes (i.e. after removal of background activity) was different among the three load conditions within the controls ($F_{2,32} = 35$, $p < 0.0001$) and within both the paretic ($F_{2,18} = 6.14$, $p = 0.009$) and non-paretic ($F_{2,18} = 3.78$, $p = 0.04$) limbs of the individuals with stroke (Figure 3A). The linear contrast on the three loading conditions (indicative of a linear modulation of load), was significant for controls ($p < 0.0001$) and both limbs (paretic: $p = 0.003$; non-paretic: $p = 0.01$) of the individuals with stroke. This was reflected by a greater magnitude postural reflex as weight-bearing load increased (Figure 3A). Figure 1 also shows this trend for one control (Figure 1B) participant and one individual with stroke (paretic limb, Figure 1C; non-paretic limb, Figure 1D). Non-normalized values for the MG postural reflex

magnitude for the control group in the Decreased Load, Neutral, and Increased Load conditions were $5.81 \pm 3.59 \mu\text{V}\cdot\text{s}$, $11.04 \pm 6.71 \mu\text{V}\cdot\text{s}$, and $14.10 \pm 7.86 \mu\text{V}\cdot\text{s}$, respectively. Non-normalized values for the MG postural reflex magnitude in the Decreased Load, Neutral, and Increased Load conditions for the paretic limb of the individuals with stroke were $16.20 \pm 10.16 \mu\text{V}\cdot\text{s}$, $21.84 \pm 11.37 \mu\text{V}\cdot\text{s}$, and $28.35 \pm 19.52 \mu\text{V}\cdot\text{s}$, respectively, while for the non-paretic limb were $25.99 \pm 11.12 \mu\text{V}\cdot\text{s}$, $36.86 \pm 20.81 \mu\text{V}\cdot\text{s}$, and $46.67 \pm 32.65 \mu\text{V}\cdot\text{s}$, respectively.

Similar to the postural reflex magnitude, the background muscle activity of the MG was different among the three load conditions (Figure 3B) within both individuals with stroke (paretic limb: $F_{2,18} = 4.37$, $p = 0.03$; non-paretic limb: $F_{2,18} = 3.96$, $p = 0.04$) and controls ($F_{2,32} = 14.02$, $p < 0.0001$). As weight-bearing load increased, so did the background muscle activity for both controls and individuals with stroke as evident from the linear contrasts (controls: $p < 0.0001$; paretic limb: $p = 0.009$; non-paretic limb: $p = 0.01$). Non-normalized values for the MG background muscle activity for the control group in the Decreased Load, Neutral, and Increased Load conditions were $9.38 \pm 8.95 \mu\text{V}\cdot\text{s}$, $14.85 \pm 11.44 \mu\text{V}\cdot\text{s}$, and $18.90 \pm 12.85 \mu\text{V}\cdot\text{s}$, respectively. Non-normalized values for the MG background muscle activity in the Decreased Load, Neutral, and Increased Load conditions for the paretic limb of the individuals with stroke were $12.14 \pm 8.37 \mu\text{V}\cdot\text{s}$, $16.28 \pm 8.71 \mu\text{V}\cdot\text{s}$, and $22.24 \pm 16.35 \mu\text{V}\cdot\text{s}$, respectively, while for the non-paretic limb were $15.99 \pm 9.23 \mu\text{V}\cdot\text{s}$, $19.65 \pm 9.59 \mu\text{V}\cdot\text{s}$, and $26.92 \pm 16.53 \mu\text{V}\cdot\text{s}$, respectively.

In response to the backward platform translations, the controls demonstrated differences in MG muscle onset latency among the three weight-bearing conditions ($F_{2,32} = 3.38$, $p = 0.05$). MG postural reflexes were faster as weight-bearing load increased (linear contrast, $p = 0.01$) with the Increased Load condition approximately 13 ms faster than the Decreased Load condition (Table 3). In contrast, no differences in MG muscle onset latency among the weight-bearing conditions were found for the individuals with stroke for either the paretic ($F_{2,18} = 0.19$, $p = 0.83$) or non-paretic ($F_{2,18} = 0.70$, $p = 0.51$) limb.

4. Discussion

4.1 Altered supraspinal control due to stroke affects timing of postural reflexes independent of load

Regardless of the subject group (i.e. controls or stroke), the timing of the observed postural reflexes in response to platform translations suggests long-latency reflexes that receive supraspinal input and may utilize a transcortical loop (Nielsen et al., 1997). Although others (Berger et al., 1988; Dietz and Berger, 1984; Di Fabio, 1987; Di Fabio et al., 1986) have reported slower postural reflex onset latencies on the paretic side, it was not possible to determine if this was simply due to the reduced load on the paretic limb since load was not monitored during the perturbation. Our finding that changes in weight-bearing load do not change postural reflex muscle onset latencies in stroke suggests that the typical asymmetric stance adopted by these individuals does not explain the delay in muscle onset latencies observed following an external perturbation. Thus, deficits in the latency of paretic lower limb muscles can be attributed to the loss of supraspinal control, inappropriate modulation of these reflexes from various descending inputs, and/or alterations in muscle properties

resulting from the stroke. The delayed postural reflex onset latencies could suggest slower motor conduction velocities to paretic leg muscles compared to non-paretic motor conduction velocities (Cruz-Martinez, 1983). This delay in paretic lower limb muscle onset latencies may increase the likelihood of a fall. In fact, paretic TA postural reflex onset latency was slower in those who fell during the forward platform translations ($n = 3$, 139.2 ± 19.6 ms) compared to those who did not fall ($n = 7$, 116.9 ± 19.6 ms) when all three load conditions were pooled together.

Interestingly, MG muscle onset latency demonstrated load-dependent modulation in that postural reflexes were elicited faster when limb load was increased in the healthy, but not stroke participants. However, the functional consequences of the 13 ms faster response in controls from the Decreased to Increased Load conditions needs further exploration.

4.2 Modulation of ankle muscle postural reflex magnitude following stroke

Following stroke, individuals demonstrated modulation of the magnitude of ankle extensor muscle postural reflexes (after removal of the background muscle activity) to varying levels of weight-bearing load. Dietz and colleagues (Dietz et al., 1989b; Dietz et al., 1992; Horstmann and Dietz, 1990) have demonstrated that the magnitude of human postural reflexes of ankle extensors is sensitive to load in healthy individuals. Thus, individuals with stroke appear to maintain the ability to modulate postural reflexes based on afferent input regarding weight-bearing load. Thus, supraspinal input may not be required for integrating load information. For example, weight-bearing load has been shown to modulate lower limb muscle activity in individuals with spinal cord injury (Harkema et al., 1997). This supports the notion that afferent input regarding weight-bearing load is integrated within the spinal cord.

A convergence of sensory afferents from GTOs, muscle spindles, joint receptors, and plantar cutaneous mechanoreceptors likely contributed to the gain modulation of postural reflexes during perturbed standing. Harkema et al. (1997) found that EMG amplitude during treadmill induced stepping in individuals with spinal cord injury was less coupled to muscle length (i.e. muscle spindle input) factors than to peak limb load. Ib afferents from GTOs have been shown to be responsible for increasing muscle activity in response to increased limb load in cats (Duysens and Pearson, 1980; Pearson, 1995; Pearson and Collins, 1993). Subsequently, Dietz et al. (1992) and Duysens et al. (2000) have argued that GTOs signal load in humans as well. In addition, cutaneous afferents from plantar mechanoreceptors in the foot have been shown to be important for postural control (Kavounoudias et al. 2001) and compensatory postural reflexes elicited in response to a forward fall while standing (Do et al., 1990).

The fact that individuals with stroke still utilize load information has important implications for body-weight supported treadmill training in rehabilitation for this population. Application of weights near the centre of mass has resulted in increased ankle extensor burst amplitude during human locomotion (Stephens and Yang, 1999). Stepping in selected spinal cord injured patients can be facilitated through body-weight supported treadmill training, presumably due to the fact that the increased load results in greater activation of ankle

musculature (Harkema et al., 1997). Studies in individuals with stroke utilizing this form of rehabilitation have shown benefits (Hassid et al., 1997; Trueblood, 2001).

For the ankle muscles of the control group and the extensor muscles of the stroke group, the concept of ‘automatic gain compensation’ was demonstrated where the muscle response (after removal of background activity) increased with increasing background activity (Matthews, 1986). This phenomenon may be of benefit during perturbed stance in that a larger reflex could be used to stabilize balance through an ankle strategy that is potentially compromised in asymmetrical posture.

Although the TA does not directly sense load (it is non weight-bearing), the modulation of the magnitude of TA postural reflexes under different loads for the controls was likely from a combination of foot mechanoreceptors, joints receptors, and antagonistic extensor muscle feedback regarding load.

Why was modulation of the ankle dorsiflexor, but not extensor postural reflex magnitude disrupted in individuals with stroke? Individuals with stroke often present clinically with weakness of the paretic ankle dorsiflexors, which may have contributed to the lack of modulation. We did not collect muscle strength for the individuals with stroke in this study, but the interactions of muscle strength and load modulation should be investigated in the future. It is also possible that a decreased number of motor units (Dietz et al., 1986), disturbed motor unit recruitment (Gemperline et al., 1995), and decreased motor unit firing rates (Dietz et al., 1986; Rosenfalck and Andreassen, 1980) contributed to the lack of modulation in the ankle dorsiflexor muscles. Alternatively, the fact that different neural pathways control ankle extensors and flexors may explain the lack of change in TA. For example, Brouwer and Ashby (1992) reported that there are stronger connections of supraspinal motor centres (i.e. corticospinal tract fibres) to the distal leg flexors than the extensors as evident from transcranial magnetic stimulation studies and thus, the corticospinal tract fibres disrupted from stroke could result in greater TA (i.e. ankle dorsiflexor) impairment. Deficits in reticulospinal connections to flexor muscles (Fredericks 1996) may also be involved. However, since extensor postural reflex modulation appears to be intact following stroke, vestibulospinal pathways, which are known to modulate antigravity muscles (Fredericks 1996), may remain viable. Changes in reflexes at the segmental level (i.e. impaired spinal interneuronal circuits, see Dietz and Berger 1984) may also contribute to the delayed postural reflex latencies and/or impaired modulation of the reflex magnitude. Further research needs to address the role of individual descending neural pathways on postural reflex modulation following stroke.

In conclusion, delayed paretic limb muscle onset latencies in conjunction with impaired modulation of dorsiflexor muscle postural reflex magnitude may contribute to the instability and frequent falls observed among individuals with stroke.

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Figure 1.

Postural reflexes evoked during platform translations under different weight-bearing load conditions. (A) Diagrammatic definitions of the onset latency and postural reflex magnitude (hatched is the postural reflex magnitude calculated from a 75 ms area from the onset latency). Typical postural reflex responses under the three load conditions following a backward platform translation of the (B) dominant limb MG of one healthy older adult control, (C) paretic MG, and (D) non-paretic MG for one individual with stroke. Time zero represents the onset of the platform movement.

Magnitude of MG postural reflex in response to backward translations

Magnitude of TA postural reflex in response to forward translations

Figure 2.

(A) Postural reflex magnitude and (B) background muscle activity from TA for the stroke and control groups. Values normalized to the Neutral condition are shown, although statistical analyses were performed on the non-normalized data. Note that background muscle activity was removed prior to calculating the magnitude. Error bars are +1 SD.

Background MG activity prior to backward translations

Background TA activity prior to forward translations

Figure 3.

(A) Postural reflex magnitude and (B) background muscle activity from MG for the stroke and control groups. Values normalized to the Neutral condition are shown, although statistical analyses were performed on the non-normalized data. Note that background muscle activity was removed prior to calculating the magnitude. Error bars are +1 SD.

Table 1

Mean (SD) of participant characteristics.

	Stroke (N = 10)	Controls (N = 10)
Age, yrs	61.3 (8.9)	60.6 (5.5)
Mass, kg	76.2 (17.6)	76.7 (10.2)
Height, cm	166.5 (18.2)	174.6 (10.0)
Stroke Duration, yrs	4.1 (2.9)	N/A
Affected Side	4 Right/6 Left	N/A
Berg Balance, max. 56	44.9 (8.3)	N/A
Type of Stroke	5 Ischemic, 4 Hemorrhagic, 1 unknown	N/A

Table 2

Weight-bearing load in percent body-weight (SD).

Platform Direction	Condition	Control (n = 10)	Stroke (n = 10)
		Non-dominant Limb	Paretic Limb
Backward	Decreased Load	22.9 (5.3)	23.8 (5)
	Neutral	49.0 (1.9)	41.9 (10.4)
	Increased Load	77.3 (8)	61.3 (17.5)
Forward	Decreased Load	23.0 (4.8)	24.0 (6.1)
	Neutral	49.0 (2.4)	40.8 (10.3)
	Increased Load	74.8 (7.3)	61.3 (17.8)

Table 3

Mean (SD) onset latencies (ms) for backward & forward platform translations.

Condition	Stroke		
	Controls (n = 10)	Paretic (n = 10)	Non-paretic (n = 10)
<i>MG onset latency in response to backward translations</i>			
Decreased Load	115.4 (20.7) *	141.1 (25.3)	115.9 (12.4)
Neutral	107.1 (10.1)	139.3 (22.7)	113.5 (20.1)
Increased Load	102.5 (17.1)	136.8 (28.1)	118.9 (21.8)
<i>TA onset latency in response to forward translations</i>			
Decreased Load	105.7 (16.5)	123.6 (22.8)	111.3 (13.2)
Neutral	102.4 (9.7)	122.8 (23.1)	107.8 (13)
Increased Load	101.3 (7.5)	124.5 (22)	109.3 (13.1)

* Significant linear contrast, $p < 0.05$.