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Spinal Motor Neuron Excitability and Balance Control Changes Following Downslope Walking

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By

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Thesis Overview

Downslope walking (DSW) has been proposed as a therapeutic tool for people with Multiple Sclerosis (PwMS) to improve clinical gait measures since DSW mechanically changes gait, muscle activity, and sensory reweighting. However, there are mixed findings when comparing studies of PwMS and neurotypical individuals, which may be due to indirect measures being used to assess balance and gait changes post DSW. Therefore, the primary aim was to determine the mechanistic effects of DSW on static and dynamic balance control using direct, objective measures and to support that these changes occurred in parallel to a decrease in spinal motor neuron (MN) pool excitability, measured by a decreased soleus (SOL) H reflex. The secondary aim was to determine the possible mechanism underlying SOL H reflex depression. The study randomly allocated 30 neurotypical young adults (23±1.4y, 6 males) to either the DSW (-10°) or level walking (0°, LW) condition. Both groups performed a single, 30-minute treadmill walking session and completed pre- and post-testing, which included: 1) 10 static balance control trials consisting of 30 s of quiet standing followed by 3 steps, alternating trials with eyes open and closed; and 2) SOL H reflex recruitment curves and conditioned H reflexes to assess reciprocal inhibition. Kinematic data was collected at 100Hz using an Optotrak collection system (NDI Inc., ON, Canada) and kinetic data was collected at 100Hz using an embedded force plate (AMTI Inc., Watertown, MA, USA) to assess static and dynamic balance control via calculations of centre of mass (COM) and centre of pressure (COP). The H reflex recruitment curves were generated by stimulating the tibial nerve at increasing intensities until a maximal M wave (M_{max}) was reached to provide an estimate of spinal MN pool excitability. Conditioned H reflexes were elicited by stimulating the peroneal nerve 2ms prior to the tibial nerve.

Static balance control measures did not change following DSW. There were no differences in RMS COP displacement (dCOP) and velocity (vCOP), or Margin of Stability (MOS) in AP or ML compared to LW. Conversely, DSW only influenced dynamic balance control measures when the participants' eyes were open. Such that, DSW decreased step length (p= 0.06, f= 0.38) and MOS_{AP} (p= 0.03, f= 0.44), and increased ML COM variability (p= 0.04, f= 0.42). LW had the opposite effect, such that step length and MOS_{AP} increased, and ML COM variability decreased from pre-test to post-test. In addition to these changes in balance control, SOL H reflex decreased following DSW, as indicated by a trend towards a greater decrease in H_{slope}/M_{slope} in the DSW group than the LW group (p= 0.07, d= 0.76) and a decrease in $H_{threshold}/M_{threshold}$ was found in the DSW group but not the LW group (p= 0.04, f= 0.48). There were no differences in conditioned H reflex in the DSW group, thus there was no evidence to support that DSW alters levels of reciprocal inhibition or that changes in reciprocal inhibition contribute to the changes in H reflex.

The changes to dynamic balance control experienced by the young adults in our study provide evidence that DSW may not improve balance control in this population. Additionally, the changes due to DSW were negated with the removal of vision on the task; DSW led to similar effects on dynamic balance control as LW when vision was not available. Therefore, we speculate that removing vision likely balanced the emphasis placed on the remaining two sensory inputs (i.e., proprioceptive and vestibular) to help control balance. Future work is required to determine whether DSW has a similar effect on static and dynamic balance control in PwMS.

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Glossary of Terms and Abbreviations

AP: Anterior - Posterior

ML: Medial - Lateral

COM: Centre of Mass

ML COM: Medial-Lateral Centre of Mass

BOS: Base of Support

COP: Centre of Pressure

dCOP: Centre of Pressure Displacement (RMS)

vCOP: Centre of Pressure Velocity (RMS)

CNS: Central Nervous System

APA: Anticipatory Postural Adjustments

DSW: Downslope Walking

LW: Level Walking

MS: Multiple Sclerosis

RMS: Root-mean-square

MOS: Margin of Stability (measure of variability)

SL: Step length

SW: Step width

MN: Motor neuron

Chapter 1: General Introduction

1.1 Control of Posture

1.1.1 Requirements of Postural Control

Postural control involves controlling the body's position in space, relative to the gravitational vector, in order to maintain an appropriate relationship between the various body segments and between the body and the environment. In addition, the control of body position helps maintain stability, which involves controlling the body's centre of mass (COM) in relation to one's base of support (BOS). The body's COM (x, y, z) is a weighted average of each body segment's COM (x, y, z) (124). The COM is independent of the centre of pressure (COP), which is the location of the vertical ground reaction force and is a weighted average of all the forces over the area in contact with the ground, whether that be one foot or both feet. When both feet are in contact with the ground, the net COP is located somewhere in between them, depending on the relative weight under each foot (124).

The requirements of postural control are dependent on both the task and the environment since various tasks will have different demands for stability and orientation of the body. For instance, sitting and standing have similar projections of the COM between the BOS, but the level of control varies between the two tasks. Standing involves a reduced BOS and thus greater stability demands compared to sitting. More specifically, quiet standing involves standing with feet parallel and all body segments in one line so that the COM is vertically projected between both feet (124). Successful postural control is possible due to an interaction between the CNS and the musculoskeletal system. The CNS has various roles during quiet standing, although this document will only focus on two. First, the CNS plays a crucial role in multisensory integration in order to coordinate incoming sensory information (23, 30, 113), including visual,

proprioceptive, and vestibular inputs. When there is inaccurate or insufficient sensory input by one of the sensory systems, the CNS is able to adapt and reweigh the reliance on the remaining unaltered sensory inputs for orientation (108). This process is termed sensory reweighting and can be elicited experimentally. For example, standing on a compliant surface, such as a foam mat, is an easy way to decrease the reliability of proprioceptive information and produce a reweighting of the sensory inputs in order to maintain postural control (106). The higher the compliance of the foam mat, the greater the instability (i.e. rotations) about the ankle. As a result, the CNS downweighs the incoming proprioceptive information and upweights both visual and vestibular information (106). In the current study, we mechanically changed the angle of the ankle by changing the slope of the walking surface to alter the sensory feedback. Increasing the support surface rotation amplitude is enough to cause sensory reweighting due to the alteration in proprioceptive information (88, 94, 106). Regardless of whether the support surface beneath one leg or both legs undergoes rotation, the weight of proprioceptive information is reduced and other sensory weights are increased (88). When vision is altered, a similar process occurs whereby the incoming visual information is weighted less heavily (75). Thus, completely removing vision would force reliance on (i.e. upregulation of) the other available sensory systems.

Sensory reweighting occurs in conjunction with postural control strategies. Postural control strategies involve selective muscle activation, or synergies, to maintain orientation of the body (51, 82). The CNS coordinates muscle synergies so that simplified movements are performed at an appropriate time and the CNS does not need to control each muscle independently, which frees up available cognitive resources (82, 83). The synergies are able to adapt depending on the sensory input available (vision, vestibular, or somatosensory) to

successfully modify the postural response (83), however this adaptation takes time to occur (59). It is possible that the selective recruitment of muscles within muscle synergies occurs via some inhibitory mechanisms, such as pre-synaptic and/ or reciprocal inhibition (62) between ankle dorsiflexors and plantar flexors (60), since inhibition is responsible for modulating muscle activity in antagonist muscles (8, 70, 84).

Moreover, the muscle synergy hypothesis suggests that the CNS is able to exert control over multiple muscles concurrently to implement the required motor skill (117) or behaviour (69, 120). Bernstein (5) believed that by reducing the degrees of freedom through coordination of multiple muscles, the complexity of movement could be decreased. However, it is important to note that there has been disagreement over the operational definition and resulting hypothesis of muscle synergies in the literature. For example, findings from Soechting and Lacquaniti (111) contradict those from Nashner (82): Nashner indicated that there are 'hip' and 'ankle' synergies to substantially reduce the degrees of freedom and the pattern of activation is more or less fixed, supporting that synergies involve a fixed pattern of activation in regards to both timing and amplitude. On the contrary, Soechting and Lacquaniti concluded that these fixed patterns of activation are not realistic; it is more likely that the activation patterns among different muscles combine in various ways to produce the observed muscle activity, however under this definition there is not necessarily a reduction in the degrees of freedom (111). Nonetheless, the muscle synergy hypothesis is used in the current study to help explain how the CNS controls posture.

1.1.2 Inverted Pendulum Model During Quiet Standing

During quiet standing, the body does not remain completely rigid, which has been established from kinetic and kinematic findings. A guiding model used to explain the constant

motion of the body during quiet standing is the inverted pendulum model (76). The inverted pendulum model assumes that the body above the ankle is rigid, acting as a unit, and this unit moves about the ankle since the majority of a person's body mass is located far from this pivot point, at the head-arms-trunk (HAT) segment (37). The unit involves spatial and temporal coupling of movement to facilitate the HAT rotating about the ankle (37). Further, Winter and colleagues (126) proposed a stiffness model to control the moment about the ankle; they hypothesized that the CNS sets the muscle tone of the plantar and dorsiflexors to control the HAT segment against gravitational forces, which attempt to push the pendulum downward. If the stiffness at the ankle is greater than a person's body weight then the model should persist (126).

The inverted pendulum model justifies that the COP controls the COM by oscillating around it to stabilize the COM around a central position between the two feet (126). Moreover, the model helps explain the dynamic relationship between the COM and COP, which is controlled by the CNS and musculoskeletal system. Specifically, the model relates the COP movement to the COM movement, which is supported if the COP amplitude is larger than COM amplitude and the onset of COP movement is delayed relative to COM movement (126). According to Winter (124, 126), a 5-stage cyclical relationship exists between the COM and COP position as the body sways forward and backwards, with the COM beginning ahead of the COP during forward sway. The CNS interprets this and corrects it by communicating with the musculoskeletal system to increase plantar flexion and shift the COP ahead of the COM, halting forward translation of the COM and forward sway (124, 125). Next, the CNS senses the posterior displacement of the COM and backwards sway, and as a result, decreases plantar flexion and increases dorsiflexion until the COP is now behind the COM. This cycle continues throughout quiet standing such that the COM and COP location are in constant fluctuation in relation to each

other about the ankle and correspondingly, the activity of the plantar and dorsiflexors regulate the net ankle moment in the sagittal plane. The COP directs the COM, such that as the COP moves posteriorly, the COM moves anteriorly. This dynamic relationship holds up when the COP moves anteriorly causing the COM to move posteriorly. Additionally, the COP oscillates between a more posterior and a more anterior position over time.

The inverted pendulum model is the basis for all assumptions made by Hof and colleague's Margin of Stability (MOS) model (47), which is used in this study. The MOS can be used in both static and dynamic stability situations to provide a measure of balance control, which describes the dynamics of body posture to prevent falls (124). The MOS varies from previous spatial stability margins in the sense that it incorporates the extrapolated COM position, which accounts for COM velocity, distance of the COM from the ankle (the point of pivot of the inverted pendulum model), and gravity, which would push the pendulum downwards. It follows that a smaller MOS relates to poorer balance control since there is a smaller distance between the BOS and extrapolated COM position. In this situation there is a higher chance of the COM exceeding the margin of the BOS and increasing fall risk (47).

1.2 Neuromechanics of Locomotion

1.2.1 Control of the Gait Cycle

Phases of Gait

Human gait can be divided into two phases: (a) stance phase (limb is supporting) and (b) swing phase (limb is non-supporting). Considering one limb, the stance phase begins at heel strike and ends at toe off. In contrast, the swing phase begins at toe-off of one foot and ends at heel strike of the same foot. Hence a single step encompasses heel strike of foot one to heel

strike of foot two. The distance between heel strikes is referred to as step length, which is commonly measured when assessing gait since step length is influenced by the amount of somatosensory and visual information available (93). Furthermore, the gait cycle can be characterized by single support (stance phase in one limb and swing phase in the other) and double support (stance phase in both limbs), where the period of double support is the area of overlap in stance phase between the left and right limbs (122). Single and double support are important for stability because the COM lies between the feet during double support, which is a more stable position than single support when the COM lies posterior and medial to the stance heel (125). Increased stability during double support explains why older adults (e.g. 70-74, 75-79, 80-84, 85+ years old) spend more time in double support, decrease their step length, and have slower overall gait speeds compared to older adults of lower age (48).

It follows that as a person's speed increases from walking to running, stance phase duration decreases (e.g. ~ 60% to 30%), which leads to a decrease in stride time and double support time (27). Reducing double support duration assists with increasing speed because double support acts as a braking force and a time for the COM to rest between both feet (125). However, an increase in speed during the walk-to-run transition can also be accomplished by increasing step length and decreasing step frequency (107). These three neuromechanical changes (decreasing stance phase, decrease double support duration, increasing stride length) help achieve the goal of increased speed however they put the body in a more unstable position (6, 48, 65).

Inverted Pendulum Model During Gait

The inverted pendulum model has been extended to explain dynamic stability during locomotion in addition to quiet standing; forward translation of the body depends on a rotation of the body segments about the ankle from heel contact to toe off of the stance phase. During the stance phase, the COM rises to a maximum and then falls forward as it follows the path of an inverted pendulum (13). The body exploits the inverted pendulum model because it is energy efficient; an idealized model would not lose mechanical energy. However, during human gait mechanical energy is lost during the double support phase as soon as heel contact occurs. During double support the leading leg performs negative external work as it contacts the ground, while the trailing leg performs positive external work (21). External work is defined as work performed by external forces to displace a person's COM: negative external work redirects the COM from the current inverted pendulum to the subsequent inverted pendulum and positive external work restores the lost energy (21, 22). More specifically, if assessing locomotion from a sagittal view and assuming the COM moves along a sinusoidal pattern, the external work being performed redirects the COM from its downward trajectory to an upward trajectory (76). However, this process comes at a high metabolic cost (22), since there is an energetic cost associated with redirecting the COM. Furthermore, the stability demands are increased compared to quiet standing and become more challenging when walking because the COM lies outside the BOS for a majority of the gait cycle; the COM only remains within the BOS during single stance (92) as the body translates over the ankle. The MOS is still applicable to explain dynamic stability because the extrapolated COM position can lie in between both feet during double support (47).

Mechanical Changes Inducing Adaptations of Gait

Locomotion can be manipulated to alter both intra and interlimb coordination and investigate the neural adaptations which ensue by having participants walk on a split-belt treadmill. This research has been performed on both quadrupeds (29) and bipeds (20, 79, 98, 99, 121) and both are able to adapt their stance and swing times to maintain a resemblance of the gait cycle while their legs move at different speeds (20). Intralimb parameters such as stance and swing times adjust almost immediately but do not persist once the belts are returned to the same speed (98). Conversely, interlimb parameters such as step length and double support time, take longer to adapt and changes persist beyond 10 minutes of split-belt training, which indicates that the CNS is able to control and adapt intra and interlimb coordination separately (98). The ability for the CNS to react and adapt intra and interlimb coordination, respectively, holds up for stroke patients after only 15 minutes on a split-belt treadmill (99). Furthermore, locomotion can also be manipulated by altering the relationship between sensorimotor information and one's perception of movement (25, 41). This is done by having participants walk forward while on a disk that rotates clockwise, which is termed a podokinetic stimulation. Podokinetic stimulations support plasticity in the neural control of locomotion by remodelling the relationship between perception of trunk displacement, visual, vestibular, and proprioceptive information (41). This manipulation produces an after-effect such that participants walk in a curved trajectory opposite to the rotation direction, whether they are walking forward (25, 41) or backward (25), following removal of the podokinetic stimulation. Therefore, the CNS is capable of adapting to walking forward on a curved trajectory and transferring these adaptations to a different situation (walking over ground).

Another way to manipulate gait is to load the limb segments and thus change the mass characteristics of the given segment while participants walk on a treadmill (127). Neural adaptations to this mechanical change depend on the location of the applied load to the nearest joint. Applied proximally, the local dynamic stability of a joint decreases but applied distally, the local dynamic stability remains constant or increases when compared to unloaded (4). To compensate for these changes and maintain dynamic stability, the nervous system alters muscle activation to increase stiffness at the unstable joints (127). As a result, step length and width varies depending on the location of the load and its respective joint stiffness (127). In sum, manipulating an external factor, whether that be through the use of split-belt treadmills, rotating treadmills, or loading joint segments, provides an understanding of how the CNS adapts locomotion to suit the corresponding manipulation. The current study manipulated the ankle angle throughout locomotion by having participants walk on a continual decline in attempt to provide insight into the plasticity of the CNS in response to the mechanical change. Altering the angle of the ankle provides different proprioceptive feedback than walking over level ground. As such, these changes must be accounted for and incorporated by the CNS with the other senses in order to maintain dynamic stability.

1.2.2 Dynamic Stability

Successful locomotion through the environment requires continuous adaptations, which are instigated by visual and somatosensory information. Vision is involved in online control, such that we are able to proactively adjust our gait characteristics based on the visual information we attain, such as regulating step length and width (90). If vision is not available, we must rely on our other senses to help guide locomotion and maintain balance. Namely, the somatosensory

system, which has an important role in successfully adapting movements for the maintenance of dynamic stability (73, 93). The ability to maintain stability is essential for successful locomotion since the COM is in constant motion relative to the BOS. Thus, control over the relationship between the COM and BOS is crucial. Dynamic stability can be measured various ways however the current study focused on gait and whole-body parameters including step length and width variability, and ML COM variability.

Step length and width variability indicates how stability is maintained throughout locomotion; increased variability is associated with instability of the balance control system since the ability to maintain a constant gait cycle is decreased (45). For example, when young adults walk on a compliant foam surface their step lengths, widths, and step times are more variable compared to walking on a flat surface (73). This is a similar gait pattern to older adults who have high stride-to-stride variability in their gait (45), especially in their initial step length and width, and increased step width variability (80). Older adults have greater variability in their step characteristics compared to young adults because they have difficulty controlling their COM (80). The act of constantly regulating foot placement demonstrates how the balance control system is continually dealing with perturbations whereby increasing step width improves COM control in the ML direction (73) and decreasing step length variability improves COM control in the AP direction (80). If DSW is a destabilizing task than we would expect greater variability in step characteristics.

Poor balance control can also be reflected by ML COM variability, which assesses whole-body movement control. ML COM variability provides a global understanding of how well someone controls their COM in the ML direction as they progress through space. Younger adults have less ML COM variability in comparison to older adults (3), suggesting that poor

balance control can be identified by this measure. In brief, using these measures to quantify dynamic stability will highlight the changes in posture and locomotion following downslope walking and therefore provide further information on how the CNS adapts to the perturbation.

1.3 Spinal Excitability

1.3.1 Hoffman Reflex (H reflex)

The H reflex is a useful tool to study the role of spinal excitability in the control of human movement. The H reflex is the electrical analog to the monosynaptic stretch reflex and provides an estimate of the overall excitability of a spinal motor neuron pool. The H reflex bypasses the muscle spindles due to a direct stimulation of the afferent fibres and a subsequent activation of a portion of the homologous alpha motor neuron pool. Accordingly, the H reflex provides information about the excitability of the neural components (afferent and efferent fibres) of the monosynaptic stretch reflex. The excitability is influenced by both faciliatory and inhibitory input onto the motor neurons as well as the intrinsic excitability of the motor neurons (38). Thus, the amplitude of the H reflex is a reflection of the net effect of the input, such that a decrease in H reflex amplitude relates to an estimated decrease in spinal motor neuron pool excitability (62, 78, 87).

The soleus H reflex is evoked by stimulating the tibial nerve in the popliteal fossa (2, 24, 44, 102). At higher intensities, this stimulation depolarizes both the Ia afferent sensory neurons and the alpha motor neurons. Depolarization of motor axons results in a short-latency mass action potential (M wave), whereas depolarization of Ia afferents results in a longer-latency H reflex (14). The H reflex waveform has a longer latency because the evoked signal must travel along the Ia afferent neurons to the spinal cord and return to the site of recording over the

muscle, whereas the signal causing the M wave only travels from the point of stimulation on the alpha motor neurons to the innervated muscle (Figure 1.1). While the H reflex is a tool to analyze the alpha motor neuron pool activated by the afferent pathway in the mixed nerve at the site of stimulation (and thus a measure of spinal motor neuron excitability), the M wave directly activates motor neurons, bypassing the spinal cord to provide a measure of peripheral transmission (74). In order to understand the activation of the entire motor neuron pool, M_{max} must be attained (87). M_{max} represents a simultaneous discharge of the entire motor neuron pool, which is important because it represents a mass action potential (128), and is elicited at supramaximal intensities.

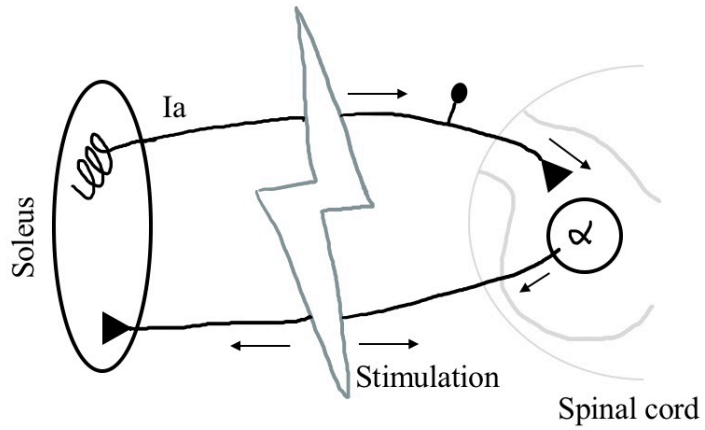

Figure 1.1: H reflex and M wave pathway for the soleus. Stimulation is applied to the mixed nerve, which depolarizes both the Ia afferent and the alpha motor neurons.

The characteristic shape of the H and M curves (Figure 1.2) occur partly due to depolarization thresholds. For example, Ia afferent nerve fibres have a larger diameter and corresponding lower depolarization threshold than the alpha motor neurons (14, 71, 87). Therefore, at low stimulus intensities only an H reflex will be recorded. However, if the stimulus is too low, it may not reach the depolarization threshold and no H reflex will be produced. As such, the H reflex threshold can be determined by slowly increasing the stimulus intensity until

the Ia afferent reaches its depolarization threshold (87). The amplitude of the H reflex will increase with increasing stimulus intensities until it reaches its maximum and then begins to decrease. The decrease in amplitude is a result of the electrical signal in the motor neuron travelling in the antidromic direction, towards the spinal cord, and colliding with the reflexive signal from the afferent fibres going in the orthograde direction, towards the muscle (104). At higher stimulus intensities, the efferent fibre will reach its threshold and an M wave will be recorded in conjunction with an H reflex. Continued administration of increasing stimulus intensities will result in M_{max}. As such, a range of stimulus intensities must be administered beginning from a low intensity and finishing at a supramaximal intensity in order to record both the H reflex and M wave maximum.

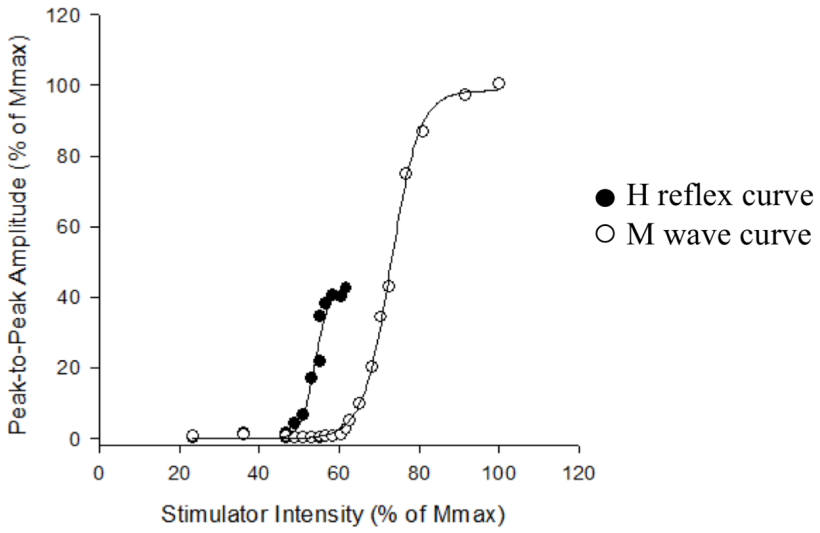

Figure 1.2: Recruitment curve plotted from the peak-to-peak amplitude pilot data of the H reflex and M wave. The peak-to-peak amplitudes of both curves were normalized to the maximum M wave peak-to-peak amplitude (M_{max}) and this was plotted against the normalized stimulus intensities. Each stimulus intensity was normalized to the intensity that elicited M_{max}. Both curves were fitted using a three-parameter sigmoid function.

From a recruitment curve, H reflex and M wave thresholds ($H_{threshold}$, $M_{threshold}$), maximum values (H_{max}, M_{max}), and slopes (H_{slope}, M_{slope}) can be analyzed to provide information

about spinal excitability and peripheral transmission. With each variable, the H reflex is normalized to the M wave to allow for comparison between participants since a limitation that occurs when measuring H reflex is the amplitude variability between participants as a result of skin resistance, amount of subcutaneous fat (87), and electrode placement (123). Normalizing the slopes (H_{slope}/M_{slope}) also reduces the influence of peripheral transmission due to activation history and fatigue (58).

H_{slope}/M_{slope} represents the excitability of a larger portion of the motor neuron pool than is reflected by other H reflex measures such as H_{max} and $H_{threshold}$ and is a standardized representation of spinal excitability (14, 74, 87). Comparing the change in slopes is suggested to be a more sensitive measure than the change in maximum values (H_{max}/M_{max}) (15, 33, 34, 57) since the slopes are less influenced by factors other than the excitability of the motor neuron pool, including postural change and the collision effect (123). The ascending slope also provides information about the reflex gain (62). $H_{threshold}/M_{threshold}$ provides information on the excitability of low threshold units, such that a higher ratio indicates that low threshold units are less excitable, and a lower ratio indicates that low threshold units are more excitable.

1.3.2 Inhibition

A disadvantage to using the H reflex as an estimate of spinal motor neuron pool excitability is the difficulty distinguishing between changes in intrinsic excitability of the alpha motor neurons, changes in inhibitory input, and changes in faciliatory input. Conditioned H reflexes are used to assess the contributions of a segmental input to the motor neuron pool that may result in H reflex modulation. The modulation can occur by inhibition or facilitation, which is induced by the conditioning stimulus depending when it is delivered, and to which nerve it is

delivered. In human models, a conditioning stimulus is applied to a nerve prior to the test stimulus to evoke a conditioned H reflex. For example, if using a conditioned H reflex to assess reciprocal inhibition, the conditioned soleus H reflex is measured by stimulating the peroneal nerve (conditioning stimulus) prior to the tibial nerve (test stimulus) and recording the change in peak-to-peak amplitude of the conditioned versus the test H reflex. Providing a conditioning stimulus assesses the effect of input, whether faciliatory or inhibitory, to the monosynaptic pathway because the conditioning stimulus will either increase or decrease the H reflex amplitude. The focus of this study was on disynaptic reciprocal inhibition because we hypothesized that changes in this pathway may explain the effect of downslope walking (DSW) on the H reflex in young adults (2, 104).

Inhibitory modulation of the H reflex can occur by pre- or post-synaptic mechanisms, both of which can be measured using conditioning stimuli. Pre-synaptic inhibition involves inhibitory interneurons that synapse onto the Ia afferent to reduce the amount of neurotransmitter it releases onto the alpha motor neuron pool (Figure 1.3). The shortest pathway to mediate pre-synaptic inhibition involves 2 interneurons (72), which are modulated by input from afferent and descending inputs through multiple pathways. Some of these pathways involve additional interneurons, some of which can be inhibitory to depress the amount of pre-synaptic inhibition or faciliatory to increase the amount of pre-synaptic inhibition (54). Reciprocal inhibition, a form of post-synaptic inhibition, involves the Ia inhibitory interneuron of an antagonist muscle (e.g. tibialis anterior) that synapses onto the alpha motor neuron pool of an agonist muscle (e.g. soleus) (Figure 1.3). The di-synaptic inhibitory input from the antagonist elicits an inhibitory post-synaptic potential in the agonist. This is important during flexion and extension about a joint because the inhibition of the antagonist muscle is linked to activation of the agonist muscle.

Similar to pre-synaptic inhibition, the Ia inhibitory interneuron can be modulated to increase or decrease the amount of reciprocal inhibition. The modulation can occur by descending drives and recurrent inhibition (54, 72). To facilitate reciprocal inhibition, a conditioning stimulus is applied to the antagonist monosynaptic loop prior to the test stimulus, which is applied to the agonist monosynaptic loop. The level of reciprocal inhibition varies throughout gait similarly to the modification of the H reflex. Stimulating the peroneal nerve during the swing phase leads to increased levels of reciprocal inhibition and a resulting decrease in conditioned soleus H reflex amplitude for up to 15 minutes following passive ground walking on a treadmill (86). Conversely, stimulating during the stance phase leads to decreased levels of reciprocal inhibition and thus an increase in conditioned soleus H reflex amplitude 5 minutes following passive ground walking, but its amplitude returned to baseline values after 30 minutes (86). Stimulating and measuring the H reflex during gait as opposed to afterwards provides slightly different results, with the conditioned H reflex being significantly depressed during late stance (43). Once participants were standing, their conditioned H reflexes were measured again and they were similarly depressed (43). Therefore, in reciprocal inhibition, the peroneal nerve is stimulated to evoke the conditioned H reflex in the soleus, which will have a smaller amplitude compared to the test H reflex. This is due to an increase in neurotransmitter release from the inhibitory interneuron onto the alpha motor neuron pool.

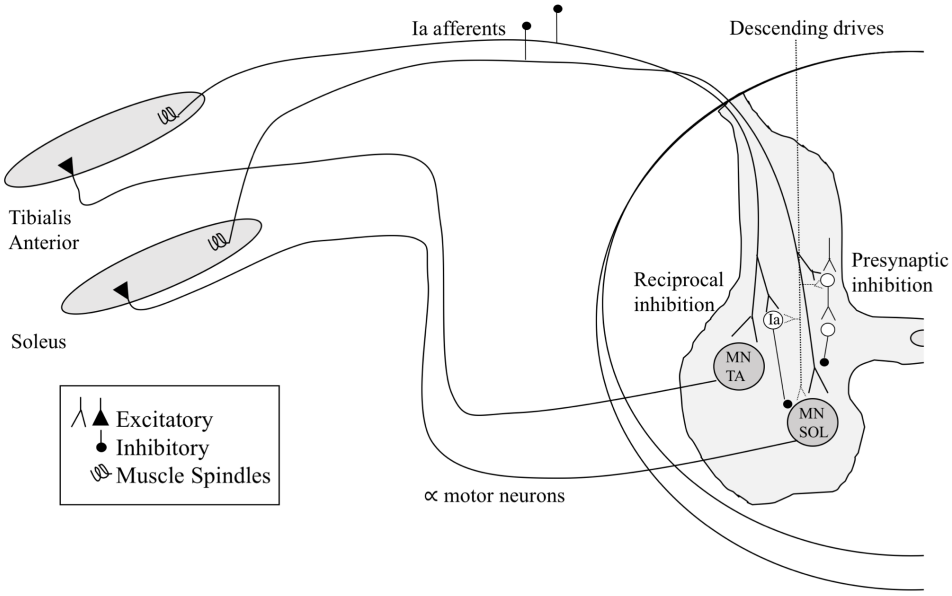

Figure 1.3: Presynaptic inhibition and reciprocal inhibition, a form of post-synaptic inhibition. Presynaptic inhibition can occur through multiple different pathways since the interneurons are modulated by various afferent and descending inputs. Descending drives also influence the Ia interneuron involved in reciprocal inhibition, however the Ia interneuron also receives input from the antagonist muscle (e.g. tibialis anterior) to decrease agonist muscle activity (e.g. soleus).

1.3.3 H reflex and Postural Sway

The H reflex is influenced by the phase of gait in which it is elicited. For example, stance and swing phase require varying stretch of the ankle musculature so the H reflex is phase dependent. During early stance and swing, stretch is ideal, however during mid to late stance, no stretch is ideal. This correlates to the H reflex gain such that there is a low gain during the former and a high gain during the latter (8, 110). For example, the soleus is most active during toe-off (8) and reflex gain is high at this point in order to assist with propulsion.

Similarly, the H reflex is modulated during unperturbed quiet standing, corresponding to the displacement of the COP. As the COP displaces anteriorly the plantar flexors contract, increasing their level of EMG activity, and simultaneously increasing the H reflex (56). This is

important to consider when measuring the H reflex in participants who are standing as opposed to seated because of the influence of COP displacement on the amplitude of the H-reflex. However, the direction of the relationship between postural sway and H reflex amplitude is unclear. When sway increases (increased COP displacement) in young adults, their soleus H reflex amplitude decreases (R^2= -0.5) (116). Similarly, a decreased H reflex amplitude has been correlated to increased variability, expressed as RMS, of COP displacement (53). Thus, poor postural control seems to be related to decreased spinal motor neuron pool excitability. However, these findings were not supported by Koceja, Markus, and Trimble (63) who found no relationship between sway area and H reflex amplitude. Similarly, findings in older adults contradict those with young adults (53, 116), where an increase in sway area was correlated to an increase in H reflex amplitude (R^2= 0.54) (63). Clearly, the degree of COP displacement influences or is influenced by H reflex amplitude however further study is warranted to decipher the relationship and its direction. The relationship between H reflex amplitude and COP displacement was thought to be mediated by pre-synaptic inhibition (118, 119). However, when pre-synaptic inhibition was tested using both heteronymous facilitation (induced by femoral nerve stimulation) and D1 inhibition (long-latency homonymous inhibition induced by peroneal nerve stimulation) (72), the conditioned H reflexes did not vary in amplitude compared to the test H reflexes (56). The authors concluded that post-synaptic inhibition as opposed to pre-synaptic inhibition may be responsible for influencing the relationship between H reflex amplitude and COP displacement.

1.4 Downslope Walking

1.4.1 Mechanisms

Uphill walking differs from downslope walking in various ways, first being the use of different control strategies (67, 68), which is likely the result of eccentric contractions in the lower limbs (2, 68). The soleus, a key muscle involved in plantar flexion (1), is strongly modulated during sloped walking (2, 68). Downslope walking (DSW) depresses the soleus H-reflex (2, 24, 103, 104) with a dose-response relationship such that the duration and degree of slope influence the amount of depression: a smaller downslope angle requires longer sessions to induce spinal plasticity (2). For example, only 10 minutes is needed to significantly depress the soleus H-reflex if walking at -25% (-14°) versus 25 minutes is needed if walking at -15% (-8.5°) (2).

A current ideology for the reason behind H-reflex depression is that the eccentric contractions provide increased afferent feedback from the muscle spindles on any changes in muscle length, which leads to a decrease in H-reflex (2, 68). However, it is not clear which muscles in the lower limbs are contracting eccentrically to produce this effect nor is it clear how increased feedback would depress the H-reflex. If the soleus is subject to an eccentric contraction it is subject to stretch, and the feedback provided by the muscle relating to stretch (information from the muscle spindles) would become altered. Arnold and colleagues (2) make the unsupported conclusion that the increased afferent feedback induces H-reflex depression with no explanation behind the mechanism. Lay and colleagues (68) go one step further and identify that the tibialis anterior contracts eccentrically from heel contact to mid-stance during downslope walking to increase power absorption. Nonetheless, Lay (68) directly relates this to increased muscle spindle afferent feedback and lacks the gap between this mechanical change and H-reflex

depression. Considering these two studies, it would seem as though increased feedback from the tibialis anterior leads to a decreased H reflex of the soleus, however this mechanism is not substantiated. Moreover, another ideology is that the H reflex modulation results from the increased motor complexity of downslope walking compared to level or upslope walking (104). Again, this is more speculation by the researchers since it was not supported with evidence. Therefore, the mechanism behind H-reflex depression remains unclear due to the non-cohesive conclusions among researchers and the lack of evidence.

1.4.2 EMG, Kinetic, and Kinematic Effects

During an acute bout of downslope walking, there are increased muscle forces produced by the soleus, tibialis anterior, quadriceps, and gluteus maximus throughout the gait cycle (1). Increased muscle forces in the soleus and tibialis anterior may explain why soreness is commonly reported in these muscles following downslope walking, however the soreness does not persist and is not of great concern (104). It is not surprising that there is increased muscle force output by the soleus since it absorbs mechanical power from the trunk when walking on a decline (96). As for the quadriceps, in addition to having increased muscle force output (1) by 20-30% compared to baseline values (100), this group of muscles was able to resist fatigue and maintain the same maximum voluntary contraction before and after a single session of downslope walking (40). It is possible that increased muscle activity (31, 68) and longer burst durations (68) are responsible for these changes.

Conversely, downslope walking promotes significantly decreased muscle activity (68) and decreased muscle forces (1) in the gastrocnemius, albeit a longer burst duration, compared to level walking (LW) during the stance phase of gait (68). Decreased muscle activity and a longer

burst duration was also present in the soleus, which indicates that onset of plantar flexor activity occurs earlier than during LW such that during early stance there is likely co-contraction with the tibialis anterior in order to stabilize the ankle (68).

Furthermore, downslope walking also induces changes to kinetic and kinematic parameters. Kinematic changes include increased knee flexion during the stance phase, which corresponds to increased activity in the knee extensor muscles (31, 96), which are being lengthened at this time to absorb energy, and increased hip flexion during mid-stance (67). Further, the maximum angle (61, 109) and the range of motion at the ankle is significantly smaller compared to LW (61, 68), which usually corresponds with significantly less trunk pitch (61). In other words, people do not lean forward while downslope walking, instead they move their COM posteriorly (96) to keep their COM away from the anterior border of their BOS and maintain balance control. People also tend to spend less time in the stance phase while walking on a decline (31, 61). Finally, the kinetic changes from walking on a downslope include increased braking forces and shock absorption during early stance and decreased propulsive forces during toe-off (67). An increased braking force reduces the net external moment and thus whole-body angular momentum (109). This is important when walking on a decline because it reduces the risk of falls; there is greater control of one's angular momentum (109).

1.4.3 Mixed Findings in Young Adults

There is disagreement regarding the benefits of downslope walking in a neurotypical population compared to PwMS. In a group of recreationally active young adults, a single 30-minute downslope walking session led to decreased single limb postural stability (52) even though their H reflex was depressed (14) similar to PwMS (50). However, this study assumed

that downslope walking leads to increased fatigue and muscle damage, which were indicated above as not significant, and the study correlated these negative changes to a decrease in balance ability, which was measured using the Biodex Balance System only. The Biodex Balance System consists of an unstable, tilt-able surface which records the degree of displacement in the AP and ML directions from its starting position. The system then calculates an Overall Stability Index (OSI), where decreased postural stability is indicated by a higher OSI, and calculates the time spent in each range of displacement. Interestingly, when PwMS were tested using the Biodex Balance System, their postural stability had increased following downslope walking (105). The soleus is an important muscle for postural control so it is interesting that the neuromechanical changes evoked by downslope walking would impact postural stability differently between both populations when they experienced similar changes in spinal motor neuron pool excitability recorded at the soleus.

In terms of kinematic parameters, an acute session of downslope walking on a treadmill significantly increased stride frequency (31, 49, 104), however walking on a graded walkway several times did not (67). It is likely that the increased stride frequency corresponds to a decrease in step length (49, 109). Conversely, positive benefits were identified in varsity level athletes who trained on a 4.8% decline twice per week for 6 weeks in addition to off-season strength and conditioning (16). These individuals ran on the decline at 95% of their maximum speed, which translated to a significantly faster 40-yard dash time following the training. It is possible that the benefits of downslope walking were due to increased muscle force output by the quadriceps following 6 weeks of training (100). Unfortunately, there are only a handful of studies examining the neuromechanical changes following a single downslope walking session and of the current published studies, they lack strong methodology and subsequent conclusions.

1.4.4 Changes Following a Downslope Walking Training Intervention

In comparison to uphill walking, downslope walking at a 10% decline for 30 minutes three times a week for 4 weeks significantly increased functional activity, as determined by the 2-minute walk test, timed 25-foot walk test (103, 105), and timed-up-and-go test (105), significantly increased balance control, as determined by postural sway evidenced by decreased COP displacement in the AP and ML directions during the Berg Balance Scale, and significantly increased muscle strength in the quadriceps in people with MS (PwMS) (105). The listed changes persisted until a follow-up 4 weeks later (105), which indicates the potential for long-term spinal plasticity as a result of a downslope walking intervention in PwMS (2). The potential for plasticity is supported by short term decreases in spinal excitability, measured by a depression in H_{max}/M_{max} of the soleus following a single, acute training episode lasting 20 minutes at a 7.5% decline (103).

In addition to PwMS, benefits were also found in hemi-paretic stroke patients (mean age 54) following 30 minutes of downslope walking 5 times per week for 6 weeks in conjunction with conventional physical therapy. Compared to uphill walking, downslope walking better improved their scores on the 6-minute walk test and the 10-meter walk test (11). The improvements on the 6-minute walk test persisted 3 months later, indicating long-term changes in endurance as a result of downslope walking. Carda and colleagues (11) believe their findings are likely a result of the eccentric contractions which occur throughout downslope walking since LW also incorporates eccentric contractions and because stroke patients generally pitch forward, downslope walking helps force trunk extension.

1.5 Objectives of the Thesis

The primary purpose of the current study was two-fold. First, we wanted to determine the mechanistic effects of an acute bout of DSW on static and dynamic balance control using direct, objective measures. It was hypothesized that dynamic, but not static balance control would improve following DSW. In order to answer this question, kinetic and kinematic data were collected. During quiet standing, changes to Margin of Stability (MOS) and COP (RMS displacement or velocity) provided information on the static balance control changes after DSW. Improvements to balance control were indicated by a decrease in MOS (3, 73) or COP displacement or velocity (97) since these are measures of variability. Moreover, the 2 steps following quiet standing provided information on dynamic balance control changes by calculating the MOS of each step, step characteristics (distance and variability), and whole-body motion (ML COM variability). With the exception of average step distances, the measures used to quantify dynamic balance control were also measures of variability. When step-by-step variability decreases, there are less errors in foot placement being made (80) and less need to regulate foot placement with each step (73), indicating improved balance control. Similarly, decreased ML COM variability indicates better balance control since there is less whole-body motion from side-to-side while walking (3). All balance control measures were done with full vision and no vision available, such that we could determine whether the effects of DSW were conditional to the amount of visual information available and if vision was overriding any of the changes to balance control. It was hypothesized that removing vision would negate any recorded changes to balance control present when vision was available, supporting that vision was likely upregulated following DSW.

Second, we wanted to support that any changes to balance control occurred in parallel with changes to spinal motor neuron (MN) pool excitability. It was hypothesized that DSW would lead to a greater decrease in spinal MN pool excitability than LW. This was measured using H reflex recruitment curves, such that a decrease in H reflex amplitude corresponded to an estimated decrease in spinal MN pool excitability (35). The secondary purpose of the study was to determine whether DSW elicits a change in the amount of reciprocal inhibition, as a possible explanation for the changes in spinal MN pool excitability. It was hypothesized that DSW would increase the amount of reciprocal inhibition thus decreasing spinal MN pool excitability. Conditioned H reflexes were used to measure the level of reciprocal inhibition before and after the acute bout of DSW.

Chapter 2:

Spinal Motor Neuron Excitability and Balance Control Changes Following Downslope Walking

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Abstract

Downslope walking (DSW) has been proposed as a rehabilitation tool for people with Multiple Sclerosis (PwMS) although there are mixed findings in young adults (YA) regarding the balance control changes, despite both populations experiencing depressed spinal motor neuron (MN) pool excitability. Our aim was to determine whether YAs could demonstrate improved balance control in conjunction with SOL H reflex depression (estimate of spinal MN excitability) following DSW. We also aimed to determine whether reciprocal inhibition was a potential mechanism for H reflex depression via conditioned SOL H reflexes. Thirty young adults (23±1.4y, 6 males) were assigned to 30-minutes of DSW (-10°) or LW (0°) on a treadmill. Pre- and post-testing included 1) 10 behavioral trials with 30-s quiet standing on a force plate and 3 steps, alternating trials with eyes open (EO) and closed (EC); 2) SOL H reflex recruitment curves, generated by tibial nerve stimulation, and conditioned H reflexes elicited by stimulating the peroneal nerve prior to the tibial nerve. Only dynamic balance control measures changed following DSW and are presented as the change from pre-test values. There was an interaction between group and vision on Margin of Stability in AP (MOS_{AP}), step length (SL), and ML COM variability. With EO, DSW decreased SL (-2.9 ± 4.9%) and MOS_{AP} (-10.3 ± 13.6%), and increased ML COM variability (6.4 ± 8.3%). However, DSW exhibited similar changes to LW when performing the task with EC: minimal change in SL (1.3 ± 8.2%) or MOS_{AP} (0.2 ± 13.7%), and decreased ML COM variability (-15.1 ± 31.9%). Balance changes occurred with SOL H reflex depression (-46.9 ± 15.4%) but there was no increase in reciprocal inhibition. Overall, DSW may not be beneficial to balance control in YA, which opposes findings in PwMS.

Introduction

Neuroplasticity is fundamental in rehabilitation strategies for people with Multiple Sclerosis (PwMS), a chronic autoimmune disease which targets the central nervous system and leads to difficulty with static and dynamic balance control. Physical therapy and exercise offer the possibility to induce neuro-plastic changes, however depending on a person's disease progression, fatigue and thermosensitivity become barriers to exercise. Recently, clinicians sought out a potential solution, downslope walking (DSW), since it can facilitate some of the benefits of exercise without overly increasing one's heart rate. For example, heart rate while DSW at -10° is lower than uphill walking (104) and similar to level walking (LW) (2). Correspondingly, average Ratings of Perceived Exertion (RPE) ranged from "very light" to "light" (2, 104). At these low levels of activity, PwMS still experienced improvements in functional outcome measures such as the 6-minute walk test, timed 25-ft walk test, and Timed Up and Go test (105). These global changes to gait likely stem from the mechanistic changes DSW provokes. Additionally, the elicited changes persist such that the soleus H reflex measured at 10-minutes (2, 50, 104) and 45-minutes (50) post DSW is still depressed compared to pre-DSW values. The soleus H reflex provides an estimate of a person's spinal motor neuron pool excitability, such that a smaller H reflex is indicative of lower levels of excitability (35). PwMS that experience high levels of spasticity have a larger H reflex than those with lower levels of spasticity (112), making DSW a viable protocol that could be used as a rehabilitation tool to decrease spasticity and improve gait.

During DSW there are changes to the kinematic and kinetic parameters of gait and changes to the lower limb muscle activity. Specifically, stance duration decreases (61) while cadence increases, and there is a decrease in step length (49). At the trunk, there is less pitch (61)

and whole-body angular momentum (109), and at the ankle there is decreased plantar flexion (61, 68). Correspondingly, propulsive forces are decreased (67) while braking forces are increased (67, 109). This aligns with decreased muscle activity in the plantar flexor muscles such as the gastrocnemius (68) and soleus (31), and increased muscle activity in the tibialis anterior (68), which is responsible for dorsiflexion. These changes to ankle muscle activations that occur during DSW continue to influence balance control once individuals begin walking on level ground. For example, post DSW there is increased stride frequency (104), increased force output by the quadriceps (100, 105), and an overall improvement in functional activity indicated by clinical tests such as the 6-minute walk test, timed 25-ft walk test, and Timed Up and Go test (105).

However, there are contradictory findings between PwMS and young adults on postural stability changes following DSW even though both populations experience a depression in their soleus H reflex after walking downhill (2, 50, 104). Using a Biodex Balance System before and after DSW, PwMS had improved postural stability (105) while young adults had impaired postural stability (52). It is possible that DSW affects static balance control strategies differently in PwMS than young adults, however neither study examining postural stability changes following DSW supported their results in comparison to changes in H reflex. Additionally, the mechanism underlying soleus H reflex depression in either PwMS or young adults has yet to be identified. It is possible that the discrepancy between populations is due to the indirect measures being used to quantify changes in balance control post DSW, such as the Biodex Balance System (52, 105), the 6-minute walk test, timed 25-ft walk test, and Timed Up and Go test (105). Thus, we currently do not have a supported understanding of how DSW influences balance control since indirect and subjective measures are currently being used in the literature with

predominantly Multiple Sclerosis populations. For these reasons, we aimed to understand the specific changes to balance control elicited by DSW using a neurotypical population before recommending DSW as a rehabilitation tool for PwMS.

Therefore, the first purpose of this study was to objectively determine whether a young adult population could demonstrate improved balance control in conjunction to a decrease in spinal motor neuron pool excitability following an acute bout of DSW. It was believed that dynamic but not static balance control would improve following DSW and that DSW would lead to a greater decrease in soleus H reflex than LW. Moreover, all balance control measures were performed with and without visual information to determine whether the effects of DSW were dependent on the amount of vision available. It was hypothesized that removing vision would negate any changes to balance control recorded when vision was available. The second purpose was to determine whether the decrease in H reflex was the result of a change in reciprocal inhibition levels due to DSW. It was speculated that DSW would increase the amount of reciprocal inhibition, thus decreasing spinal MN pool excitability, which would be recorded as a decrease in soleus H reflex.

Methodology

Participants

Thirty young adults (age: 23 ± 1.4, 6 males) with no history of neurological or musculoskeletal disease, no recent musculoskeletal injuries (within the past 90 days), or history of shin splints (anterior leg pain induced by exercise) participated in the study. Any formally trained ballet dancers or gymnasts were excluded due to their large a range of motion about the ankle and practice of postures which involve sustained co-contraction of the dorsi-and plantar-

flexors to maintain balance. As a result they have smaller H reflex amplitudes, lower levels of reciprocal inhibition, and greater levels of pre-synaptic inhibition (84). Participants self-reported their level of physical activity ($\bar{x}$= 3.8 days, SD= 1.7) and all participants provided written informed consent prior to participation. The experimental procedure was approved by the Research Ethics Board of Wilfrid Laurier University and is in compliance with the Declaration of Helsinki.

Table 1: Participant Characteristics of Downslope and Level Walking Groups

Participant	Sex	Age (Y)	Average Days of PA per Week	Low, Moderate, or High Levels of PA	Frequency of PA on Treadmill	Average RPE on Treadmill	Average Treadmill Speed (m/s)
Downslope Participants							
P1	F	24	3	Moderate	Monthly	7.4	0.67
P2	F	23	3	Moderate	Bi-weekly	11.3	1.07
P7	F	23	5	High	Weekly	9.8	1.16
P14	F	23	5	Moderate	Monthly	9.1	0.80
P15	F	26	7	High	None	9.2	0.67
P16	M	22	4	Moderate	None	9.0	0.72
P17	F	23	7	High	Monthly	10.2	0.98
P18	F	25	3	Moderate	None	9.6	0.63
P19	M	24	5	Moderate	Monthly	7.7	1.03
P20	F	22	3	Low	None	8.8	0.58
P22	F	23	4	Moderate	Monthly	9.0	0.80
P27	M	24	6	High	Weekly	6.9	0.76
P28	F	21	6	High	Weekly	9.2	0.94
P32	F	21	3	Moderate	Bi-Weekly	6.0	0.67
P33	F	22	3	Moderate	None	7.5	0.63
Mean	--	23.07	4.47	--	--	8.7	0.81
SD	--	1.39	1.51	--	--	1.4	0.18
Level Walking Participants							
P3	F	24	4	Moderate	Weekly	7.8	1.03
P4	F	22	5	High	Monthly	6.7	1.39
P5	F	23	3	Moderate	None	7.0	0.98
P8	F	22	2	Moderate	None	7.5	0.80
P10	F	24	2	Moderate	None	9.8	0.98
P11	M	23	0	Moderate	None	8.9	0.89
P12	F	23	3	Moderate	Weekly	7.8	1.30
P21	F	23	3	Moderate	Bi-weekly	10.8	0.94
P23	F	22	0	Low	None	6.6	1.07
P24	F	21	3	Moderate	Weekly	6.0	1.11
P25	M	23	5	High	None	6.6	1.25
P26	F	23	3	Moderate	Monthly	6.4	1.07
P29	F	24	6	High	None	6.0	1.21
P30	F	20	2	Low	Monthly	6.8	0.98
P31	M	26	6	High	Bi-weekly	10.2	0.76
Mean	--	22.87	3.13	--	--	7.7	1.05
SD	--	1.41	1.85	--	--	1.6	0.18

Experimental Design

A randomized control study design was used, in which two groups (DSW and LW) each completed pre-test and post-test assessments of static and dynamic balance control, and measures of H reflex (Figure 2.1). In both the pre-test and post-test assessments, there were 10 trials for the balance control measures (5 with eyes open and 5 with eyes closed). The visual condition of trial 1 was counterbalanced across participants and the visual condition alternated with each trial. In order to test both static and dynamic balance control, participants stood quietly on a force plate for 30-s at which point they initiated gait and took 3 steps at a self-selected pace and step length. To assess the excitability of the soleus motor neuron pool, a soleus H reflex recruitment curve was obtained, followed by a series of conditioned and unconditioned H reflexes to assess the level of reciprocal inhibition. The soleus was selected based on previous findings that it is strongly modulated by DSW (2, 68) and because of its role in postural control. H reflex recordings occurred following behavioural measures based on findings that the changes to soleus H reflex should persist for 10 minutes (50, 104) and up to 45 minutes post DSW (50).

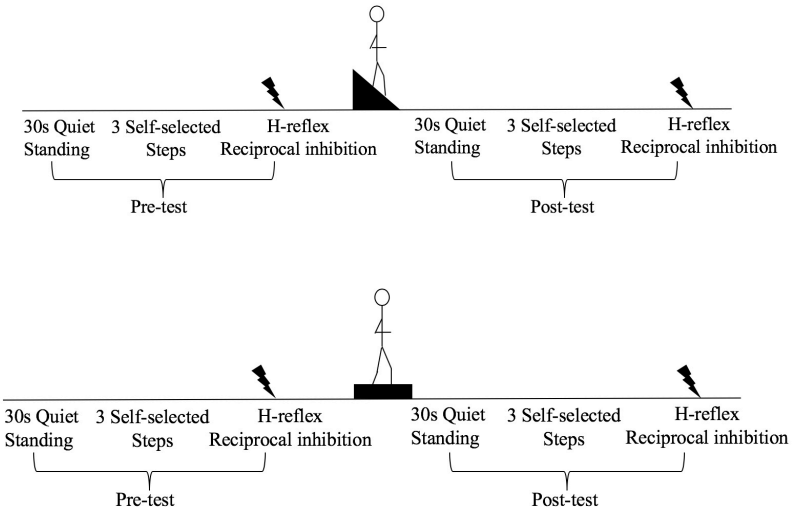

Figure 2.1: Visual depiction of pre- and post-test surrounding a single 30-minute bout of downslope (top panel) or level walking (bottom panel). The pre- and post-test consisted of (a)

quiet standing for 30-sec, (b) 3 self-selected steps with eyes open and eyes closed, and (c) elicitation of H reflex recruitment curve and reciprocal inhibition.

Downslope and Level Walking

Participants were randomly selected to either the DSW or LW group. All participants walked for 30-minutes at a self-selected pace and were allowed to alter their speed to maintain a consistent intensity level (DSW: 0.81 ± 0.18 m/s; LW: 1.05 ± 0.18 m/s). Intensity level was monitored by asking participants for their Rate of Perceived Exertion (RPE) every 5 minutes and an average RPE across the entire 30-minute bout was calculated (DSW: 8.7 ± 1.4; LW: 7.7 ± 1.6). Participants in the DSW group walked on a -10° slope and participants in the LW group walked on a 0° slope. Participants wore their own pair of self-selected running shoes throughout the duration of the testing procedures to maintain consistency in foot and ankle support.

Setup

Static and Dynamic Balance Control

Triaxial ground reaction forces were measured using an embedded strain-gauge force plate (AMTI Inc., Watertown, MA, USA) during quiet standing. Consistency in foot position was maintained between trials by placing a foot tracing over the force plate. The force plate data were digitized at a sampling frequency of 100Hz. Kinematic data was collected with a single Optotrak camera (NDI Inc., Waterloo, ON) positioned at the end of the path such that it continually captured position information from forward-facing markers worn by the participants at 100Hz. The markers consisted of 3 rigid bodies, worn over the xiphoid process and the dorsum of each foot, and each rigid body contained three Infared Emitting Diodes (IREDs) for a total of 9 IREDs. The rigid bodies acted as reference points for digitized (i.e. imaginary marker) points,

which were located on both glenohumeral joints, both anterior superior iliac spines (ASIS), and the heads of both 5th metatarsals for a total of 6 digitized points (Figure 2.2).

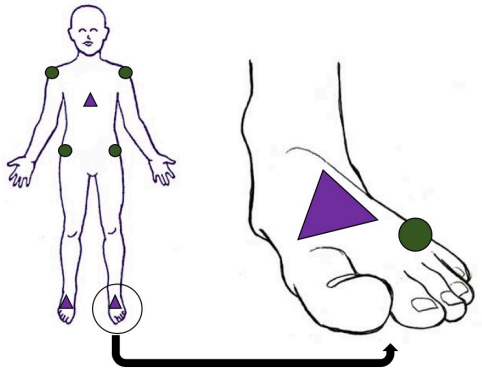

Figure 2.2: Participant marker set-up with 3 rigid bodies (triangles) on xiphoid and dorsum of each foot and 6 digitized points (circles) located at the left and right glenohumeral joints, right and left ASIS, and right and left 5th metatarsal heads.

Soleus H reflex During Standing

Spike2 software (version 7.0, Cambridge Electronics Design Ltd., Cambridge, UK) was used to record and analyze the H reflex recruitment curves. Signal software (version 4.05, Cambridge Electronics Design Ltd., Cambridge, UK) was used to record the conditioned and test H reflexes in the reciprocal inhibition protocol. Participants were outfitted with an active (1.7 x 1.6 cm) and dispersive (3.6 x 4.5 cm) stimulating electrode. The active electrode was placed in the right popliteal fossa over the tibial nerve, whereas the dispersive electrode was placed superior to the patella. The stimuli were delivered to the active electrode using a Digitimer Constant Current Stimulator (model DS7AH), which sent the electrical current anteriorly to the dispersive electrode. A second stimulus was delivered using another Digitmer (model DS7A) to bi-polar electrodes (2 cm interelectrode distance) over the peroneal nerve, inferior to the fibular head. This stimulus delivered pulses for the conditioned H reflex. To ensure the active and dispersive electrodes were in the correct placement, preliminary stimulation was administered to

the tibial and peroneal nerve and the soleus and TA were monitored for a muscle response, respectively.

Two surface electrodes (2 cm interelectrode distance) were placed on the posterior aspect of the leg over the soleus muscle and on the anterior aspect of the leg over the TA. A ground electrode was placed over the lateral malleolus. The skin under those areas was shaved and wiped with 99% isopropyl alcohol to improve the signal-to-noise ratio. The surface EMG signal was amplified 1000x (AMT-8, Bortec Biomedical Ltd., Calgary, CA), digitized (micro1401-3, Cambridge Electronics Design Ltd., UK) at 1000Hz, and high-pass filtered at 10Hz offline.

Participants stood quietly with their feet approximately shoulder width apart during collection of both the soleus H reflex recruitment curve and conditioned H reflex (32, 91, 102, 110, 115). Standing is a more functional position than sitting, the conventional H reflex testing position. During standing, the extensor muscles throughout the body are continuously active and there is a tonic level of background activity in the plantar flexors. Given that the soleus is a postural muscle and one of the main muscles influenced during DSW, an upright standing posture may be more relevant to the changes elicited from DSW if the measures are task dependent. To support the participant while standing, a four-point walker was placed in front of them to rest their hands on.

Procedures

Static and Dynamic Balance Control

Participants stood with both feet approximately shoulder width apart on the force plate and were instructed to remain as still as possible for 30-sec, after which the participants were instructed to walk forward taking the first step with their left foot. All participants took 3 steps

(on average) before reaching the end of the pathway at which point, they were instructed to stop walking. These steps were of self-selected length, width, and speed. Only 4 steps were needed to gain information on variability in foot placement (73, 81). Throughout these trials, participants alternated having their eyes open and closed. During the no visual condition, participants were asked to keep their eyes closed from the start of quiet standing to the end of the 3 steps to identify any changes in sensory reweighting and the influence of vision during both static and dynamic balance control.

Soleus H Reflexes During Standing

Participants were instructed to keep their eyes open and remain as still as possible throughout the duration of the H reflex protocol. The soleus H reflex recruitment curve was elicited by delivering a series of 1000-us square pulses to the tibial nerve at the active electrode. A single stimulus was applied once every 10-sec at increasing stimulus intensities until M_{max} was elicited. The inter-stimulus duration was 10-sec to reduce the risk of post-activation depression, which would have led to H reflex depression because of a reduced quanta released from the recently activated synaptic terminals (78) as opposed to due to DSW.

To verify that the M wave was the true maximum value, 3 supramaximal stimuli were delivered. If the peak-to-peak amplitude of the M wave did not increase with 3 successive increases in stimulus intensity, it was assumed that M_{max} had been reached.

To assess reciprocal inhibition, the soleus H reflex was conditioned by stimulating the peroneal nerve prior to tibial nerve stimulation. The peroneal nerve was stimulated with a single pulse (1000-us duration) at the participant's TA motor threshold and the tibial nerve was stimulated at 15% of their M_{max} (18, 70). The conditioning-to-test interval was 2 milliseconds

(43, 70). Either a conditioned H reflex or test H reflex was elicited every 10-sec for a total of 10 test and 10 conditioned soleus H-reflexes (86). The order of conditioned and test H reflexes was randomized. Participant's TA motor threshold was identified as the lowest stimulus intensity required to elicit responses with an amplitude greater than 100-uV in 50% of stimuli (e.g. 5 out of 10 stimuli) (101).

Data Analysis

Static and Dynamic Balance Control

Static Balance Control: Kinetics and Kinematics

Average COP position was calculated over the first 30-sec in AP and ML directions and was subtracted from the data to remove the position bias. From the unbiased COP position data at each iteration in time (COP_i), the COP displacement (dCOP) was calculated using the root-mean-square (RMS). COP velocity (vCOP) was calculated using the first central difference method of COP_i followed by calculating the RMS. The RMS provides an understanding of the variability in COP control.

$$dCOP = \sqrt{\frac{1}{n}(COP_1^2 + COP_2^2 + \cdots + COP_n^2)}$$

$$COPvel_i = \frac{Unbiased\ COP\ (i+1) - Unbiased\ COP\ (i-1)}{(i/100)}$$

$$vCOP = \sqrt{\frac{1}{n}(COPvel_1^2 + COPvel_2^2 + \cdots + COPvel_n^2)}$$

Where: *i* is each iteration in time, used within the first central difference method to calculate COPvel (mm/s) using COP position data (mm).

Margin of Stability (MOS) was calculated by subtracting the extrapolated COM position (xCOM) from the base of support (BOS) in AP and ML. The standard deviation of the MOS values was calculated over the duration of quiet standing as opposed to the average value (average location of the COM in relation to the BOS), where the average demonstrates how close one is to becoming unstable. MOS variability was used instead because it represents a dispersion in the relationship between the COM and BOS during double support; larger dispersion suggests a larger range which would indicate a smaller average MOS. Subsequently, higher variability indicates poorer COM control by the CNS. Moreover, MOS variability has been indicated in the literature as an important measure of stability (77).

$$MOS = BOS - xCOM$$

$$xCOM = COM + \frac{v\,COM}{\sqrt{\frac{g}{l}}}$$

Where: *vCOM* is the velocity of the COM (mm/s), *g* is gravity (mm/s^2), and *l* is the length from the COM position to the AP or ML position of the ankle (mm).

Dynamic Balance Control: Kinematics

MOS was calculated in AP and ML directions from the start (i.e. heel contact of lead foot) until the end (i.e. toe off of trail foot) of the double support phase when the COM was located within the BOS boundaries of both feet. In total, there were three double support phases for each trial (i.e. from quiet standing to the first step; between the first and second steps; and between the second and third steps) and MOS variability (i.e. standard deviation) was calculated during each double support phase. Average step length and width and their corresponding variabilities were examined over the first 2 steps; participants took 3 to 4 steps however

kinematic markers were only consistently visible on the first 2 steps. Variability was calculated independently for each step, and within each step variability was determined separately for eyes open and eyes closed. Therefore, for each step, the standard deviation of the 5 eyes open trials was calculated separately from the 5 eyes closed trials. Variability in step length and width indicates how well participants are able to control their COM displacement, since minimal variability demonstrates less need to correct foot placement (80). ML COM variability was determined to identify whether DSW influenced whole-body COM control.

Soleus H Reflexes During Standing

H reflex Recruitment Curve

The recruitment curves were constructed using normalized stimulus intensity, H reflex, and M wave data. The peak-to-peak amplitudes of the H reflex and M wave waveforms were normalized to M_{max} and intensity was normalized to the intensity which elicited M_{max}. From the recruitment curve, the slope of the H reflex was recorded in relation to the slope of the M wave (H_{slope}/M_{slope}). The slope of each curve was calculated at 50% between the upper and lower asymptotes by fitting each curve to a 3-parameter sigmoid function using SigmaPlot 14.0. To improve the fit of the curve, the H reflex and the M wave curves were cut off at their maximum values. The sigmoid function used to fit the two curves were as follows:

$$H(s)=\frac{H\ max}{[1+e^{\frac{(s-s_{50})}{b}}]} \qquad M(s)=\frac{M\ max}{[1+e^{\frac{(s-s_{50})}{b}}]}$$

Where: *H(s)* and *M(s)* are the amplitudes of the respective wave at any given stimulus intensity, H_{max} *and* M_{max} are the upper limits of the curve, *b* is the slope parameter, s_{50} is the stimulus intensity at 50% of the maximum value, and *s* is a given stimulus intensity.

Next, the slope at 50% of the curve was estimated using the following equation:

$$\text{Slope}_{50} = \frac{\text{H max}}{4b} \qquad \text{Slope}_{50} = \frac{\text{M max}}{4b}$$

Additionally, H reflex and M wave threshold were calculated by solving for *s* in the sigmoid function equation at 5% of Hmax and 10% of Mmax. H reflex and M wave maximum were calculated directly from the recruitment curve.

$$s = s_{50} - b \ln \frac{H\ max}{y} \qquad s = s_{50} - b \ln \frac{M\ max}{y}$$

Where: *s* is the stimulus intensity, s_{50} is the stimulus intensity at 50% of the maximum value, *y* is the amplitude at 5% of Hmax or 10% Mmax, and *b* is the slope parameter.

Conditioned H reflex

Reciprocal inhibition was measured by comparing the average peak-to-peak amplitude of the conditioned (peroneal and tibial nerve stimulation) soleus H reflex to the unconditioned (tibial nerve stimulation only) H reflex. Specifically, the average peak-to-peak amplitude of the H reflex and M wave from the 10 frames of the unconditioned state (tibial nerve stimulation only) and 10 frames of the conditioned state (peroneal and tibial nerve stimulation) were calculated. The average H reflex peak-to-peak amplitude was normalized to the average M wave peak-to-peak amplitude in both states (H:M). Next, the amplitude of the conditioned H reflex (H:M) was expressed as a percentage of the unconditioned H reflex (H:M) (56, 86). Therefore, a value below 100% indicates a depression in H reflex due to conditioning. The conditioned H:M was compared from pre- to post-test to determine the degree of conditioning that DSW had on the spinal pathway.

Statistical Analysis

Statistical analyses were performed using SPSS (version 23, IBM) and meaningful results were determined based on the *p*-value and effect size. Cohen's F was used to determine effect size where 0.1- 0.24 was considered low, 0.25 - 0.3 was considered medium, and ≥0.4 was considered high. The statistic was considered meaningful if $p < 0.05$ or if any p value between 0.051 and 0.09 had a medium to large effect size (114). All data are presented as mean ± SD. Normality was checked for each variable according to non-significance of Shapiro-Wilk, Levene's test was used to check for homogeneity of variance, and statistical outliers were indicated as any trial value greater than 2SD from the mean. Trial values outside this range were removed from the average.

Static and dynamic balance control measures are presented as a percent change [((post-test – pre-test)/post-test) x100] to clearly identify the change in each dependent measure as a result of the walking angle. All static balance control measures and ML COM variability were examined for the effect of vision (eyes open, eyes closed) across both groups (DSW, LW) so repeated measures mixed ANOVAs with 1 within and 1 between factor were performed. A DSW participant was removed from the COP analysis in ML direction because their average percent changes were greater than 2SD from the group mean. Dynamic balance control measures including MOS during locomotion (AP and ML) and step length and width average and variability were examined for the effect of vision (eyes open, eyes closed) and step number (1, 2) across groups (DSW, LW) using repeated measures mixed ANOVAs with 2 within and 1 between factor.

The standing H reflex measures (H_{slope}/M_{slope}, H_{max}/M_{max}, H_{th}/M_{th}, conditioned H) were compared for the effect of time (pre, post) between groups (DSW, LW) using two-way repeated

measures mixed ANOVAs. In addition, percent change was calculated for each variable following the ANOVA and an independent t-test was used to compare the mean percent change between groups. One participant in the level group was completely removed from the H reflex analysis due to an unmeasurable H reflex in both the pre- and post- test, leaving 29 participants in the analysis (DSW n= 15, LW n= 14). Additionally, any participants with a value in either the pre-test or post-test which was outside 2SD from the mean were removed from their respective ANOVAs. No participants were removed from the H_{max}/M_{max} analysis however 3 participants were removed from H_{slope}/M_{slope} (DSW n= 14, level n= 12) and 2 participants were removed from the H_{th}/M_{th} analysis (DSW n= 14, level n= 13). When percent change was used to compare variables between groups, significant outliers were still removed using the same criteria (H_{slope}/M_{slope} and H_{max}/M_{max}: DSW n=13, LW n=13; H_{th}/M_{th}: DSW n=15, LW n=13). As for the conditioned H reflex, 4 participants were removed from the ANOVA (DSW n= 13, level n= 12) and 2 participants were removed from the t-test (DSW n= 14, LW n= 13).

To determine whether the changes in H reflex could explain the changes in behavioural measures, correlations were run between the variables. The correlation coefficient (r) and coefficient of determination (R^2) were recorded. For all ANOVAs, only main effects and interactions involving the effect of group (DSW or level) are presented.

Results

Behavioural Measures

Static Balance

Measures of static balance control did not change following an acute bout of DSW. For pre- and post- test values refer to Appendix A. There was no interaction effect between vision

and group on MOS_{AP} [F (1, 28) = 0.61, *p*= 0.44, *f*= 0.15] (Figure 2.3a), nor were there any differences between DSW (3.5 ± 28.4%) and LW (15.8 ± 23.4%) [F (1, 28) = 2.38, *p*= 0.13, *f*= 0.29]. MOS_{ML} was similarly not affected by an interaction between vision and group [F (1, 28) = 1.62, *p*= 0.21, *f*= 0.24] (Figure 2.3b) nor by walking condition (DSW: 10.3 ± 40.5%; LW: 22.3 ± 32%) [F (1, 28) = 1.00, *p*= 0.33, *f*= 0.19]. Moreover, there was no interaction effect between group and vision on $dCOP_{AP}$ [F (1, 28) = 0.001, *p*= 0.972, *f*= 0.00] (Figure 2.4a) or between walking conditions [F (1, 28) = 1.37, *p*= 0.25, *f*= 0.22]. In the ML direction ($dCOP_{ML}$), there was also no interaction between group and vision [F (1, 27) = 0.43, *p*= 0.52, *f*= 0.13] (Figure 2.4a) and there was no meaningful difference between DSW (2.9 ± 66.3%) and LW (20.6 ± 36.8%) [F (1, 27) = 2.11, *p*= 0.16, *f*= 0.28]. Likewise, $vCOP_{AP}$ was not affected by the interaction between group and vision [F (1, 28) = 1.34, *p*= 0.26, *f*= 0.22] (Figure 2.4b) and the effect of group was not meaningful for $vCOP_{AP}$ [F (1, 28) = 0.09, *p*= 0.76, *f*= 0.05]; DSW (-2.1 ± 57%) and LW (1.6 ± 27%) were not different. In the ML direction ($vCOP_{ML}$), there was no group by vision interaction [F (1, 27) = 0.39, *p*= 0.54, *f*= 0.12] (Figure 2.4b) nor any differences between DSW (0.80 ± 70.0%) and LW (10.4 ± 38.8%) [F (1, 27) = 0.73, *p*= 0.40, *f*= 0.16].

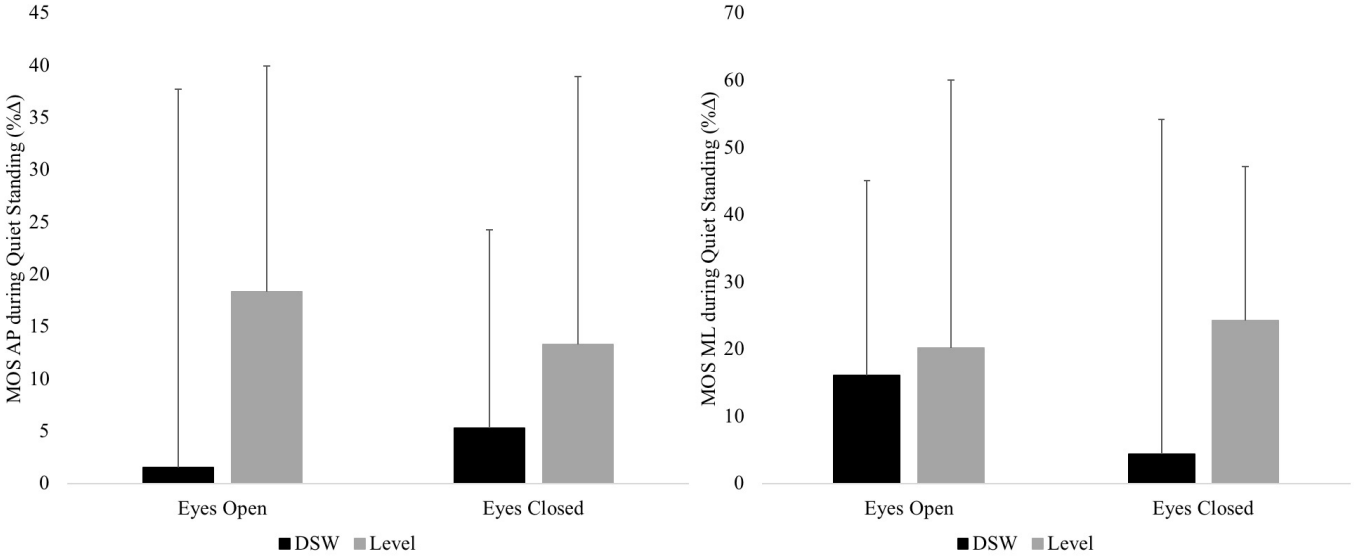

Figure 2.3: Percent Change in Margin of Stability during quiet standing in both visual conditions in AP and ML. **(A)** In AP, MOS was not affected by group or visual condition. **(B)** In ML, MOS was also not affected by group or visual condition.

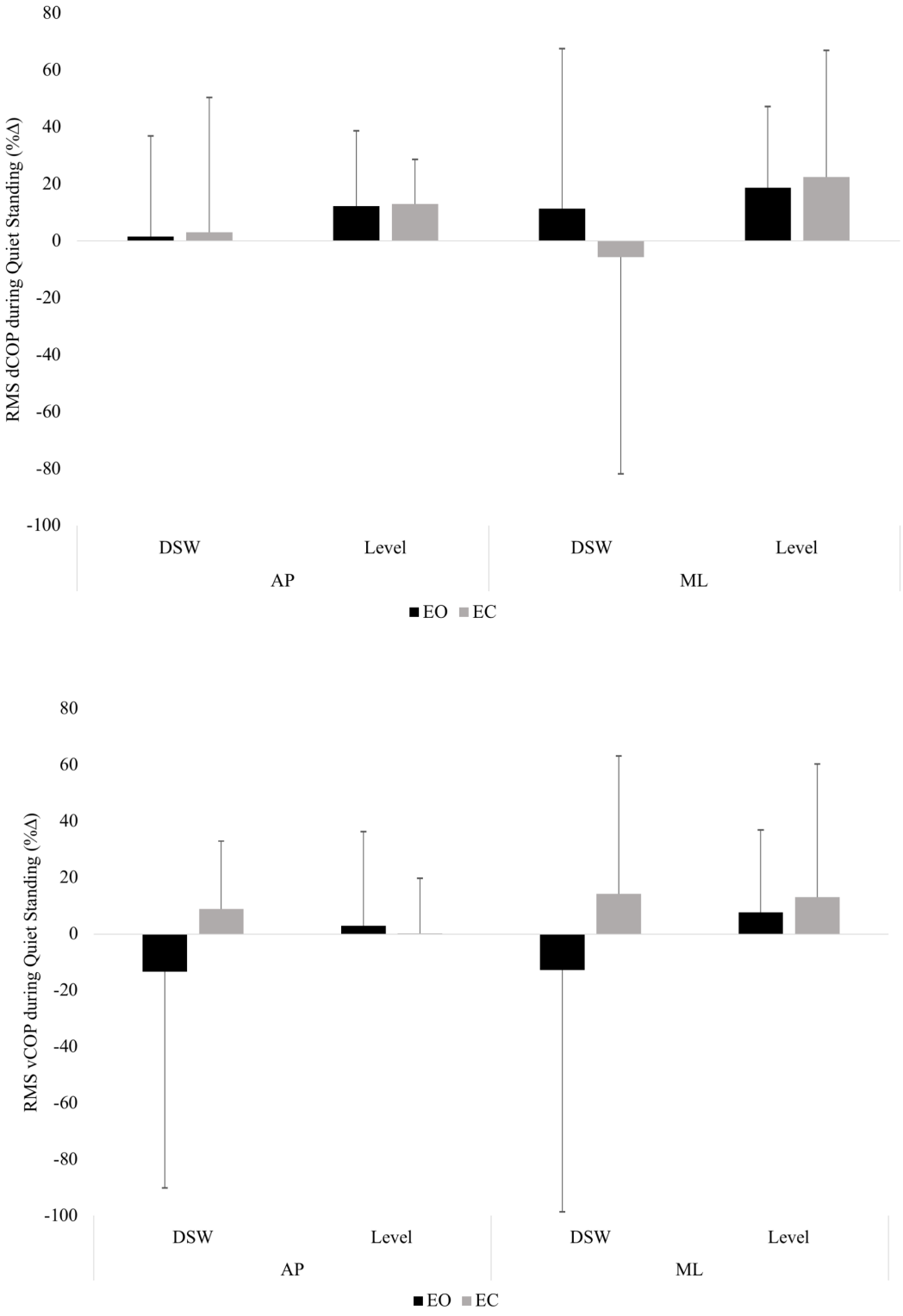

Figure 2.4: **(A)** Percent change of COP displacement (dCOP) during quiet standing was not significant in ML or AP for groups or visual condition. **(B)** Percent change of COP velocity (vCOP) during quiet standing was not significant in ML or AP for groups or visual condition.

Dynamic Balance

Unlike static balance, DSW was able to modify dynamic balance control (for pre- and post- test values refer to Appendix A). There was a strong interaction effect between vision and group on MOS_{AP} [F (1, 28) = 5.48, p= 0.03, f= 0.44] (Figure 2.5a). When full vision was available, DSW decreased MOS_{AP} (-10.3 ± 13.6%), whereas LW increased MOS_{AP} (2.9 ± 13.8%). However, when vision was removed, both DSW and LW had minimal change in MOS_{AP} (DSW: 0.2 ± 13.7%; LW: 0.6 ± 18.3%).Thus, MOS_{AP} across both steps was different between groups [F (1, 28) = 4.21, p= 0.05, f= 0.39]; MOS_{AP} decreased following DSW (-5.1 ± 14.5%) and increased following LW (1.7 ± 16.1%). Recall MOS provides an understanding of the variability in COM control during double support. As such, a decrease in MOS denotes improved balance control since there is less variability in COM position between the boundaries of the base of support. There was no interaction effect between step and group on MOS_{AP} [F (1, 28) = 0.25, p= 0.62, f= 0.10] nor was there any interaction between step, vision, and group [F (1, 28) = 0.36, p= 0.55, f= 0.11.

There was also an interaction between vision and group on MOS_{ML} [F (1, 28) = 4.02, p= 0.06, f= 0.38] (Figure 2.5b), where both groups decreased MOS_{ML} in the no visual condition (DSW: -5.7 ± 22.6%, LW: -9.3 ± 28.1%) but had opposing effects when vision was available. In the eyes open condition, DSW experienced minimal change in MOS_{ML} (0.5 ± 18.6%) whereas LW decreased MOS_{ML} (-18.5 ± 40.9%). Accordingly, MOS_{ML} was different between walking conditions, where DSW (-2.6 ± 20.8%) decreased MOS_{ML} less than LW (-13.9 ± 35.1%) [F (1, 28) = 3.72, p= 0.06, f= 0.36]. There was no interaction between step and group [F (1, 28) = 1.24, p= 0.28, f= 0.21] nor was there an interaction between step, vision, and group [F (1, 28) = 0.55, p= 0.46, f= 0.14].

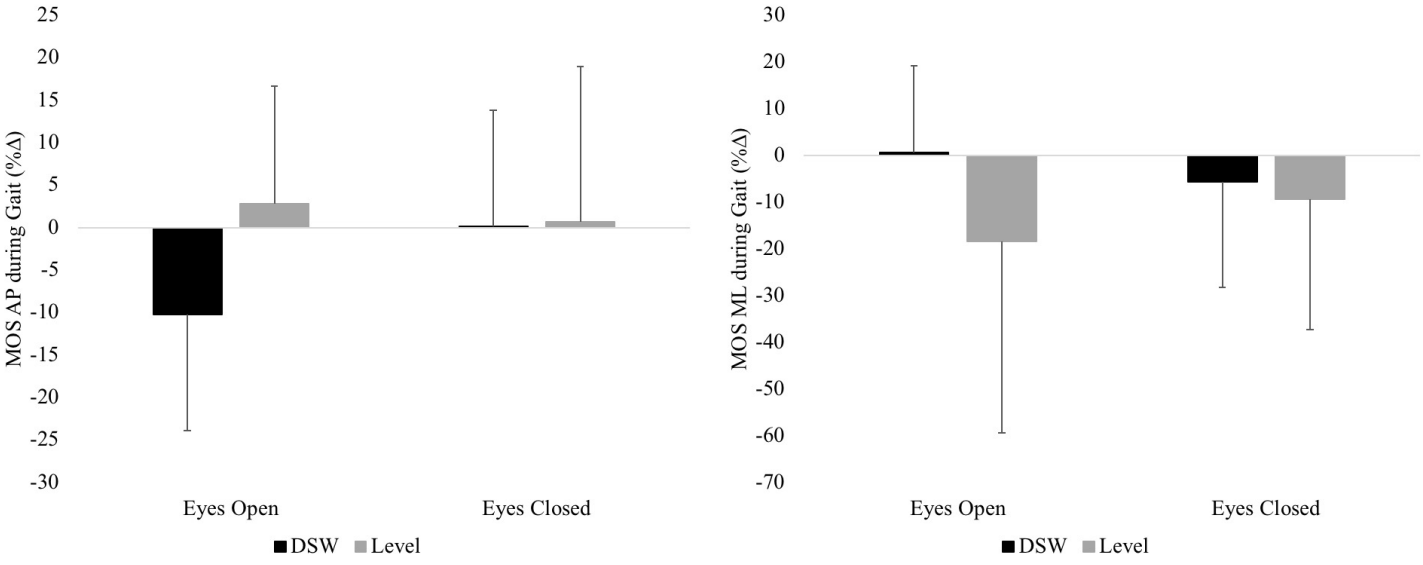

Figure 2.5: Percent change in MOS during gait (step 1 and 2) in AP and ML. **(A)** In AP, MOS was affected by group (p= 0.05, f= 0.39) and an interaction between group and vision (p= 0.03, f= 0.44). MOS_{AP} was not affected by the interaction between group and step nor the interaction between group, step, and vision. **(B)** In ML, MOS was affected by group (p= 0.06, f= 0.36) and an interaction between group and vision (p= 0.06, f= 0.38). MOS_{ML} was not affected by the interaction between group and step nor the interaction between group, step, and vision.

Step length was moderately affected by the interaction between group, step number, and visual condition [F (1, 28) = 4.02, p= 0.06, f= 0.38] (Figure 2.6, Table 2). In the eyes open condition, step length increased from S1 to S2 but was overall decreased following DSW and remained fairly consistent following LW. In the eyes closed condition, step length after DSW remained relatively unchanged and step length did not increase from S1 to S2. Conversely, S2 length was increased following LW. There was also a moderate interaction between group and vision [F (1, 28) = 3.74, p= 0.06, f= 0.37]. When vision was available, DSW decreased step length (-2.9 ± 4.9%) and LW stayed fairly consistent (0.9 ± 3.5%). When no vision was available, both DSW (1.3 ± 8.2%) and LW (1.0 ± 5.6%) slightly increased step length. There was no interaction effect between group and step number [F (1, 28) = 1.11, p= 0.30, f= 0.20] nor were

there any differences between DSW (-0.8 ± 7.0%) and LW (0.9 ± 4.6%) [F (1, 28) = 1.65, p= 0.21, f= 0.24].

TABLE 2
Percent Change in Step Length 3-way Interaction. Values are reported as mean (SD).

	Eyes Open		**Eyes Closed**	
	S1	**S2**	**S1**	**S2**
DSW	-3.7 (6.1)	-2.1 (3.3)	1.4 (10.3)	1.2 (5.9)
Level	0.3 (4.1)	1.4 (2.9)	-1.3 (6.34)	3.3 (3.5)

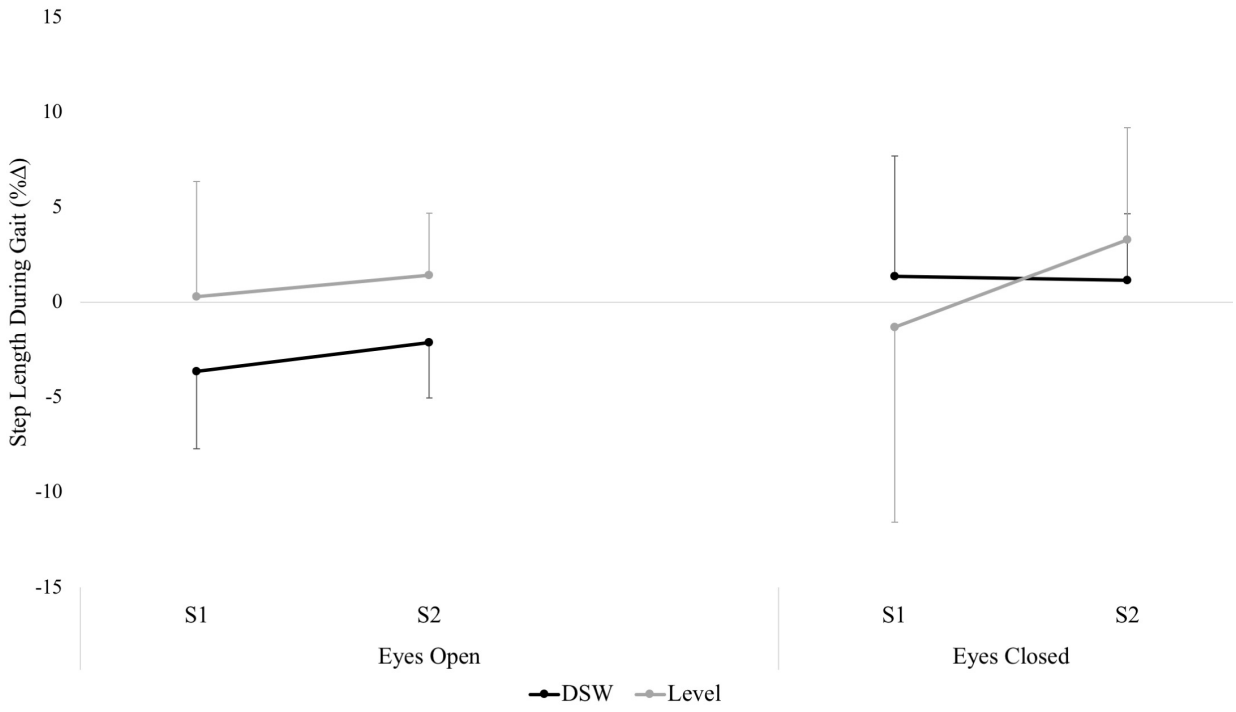

Figure 2.6: Percent change of step length in DSW and LW with eyes open and eyes closed across step 1 (S1) and step 2 (S2). Group x vision x step interaction affected step length (p= 0.06, f= 0.38).

Step length variability was moderately affected by the interaction between group and step number [F (1, 28) = 3.39, p= 0.08, f= 0.35](Figure 2.7). Overall, both DSW and LW decreased step length variability and there was less variability in S2 compared to S1 (Figure 2.7). On S1, LW (-33.4 ± 66.8%) had a greater decrease in step length variability than DSW (-4.7 ± 63.1%). Conversely on S2, DSW had a greater decrease in variability (-48.8 ± 73.6%) than LW (-39.4 ± 88.9%). There was no interaction effect between group and vision on step length variability [F (1, 28) = 0.90, p= 0.35, f= 0.18] nor was there an interaction between group, step number, and visual condition [F (1, 28) = 2.00, p= 0.17, f= 0.27]. Moreover, step length variability was not different between DSW (-26.8 ± 71.5%) and LW (-36.4 ± 78.0%) [F (1, 28) = 0.36, p= 0.55, f= 0.11].

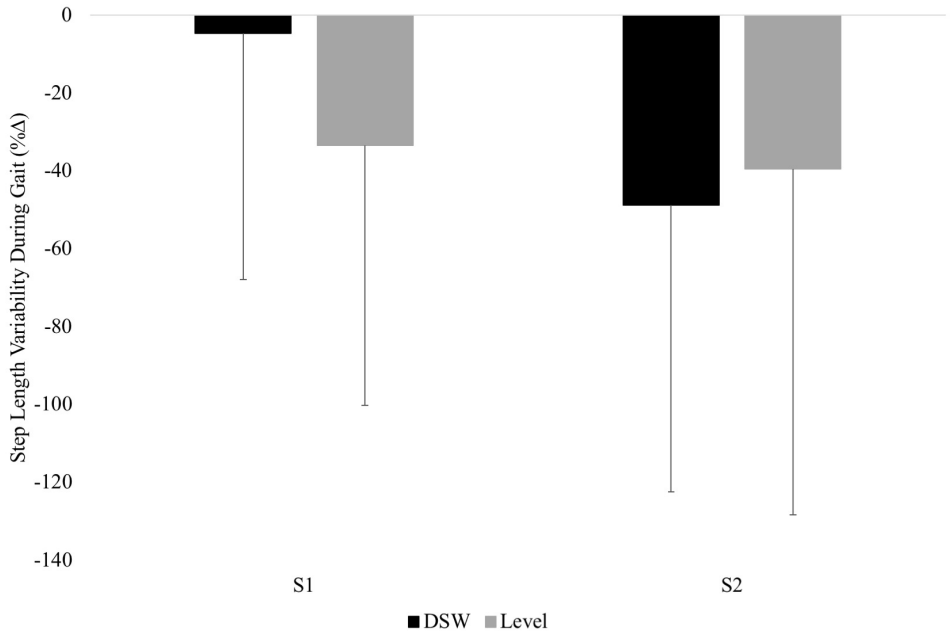

Figure 2.7: Percent change of step length variability in DSW and LW across step 1 (S1) and step 2 (S2). Group x step number interaction moderately affected step length variability (p= 0.08, f= 0.35).

Step width was strongly affected by the interaction between group, step number, and visual condition [F (1, 28) = 5.97, p= 0.02, f= 0.46] (Figure 2.8). When full vision was available, both DSW and LW experienced a decrease in SW across both S1 and S2. However, when vision was not available SW increased on S2 after DSW; SW was decreased on S1 by both groups and on S2 after LW (Table 3). There were no interaction effects between group and step number [F (1, 28) = 1.73, p= 0.20, f= 0.25] nor group and vision [F (1, 28) = 0.12, p= 0.74, f= 0.06]. Step width was also not influenced by walking condition [F (1, 28) = 0.97, p= 0.33, f= 0.18]; both DSW (-6.1 ± 28.3%) and LW (-12.2 ± 28.1%) decreased step width but the difference was not meaningful.

For step width variability, there were no interactions between group and step number [F (1, 28) = 1.17, p= 0.29, f= 0.20], group and vision [F (1, 28) = 1.40, p= 0.25, f= 0.22], nor group, step number, and visual condition [F (1, 28) = 0.26, p= 0.61, f= 0.10]. There was also no effect of group on step width variability [F (1, 28) = 0.64, p= 0.43, f= 0.15]; the decline in variability experienced by DSW (-11.1 ± 60.8%) was not different from LW (-27.2 ± 124.6%).

TABLE 3
Percent Change in Step Width 3-way Interaction. Values are reported as mean (SD).

	Eyes Open		**Eyes Closed**	
	S1	**S2**	**S1**	**S2**
DSW	-2.7 (22.8)	-9.8 (22.5)	-21.9 (37.1)	9.8 (20.2)
Level	-14.1 (19.2)	-13.9 (19.9)	-9.7 (44.1)	-11.1 (24.4)

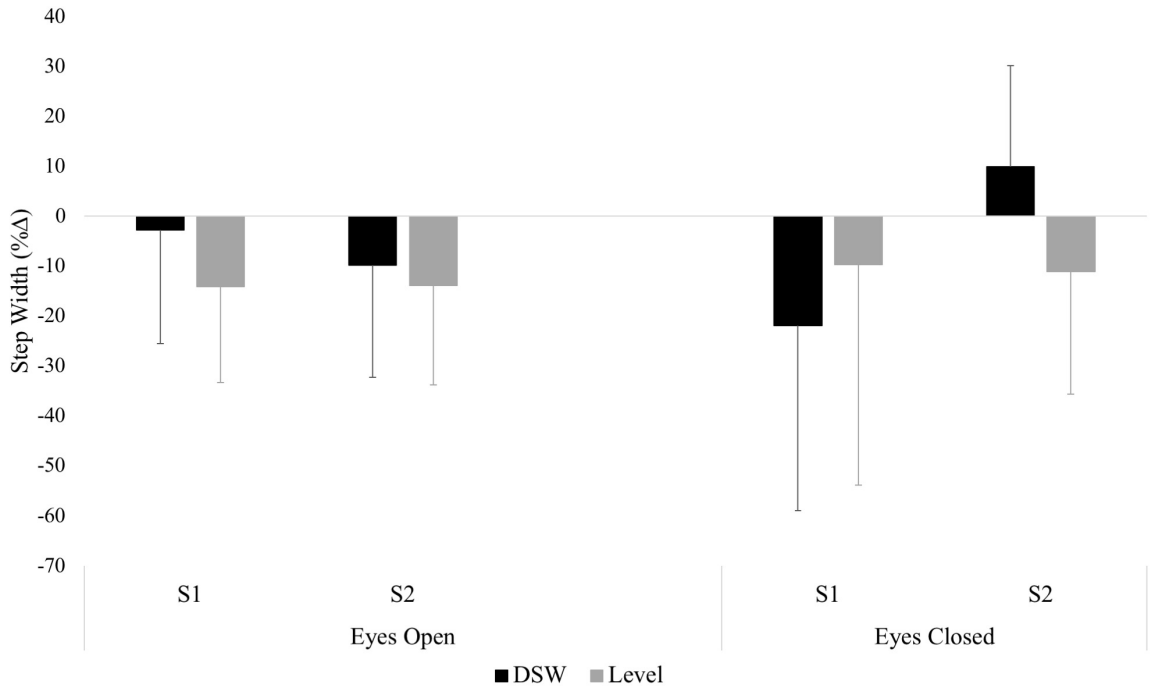

Figure 2.8. Percent change of step width in DSW and LW with eyes open and eyes closed across step 1 (S1) and step 2 (S2). Group x vision x step interaction affected step length (p= 0.02, f= 0.46).

ML COM variability from gait initiation to termination was strongly affected by an interaction between group and vision [F (1, 28) = 4.50, p= 0.04, f= 0.40] (Figure 2.9). When no vision was available, DSW decreased ML COM variability (-15.1 ± 31.9%), which was consistent to the effects of LW regardless of whether vision was available to them (-7.3 ± 20.7%) or not (-4.7 ± 18.9%). When vision was available, DSW increased ML COM variability (6.4 ± 8.3%). ML COM variability was not different between DSW (-4.4 ± 25.4%) and LW (-6.0 ± 19.5%) [F (1, 28) = 0.09, p= 0.77, f= 0.05].

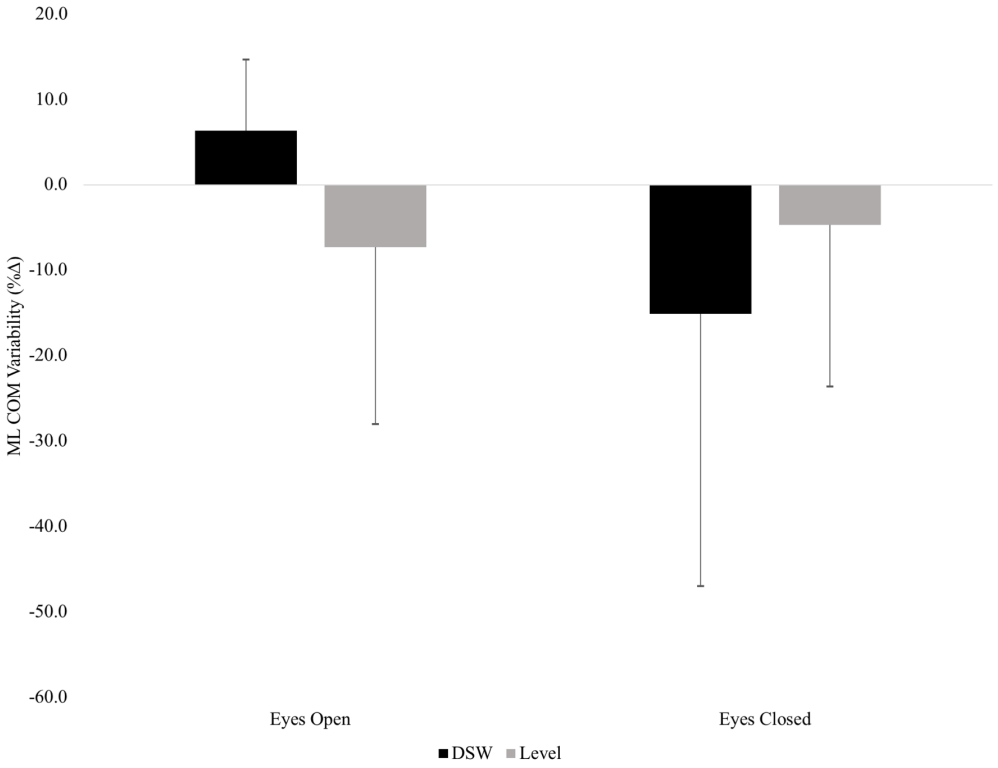

Figure 2.9: Percent change in ML COM variability across both visual conditions and both groups. The interaction between group x vision (p= 0.04, f= 0.40) influenced ML COM variability.

H reflex Measures

H reflex Recruitment Curves

The degree of spinal motor neuron pool excitability following DSW was estimated through the slopes, maximum values, and threshold values of the H reflex recruitment curves in order to support that the behavioural measures occurred in conjunction to a depression in H reflex. H_{slope}/M_{slope} was not affected by any interactions between group and time [F (1, 24) = 0.87, p= 0.36, f = 0.19] (Figure 2.10a) and was not different between DSW (0.91 ± 0.63) and LW (1.06 ± 0.84) [F (1, 24) = 0.48, p= 0.50, f= 0.14]. Thus, from pre-test to post-test there were no differences across walking conditions (Table 4). Similarly, there was no interaction between

group and time for H_{max}/M_{max} [F (1, 27) = 0.02, p=0.89, f=0.03] and no group effect [F (1, 27) = 1.94, p= 0.19, f= 0.26]. H_{max}/M_{max} from DSW (0.39 ± 0.16) and LW (0.46 ± 0.18) were not different and neither was the decrease experienced by both groups from pre-test to post-test (Table 4). Therefore, percent change was used to determine the difference in H reflex depression between groups. DSW trended towards a greater percent change compared to LW on H_{slope}/M_{slope} [t (15.57) = -1.94, p= 0.07, d= 0.76] (Figure 2.10b) but not H_{max}/M_{max} [t (24) = -0.01, p= 0.99]. Specifically, DSW led to a 46.94% (*SD*= 15.4) decrease in H_{slope}/M_{slope}, while LW led to a 24.16% (*SD*= 39.46) decrease. Comparatively, H_{max}/M_{max} decreased 11.84% (*SD*= 28.76) by DSW and 11.70% (*SD*= 24.49) by LW. For the threshold values of the curve, there was an interaction effect between group and time, such that DSW decreased H_{th}/M_{th} and LW increased H_{th}/M_{th} [F (1,25) = 4.76, p= 0.04, f= 0.48] (Figure 2.11, Table 4). Accordingly, there was a strong effect of group where DSW (0.72 ± 0.10) had an overall lower threshold than LW (0.81 ± 0.08) [F (1,25) = 6.64, p= 0.02, f= 0.52]. The percent change scores for H_{th}/M_{th} were not meaningfully different between DSW (-4.95 ± 9.88) and LW (0.38 ± 7.27) [t (26) = -1.60, p= 0.12, d= 0.61].

TABLE 4
Averages for H reflex recruitment curve slopes, maximum values, and threshold values during the pre- and post-test, and average percent change for slope and maximum values. Values are reported as mean (SD).

	H slope/ M slope		H max/ M max		H th / M th	
	DSW	**Level**	**DSW**	**Level**	**DSW**	**Level**
Pre	1.11 (0.69)	1.10 (0.66)	0.40 (0.17)	0.47 (0.16)	0.74 (0.11)	0.79 (0.08)
Post	0.70 (0.51)	1.02 (1.03)	0.38 (0.16)	0.45 (0.21)	0.70 (0.10)	0.82 (0.09)
%Change	-46.94 (15.40)	-24.16 (39.46)	-11.84 (28.76)	-11.70 (24.49)	-4.95 (9.88)	0.38 (7.27)

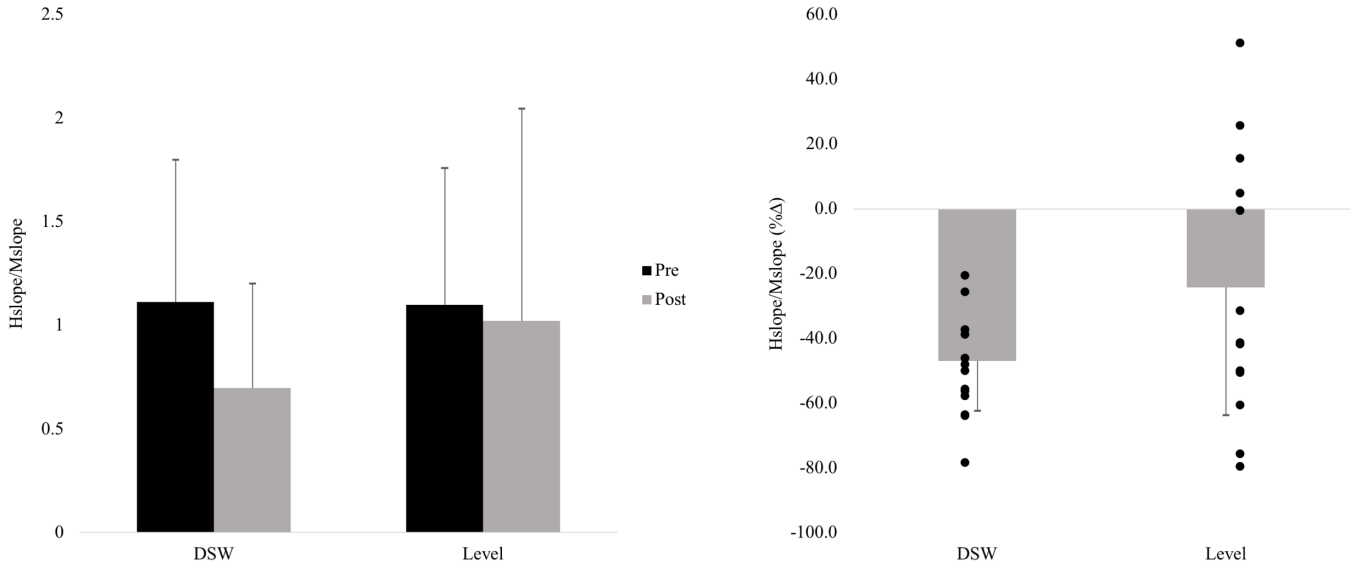

Figure 2.10: **(A)** H_{slope}/M_{slope} in the pre- and post-test for DSW and LW. Neither effect of group nor time meaningfully affected H_{slope}/M_{slope}. **(B)** Percent change of H_{slope}/M_{slope} for DSW and LW. Points represent each participant's H_{slope}/M_{slope} in their respective group. Trend towards greater percent change in H_{slope}/M_{slope} due to DSW compared to LW (p= 0.07).

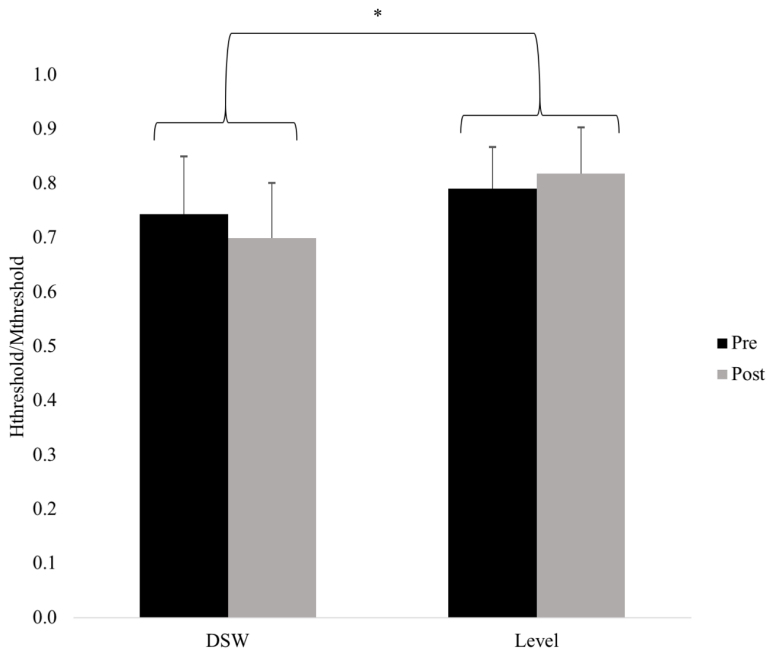

Figure 2.11: $H_{threshold}/M_{threshold}$ (H_{th}/M_{th}) between groups and pre- and post-testing. H_{th}/M_{th} was affected by group (p= 0.02, f= 0.52) and the interaction between group x time (p= 0.04, f= 0.48).

Conditioned H reflex

The average peak-to-peak amplitude of the H and M waves from the 10 conditioned H reflex trials and the 10 unconditioned H reflex trials were measured to determine the amount of conditioning before and after treadmill walking. Greater amounts of conditioning following DSW would indicate that there was a greater amount of reciprocal inhibition during the task, which may have explained why the H reflex depressed. However, the conditioned H reflex was not affected by the interaction between group and time [F (1,23) = 0.15, p= 0.71, f= 0.08] (Figure 2.12a), since both DSW and LW experienced an increase in the amount of conditioning from pre-test to post-test (Table 5). Accordingly, the conditioned H reflex was not affected by group [F (1,23) = 2.82, p= 0.11, f= 0.35], such that DSW (100.2 ± 12.85) and LW (106.52 ± 13.90) were not different in their overall amount of conditioning. When considering the percent change

in reflex conditioning, DSW did not lead to greater amounts of conditioning than LW [t (25) = 0.55, p= 0.59, d= 0.21] (Figure 2.12b).

TABLE 5
Averages for conditioned H, expressed as peak-to-peak amplitude of conditioned H in relation to that of the unconditioned H, and average percent change of conditioned H. Values are reported as mean (SD).

	Conditioned H / Unconditioned H	
	DSW	**Level**
Pre	103.23 (7.1)	110.94 (11.3)
Post	97.19 (16.5)	102.11 (15.3)
%Change	-0.40 (25.6)	-4.98 (16.1)

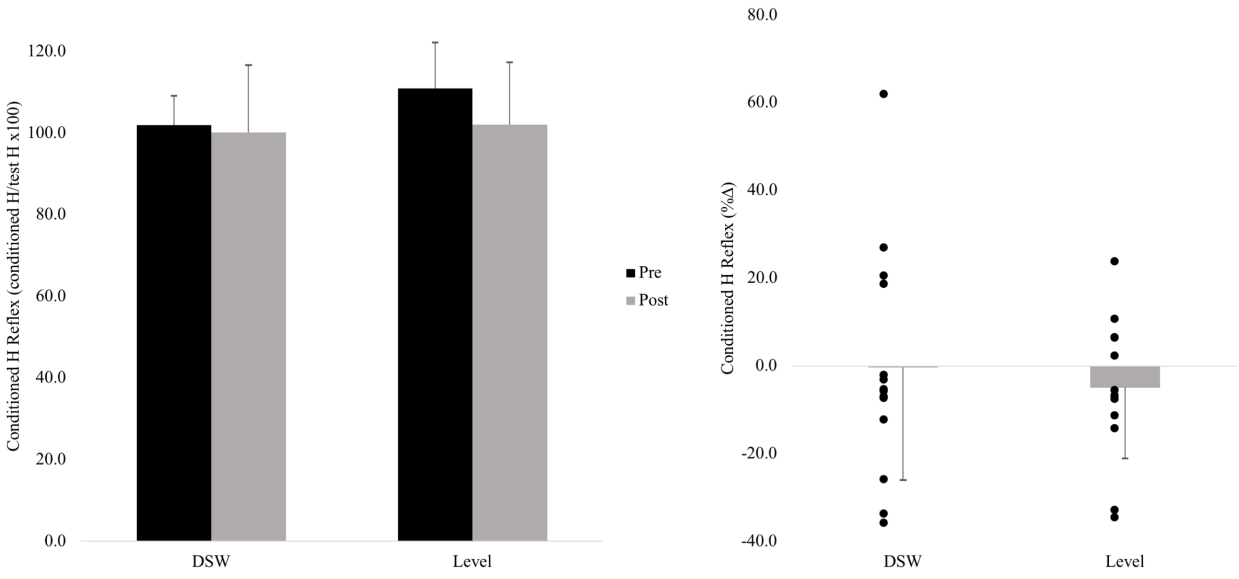

Figure 2.12: **(A)** Conditioned H reflex (represented as the percentage of the unconditioned H reflex). Neither group nor testing session affected the amount of conditioning. **(B)** Percent change of conditioned H reflex of all participants in both groups. Neither group had a larger percent change in conditioning.

H reflex Correlation to Balance Control

An unplanned comparison between the H reflex and step 3 MOS was undergone since DSW and LW were significantly different on this step when full vision was available [t (41.15) = -2.26, p= 0.03, d= 0.59]. DSW had a strong negative correlation between the percent change of H_{slope}/M_{slope} and step 3 MOS_{AP} (R^2 = 0.68, r= -0.83), whereas LW did not (R^2 = 0.01, r= -0.1) (Figure 2.10). Similarly, there were weak correlations between H_{slope}/M_{slope} and step 3 MOS_{ML} in both DSW (R^2 = 0.02, r= -0.14) and LW (R^2 = 0.14, r= -0.37).

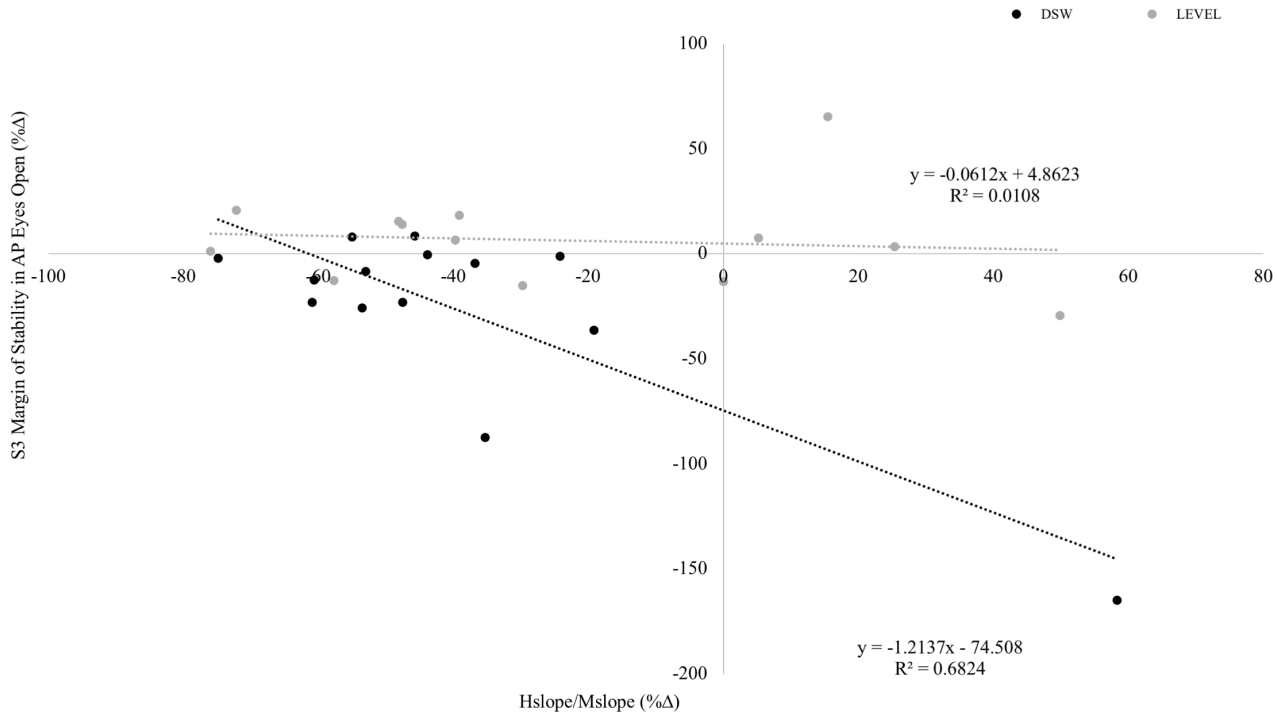

Figure 2.13: Correlation between percent change of MOS_{AP} on step 3 and H_{slope}/M_{slope}. Strong negative correlation between MOS_{AP} and H_{slope}/M_{slope} in DSW (r= -0.83) but not LW (r= -0.1). Correlation between variables excluding outlying point had R^2 = 0.086 (r = -0.29).

Discussion

The purpose of this study was two-fold. First, our study aimed to objectively determine whether young adults could demonstrate improved balance control in parallel to a decrease in spinal motor neuron (MN) pool excitability after an acute bout of DSW. To achieve this, kinematic and kinetic data were collected while participants stood quietly on a force plate for 30-sec and then walked forward taking 3 steps at a self-selected speed and step length. Next, participants underwent repeated tibial nerve stimulation for generation of a soleus (SOL) H reflex recruitment curve and conditioned SOL H reflexes. We believed that DSW would improve dynamic but not static balance control and that DSW would cause greater H reflex depression than LW. In partial accordance with our hypotheses, changes due to DSW were only evident once the person began walking. However, the changes did not lend support for DSW improving balance control in young adults since step length decreased and ML COM variability increased. Regardless, as expected, the changes to balance control occurred in conjunction to a decrease in spinal MN pool excitability, indicated by greater SOL H reflex depression following DSW than LW. Our second aim was to determine whether reciprocal inhibition was one of the possible mechanisms responsible for SOL H reflex depression. To accomplish this aim we elicited a conditioned H reflex by stimulating the peroneal nerve prior to the tibial nerve. This pattern of stimulation should decrease the H reflex by increasing activity of the Ia inhibitory interneuron from the TA onto the SOL. We believed that reciprocal inhibition would increase following an acute bout of DSW, measured as an increase in the amount of SOL H reflex conditioning. However, we found no change in the level of reciprocal inhibition after an acute bout of DSW, so it is likely not the mechanism responsible for the recorded H reflex depression.

In order to see the effects of DSW in a young adult population, participants need to be walking and using dynamic balance control. Following an acute bout of DSW, young adults demonstrated decreased MOS_{AP} (Figure 2.5a) and decreased ML COM variability (Figure 2.9). However, the decrease in MOS_{AP} can be explained by participants decreasing their step length (SL) (Figure 2.6) (77). With less distance for the COM to travel between the AP borders of their base of support, the possible variability in COM position is restricted. SL decreased following a single DSW session most likely because during DSW there was increased AP breaking forces (67) and less time spent in stance (61), leading participants to walk with shorter steps (49). One benefit to decreasing SL is that the COM can be better controlled in the plane of progression. However, decreased SL also corresponds to a more cautionary gait (81) and a decrease in speed (26, 28, 65), indicating that YA would likely take longer to walk towards their destinations after DSW. This is further supported by an increase in ML COM variability and a slight increase in MOS_{ML}. Accordingly, the ability for participants to control their whole-body movement from side-to-side was negatively affected over the entire duration of the task and participants had difficulty controlling their COM in relation to their BOS in the ML direction. Considering that participants walked with shorter steps, had greater variability in ML whole-body motion and within their BOS, and participants likely walked at a slower speed, DSW does not appear to be beneficial to the balance control of young adults.

There were also differences in SL variability between groups based on step number (Figure 2.7). If DSW was destabilizing and affected COM control, then SL variability should have increased since participants would be continually changing their foot placement in order to regain control of their COM (73, 81). This was partially the case when comparing DSW and LW on both steps: SL variability decreased less after DSW than LW on step 1, but overall both

groups decreased SL variability. A possible explanation for the greater decrease in SL variability on step 2 is that this was the only non-transitional step (i.e., did not involve gait initiation or termination). As such, DSW seemed to negatively influence COM more on the transitional step (i.e., step 1) since transitional steps challenge the balance control system (42); gait initiation is a self-perturbation requiring posterior displacement of the COP (55). There were no differences in SW variability due to DSW, likely because the nature of the perturbation was in the AP direction.

Our findings that an acute bout of DSW would likely lead to decreased walking speed during walking tasks are in direct contradiction to the DSW literature in PwMS. DSW for 30-minutes increased walking speed of PwMS, measured with improvements during the timed 25-ft walk test, in conjunction to improvements in the 2-minute walk test and Timed-Up-and-Go Test (105). However, similar to our findings, Hosp and colleagues (52) identified that an acute bout of DSW led to decreased postural stability in young adults. This suggests that an acute bout of DSW improves balance control in PwMS but not in young adults, so the populations must react to DSW differently. DSW mechanically changes gait and one of these changes is an alteration in the angle of the ankle (31, 61, 68). Compared to level walking, the maximum angle about the ankle is decreased (61), which corresponds to a smaller peak plantar flexor moment (68). It is speculated that manipulating the angle of the ankle alters joint position sense, so the proprioceptive information being received is novel compared to standard walking over flat ground. Put differently, DSW is a possible perturbation to the somatosensory system. Accordingly, people will likely decrease their reliance on proprioceptive information through changes in sensory reweighting (88, 94, 106). It is possible that young adults respond differently to DSW than PwMS because of the pathology of the disease. In MS, demyelination throughout

the CNS impairs conduction of spinal somatosensory information (7) and integration of sensory information (12, 39). As a result, PwMS have difficulty with sensory reweighting (19). DSW may act as a form of sensory integration balance training (12, 39) to improve their usage of the different sensory systems. PwMS rely heavily on vision and less on proprioception (19) so DSW could force PwMS to re-evaluate the emphasis placed on the various sensory systems. Conversely, young adults do not seem to respond favourably to the changes in sensory integration which occur as a result of DSW. Locomotion of young adults is not heavily dominated by any one sensory system; vision, proprioception, and vestibular information all contribute to successful locomotion. As such, we speculate that when you force someone to reconfigure their sensory inputs by having them walk under a novel condition (i.e. altered ankle angle), you are now forcing them to rely less on proprioception. Similar to our findings, downregulating proprioception has been shown to cause transient postural instabilities in young adults once proprioceptive information is restored (46, 95). Thus, young adults have difficulty readjusting the emphasis placed on each sensory system necessary to complete a balance task with control.

However, it should be noted that the effects of DSW were only exhibited when sensory system availability was congruent between training and the walking task. The decrease in SL and MOS_{AP} and increase in ML COM variability all occurred when vision was available during the behavioural task. These findings did not occur when vision was removed by having participants close their eyes. Without vision, the effects were similar after DSW and LW. For example, there was only a minimal increase in step length (Figure 2.6) which explains the minimal change in MOS_{AP} (Figure 2.5a) (77) and ML COM variability decreased (Figure 2.9) with a subsequent decrease in MOS_{ML} (Figure 2.5b). Since both groups demonstrated similar effects when vision

was not available, it is possible that the findings were simply properties of walking on a treadmill. Additionally, there was likely some influence from having to reconfigure the emphasis placed on the sensory systems in order to complete the walking task. If DSW decreased reliance on proprioception, then once back on flat ground reliance on proprioception likely returned to "normal". However, because vision was unavailable during the behavioural task, it is possible proprioception had to be further upregulated for balance control. Overall, this means that the effects of an acute bout of DSW seem to be washed out when performing the walking task without vision and the best way to identify any changes from DSW is to have the same sensory systems (i.e., vision) available during training and the task. It should be noted SW was different between DSW and LW when no vision was available; SW increased from S1 to S2 after DSW but remained decreased on both steps after LW, which was similar to the changes in both groups when vision was available (Figure 2.8). The increase in SW on S2 may have been the result of more cautionary gait (decreased MOS_{ML}) when vision was unavailable, especially if DSW was a destabilizing task.

As hypothesized, DSW did not affect any measures of static balance control, regardless of whether vision was available during the task or not. Specifically, there were no changes in $MOS_{AP\ or\ ML}$ (Figure 2.5a, 2.5b), $dCOP_{AP\ or\ ML}$ (Figure 2.4a), or $vCOP_{AP\ or\ ML}$ (Figure 2.4b). No changes were expected despite previous literature demonstrating significant differences in static balance control following DSW because these studies used a Biodex Balance System, which does not incorporate measures of COM and COP control (52, 105). Regardless, studying static balance provides an important understanding of balance control at a fundamental level since static balance control occurs predominately without cortical involvement. Muscle activity for voluntary movements occurs after 150-ms (17), whereas muscle responses to platform

displacements typically occur between 70 and 120-ms (51, 82, 83). Thus static balance control can occur without the spinal cords' control of rhythm or the supra-spinal control of ML balance control and propulsion (89). The monosynaptic pathway involved in the control of static balance to perceive changes in somatosensory information and react through changes in muscle activity was measured with the SOL H reflex to understand the excitability of the spinal MNs. Since we identified a decrease in spinal MN pool excitability, we have support that DSW influenced the monosynaptic pathway in some capacity. However, there was no effect of DSW on static balance control despite measured changes at the spinal level. We identified changes to dynamic balance control only, which involves supra-spinal structures. Thus, it is possible that the changes due to DSW occurred at both spinal (i.e. decreased spinal MN pool excitability) and supra-spinal levels. This is supported by changes to ML balance control via increased ML COM variability (Figure 2.9) and changes to propulsion via MOS_{AP} (Figure 2.5a).

In accordance to previous literature (2, 50, 104), the balance control changes identified after DSW were supported by a decrease in spinal MN pool excitability, measured as a decrease in H_{slope}/M_{slope} amplitude (Figure 2.10b). It should be noted that the change in H reflex was not statistically significant between groups ($p = 0.07$) although a moderate effect size ($d= 0.76$) suggests that this trend may represent a meaningful difference. Power analysis revealed that a sample size around 30 was required, so increasing the sample size from 15 participants in each group would have likely revealed a significant difference between the groups ($\beta= 0.15$). Therefore, based on the moderate effect size it is likely there was a difference between DSW and LW, but the difference was not statistically significant due to low power. Moreover, another possible explanation for the non-significance is the high variability in percent change scores across participants in the LW group (Figure 2.10b). Hoque and colleagues (50) also had

participants whose H reflex increased, however their ranges in percent change scores between groups were similar, unlike ours. Furthermore, it is possible that the difference between groups did not reach significance since H reflex measures occurred following the 10 walking trials, which was on average 16 (*SD*= 2.2) minutes following treadmill walking. Previous literature indicates significant differences in H reflex depression between groups after 10-minutes (2, 50, 104) and occasionally 45-minutes (50), although these studies had participants resting from cessation of the treadmill to H reflex recordings.

In spite of the large range of positive and negative percent change scores in the LW group, on average their H_{slope}/M_{slope} percent change was negative. This indicates that both DSW and LW elicited an overall decrease in H reflex but there was a greater decrease following DSW. H reflex depression following LW has been identified previously for H_{max}/M_{max} but not H_{slope}/M_{slope} (50, 104). We found no changes in H_{max}/M_{max} which is dissimilar to the current DSW literature and may suggest that H_{slope}/M_{slope} is a more sensitive measure (34). Our findings suggest that H reflex depression (H_{slope}/M_{slope}) was likely due to a change in the input-output relationship or a decrease in H reflex gain (35). Since the H reflex is a measure of spinal MN pool excitability, a decrease in H reflex indicates a decrease in the overall excitability of the MN pool. When this happens, the MN pool becomes less saturated (9), which enhances its capacity for modulation by afferent feedback (64). This is important in a situation like DSW where proprioceptive information is continuously changing due to alterations in ankle angle (61, 68) and the corresponding muscle activity must be adapted (31, 68) to maintain balance control while DSW.

DSW also influenced the thresholds of the H reflex recruitment curves (H_{th}/M_{th}), but in a manner opposite to LW: H_{th}/M_{th} decreased due to DSW and increased due to LW (Figure 2.11).

In other words, DSW shifted H_{th} to the left, suggesting that low threshold motor units became more excitable. In contrast, LW shifted H_{th} to the right and made low threshold motor units less excitable. However, interpreting changes in H reflex thresholds requires some caution. It is understood that MNs are recruited from low to high resting conductance (10) but H reflex thresholds estimate the excitability of the lowest threshold motor neurons only (36). Therefore, assumptions cannot be made about the excitability of the entire MN pool (36). Nonetheless, adjustment of H_{th} by DSW supports that the changes in H reflex are most likely due to changes in spinal excitability rather than changes in peripheral transmission, accidental shifts in electrode position, or adherence from pre-test to post-test as these explanations would have shifted M_{th} instead.

Furthermore, a decrease in spinal MN pool excitability has been previously related to a decrease in postural control. Specifically, a decrease in soleus H reflex has been related to an increase in COP displacement (116) and an increase in RMS of COP displacement (53). For this reason, we wanted to correlate whether our decrease in H reflex was related to MOS, one of our primary measures of balance control. MOS_{AP} during gait was selected because of the difference between groups on this measure. We identified that within the DSW group there was a strong negative correlation between H_{slope}/M_{slope} and MOS of the third step, whereas there was no correlation between these factors within the LW group (Figure 2.10). In essence, it seems that greater levels of H reflex depression relate to more variable COM control, which is indicative of poorer balance control. This finding is in line with previous literature (53, 116) and may suggest that the mechanism underlying H reflex depression is related to the soleus' role in postural control. However, the strength of this correlation was driven by an outlying participant in the

DSW group. The participant was not removed from analysis because similar changes in H_{slope}/M_{slope} occurred in participants in the LW group.

The decrease in soleus H reflex was thought to be due to an increase in reciprocal inhibition in consideration to the known muscle activity changes during DSW: TA activity increases (68) and soleus activity decreases (31). It was assumed that if TA muscle activity increased, there would be a subsequent increase in Ia inhibitory interneuron activity, and a resulting decrease in soleus alpha MN activity, which would be recorded as a decrease in soleus EMG. Contrary to this assumption, the effect of the antagonist nerve conditioning stimulus on the H reflex conditioning did not increase following DSW, suggesting that there were no significant effects of DSW on the level of reciprocal inhibition during quiet standing following the treadmill task (Figure 2.12a). There was low power (β= 0.07) and high variability in the percent change of conditioned H reflex among DSW participants, which likely contributed to the non-significance (Figure 2.12b) and low effect size (d= 0.21).

Our hypothesis that reciprocal inhibition would increase was rooted in the idea that soleus inhibition would increase with increasing TA activity. However, this relationship is more valid during tonic muscle activity and thus the strength of soleus inhibition is not always proportional to the level of activity in the TA (66). Reciprocal inhibition of the soleus can occur independent of TA activity because inhibition is partly centrally mediated; afferent activity is not required (66). For example, when the peroneal nerve is anesthetized, reciprocal inhibition of the soleus can still occur (85). It is possible that reciprocal inhibition still has a role in decreasing the soleus H reflex, although it is more likely that the level of reciprocal inhibition is being modified by either an increase in pre-synaptic inhibition or descending drive, not an increase in TA activity (66). Pre-synaptic inhibition has been previously identified as a possible mechanism for

changing the gain of the monosynaptic reflex (9, 10) and it contributes to soleus H reflex modulation during gait (8, 9). Increasing activity of the presynaptic inhibitory interneurons onto soleus Ia afferent terminals or increasing activity of the Ia inhibitory interneurons through descending projections would both increase soleus inhibition and thus decrease the soleus H reflex (66). At this time, we do not know which is more likely contributing to the soleus H reflex depression.

Conclusion

The effects of DSW were only apparent once participants began walking and using dynamic balance control strategies. However, it was not expected that the balance control changes would be negative. Additionally, the same sensory systems must be available during training and the task since removing vision during the task eliminated the effects of DSW. The balance control changes were supported by a decrease in spinal MN pool excitability, however an increase in reciprocal inhibition was not likely the mechanism. Overall, these findings provide support that DSW affects the balance control strategies of young adults differently than PwMS. Young adults provide a fundamental understanding of the effects of DSW although caution should be taken when trying to apply these findings to understand how DSW effects PwMS.

Chapter 3: General Conclusion

The basis of this study was grounded in Multiple Sclerosis literature in the hopes of applying the findings with young adults to better understand why clinicians are implementing DSW into the rehabilitation programs of PwMS. Our findings suggest that young adults may respond differently than PwMS to an acute bout of DSW in regard to their balance control strategies. However, none of the DSW studies in PwMS include measures of COM or COP control; only global improvements in PwMS have been identified in terms of being able to walk further and faster and having better postural stability (105). We have highlighted that DSW influences the COM control of young adults so it would be beneficial for a future study to use our experimental protocol but in an MS population. We learned that having participants close their eyes and perform the walking task washed out all effects of DSW, so future study should involve full vision only, keeping available sensory conditions congruent between DSW and the task. We identified no differences due to DSW in static balance control, however PwMS should still be monitored for changes in static balance. PwMS may be more susceptible to the effects of DSW because they have greater room for improvement in balance control than young adults, who are more likely to encounter a ceiling effect. It should be noted that there were no differences between the first and last trail across our balance control measures. Thus, DSW seemed to have a conditional effect on balance control strategies versus a temporal effect. As such, walking in the behavioural task for 10 trials likely did not dilute the effects of DSW. Moreover, in a future study each participant should complete both DSW and LW in a repeated measures design so participants could be compared for their performance on both walking conditions. The behavioural findings from our study highlight specific features which should be employed in future DSW testing if done in PwMS.

Further testing is also needed to identify the mechanism responsible for H reflex depression. We identified that reciprocal inhibition was likely not the mechanism, however we had a small sample size. Post-hoc power analysis indicated that a sample size over 300 would have been required to detect a difference in the amount of conditioning between DSW and LW. A difference would have supported that reciprocal inhibition increased after DSW. Nevertheless, increasing the sample size to that degree would be forcing a statistical difference between the groups which may or may not exist. All that we can conclude from our non-significance is that another mechanism, such as pre-synaptic inhibition, may be responsible for decreasing the soleus H reflex. A future study should test whether there are changes in the level of pre-synaptic inhibition following an acute bout of DSW. The change in level of pre-synaptic inhibition could be compared to any changes in the level of reciprocal inhibition through repeated measures testing.

Our study provides insight into the underlying changes due to an acute bout of DSW in regard to changes at the spinal (i.e. motor neuron pool excitability) and supra-spinal (i.e. ML balance control, propulsion) levels using young adults with no known neurological disorders or diseases. DSW is an interesting tool to alter balance control strategies through manipulating the angle about the ankle. DSW is a novel task and easily accessible for clinicians to incorporate into rehabilitation programs. However, before this can occur, we must understand the basic changes to COM and COP control in PwMS since we have identified that COM control decreases following DSW in young adults in parallel to a decrease in spinal motor neuron pool excitability.

Appendices

Appendix A: Data Tables

Table A1: Hslope/Mslope

Participant	Pre	Post	Percent Change
DS 1	0.87	0.55	-36.8
DS 2	0.68	0.44	-35.3
DS 3	2.07	1.57	-24.2
DS 4	1.83	0.86	-53.0
DS 5	0.20	0.09	-55.0
DS 6	0.35	0.19	-45.7
DS 7	0.89	0.50	-43.8
DS 8	1.51	0.38	-74.8
DS 9	2.52	3.99	58.3
DS 10	1.32	0.52	-60.6
DS 11	1.33	0.52	-60.9
DS 12	0.19	1.84	868.4
DS 13	2.17	1.14	-47.5
DS 14	1.70	0.79	-53.5
DS 15	0.47	0.38	-19.1
AVERAGE	**1.21**	**0.92**	**21.1**
SD	**0.75**	**0.98**	**236.4**
L 16	0.74	3.94	432.4
L 17	0.84	0.97	15.5
L 18	1.21	0.85	-29.8
L 19	0.79	0.19	-75.9
L 20	0.92	0.56	-39.1
L 21	1.33	0.37	-72.2
L 22	0.71	0.89	25.4
L 23	3.85	1.63	-57.7
L 24	0.63	0.38	-39.7
L 25	0.30	0.30	0.0
L 26	1.55	1.63	5.2
L 27	3.53	5.29	49.9
L 28	1.29	0.67	-48.1
L 29	2.88	1.51	-47.6
AVERAGE	**1.47**	**1.37**	**8.4**
SD	**1.12**	**1.48**	**127.8**

Table A2: Hmax/Mmax

Participant	Pre	Post	Percent Change
DS 1	0.44	0.27	-38.6
DS 2	0.20	0.43	115.0
DS 3	0.39	0.38	-2.60
DS 4	0.24	0.62	158.3
DS 5	0.07	0.10	42.9
DS 6	0.33	0.35	6.1
DS 7	0.44	0.40	-9.1
DS 8	0.73	0.35	-52.1
DS 9	0.55	0.59	7.3
DS 10	0.39	0.39	0.0
DS 11	0.42	0.39	-7.1
DS 12	0.47	0.16	-66.0
DS 13	0.40	0.42	5.0
DS 14	0.66	0.61	-7.6
DS 15	0.28	0.19	-32.1
AVERAGE	**0.40**	**0.38**	**8.0**
SD	**0.17**	**0.15**	**59.2**
L 16	0.60	0.73	21.7
L 17	0.62	0.51	-17.7
L 18	0.34	0.46	35.3
L 19	0.21	0.23	9.5
L 20	0.45	0.43	-4.4
L 21	0.46	0.21	-54.3
L 22	0.30	0.90	200*
L 23	0.56	0.50	-10.7
L 24	0.30	0.24	-20.0
L 25	0.31	0.24	-22.6
L 26	0.62	0.57	-8.1
L 27	0.66	0.48	-27.3
L 28	0.45	0.25	-44.4
L 29	0.66	0.60	-9.1
AVERAGE	**0.47**	**0.45**	**3.4**
SD	**0.15**	**0.21**	**61.3**

Table A3: Hthreshold/Mthreshold

Participant	Pre	Post	Percent Change
DS 1	0.75	0.82	9.4
DS 2	0.84	0.74	-11.6
DS 3	1.05	1.08	2.8
DS 4	0.92	0.91	-1.0
DS 5	0.88	0.79	-10.8
DS 6	0.68	0.66	-2.9
DS 7	0.68	0.63	-7.5
DS 8	0.68	0.62	-9.0
DS 9	0.59	0.66	11.3
DS 10	0.73	0.59	-19.5
DS 11	0.66	0.59	-11.3
DS 12	0.72	0.79	10.4
DS 13	0.91	0.74	-19.5
DS 14	0.73	0.68	-6.3
DS 15	0.63	0.58	-8.8
AVERAGE	**0.77**	**0.73**	**-5.0**
SD	**0.13**	**0.14**	**9.89**
L 16	0.67	0.95	42.4
L 17	0.67	0.78	15.9
L 18	0.60	0.57	-5.8
L 19	0.76	0.69	-10.1
L 20	0.83	0.80	-3.5
L 21	0.80	0.83	4.4
L 22	0.89	0.95	7.2
L 23	0.82	0.73	-11.1
L 24	0.86	0.84	-2.7
L 25	0.73	0.75	2.8
L 26	0.81	0.84	3.7
L 27	0.79	0.78	-0.6
L 28	0.92	0.94	2.4
L 29	0.73	0.75	2.3
AVERAGE	**0.78**	**0.80**	**3.4**
SD	**0.09**	**0.11**	**13.23**

Table A4: Conditioned H Reflex

Participant	Pre	Post	Percent Change
DS 1	107.52	100.12	-6.9
DS 2	67.14	111.32	65.8
DS 3	107.06	71.75	-33.0
DS 4	105.05	99.40	-5.4
DS 5	86.17	139.21	61.6
DS 6	90.03	114.29	26.9
DS 7	97.33	94.67	-2.7
DS 8	98.36	91.83	-6.6
DS 9	106.06	79.24	-25.3
DS 10	100.95	121.74	20.6
DS 11	113.05	99.78	-11.7
DS 12	104.02	67.53	-35.1
DS 13	115.41	113.37	-1.8
DS 14	94.40	112.09	18.7
DS 15	102.76	97.67	-5.0
AVERAGE	**99.69**	**100.93**	**4.0**
SD	**11.99**	**18.97**	**30.0**
L 16	126.29	83.58	-33.8
L 17	94.68	88.68	-6.3
L 18	104.16	92.83	-10.9
L 19	116.16	78.74	-32.2
L 20	103.28	96.65	-6.4
L 21	81.30	41.54	-48.9
L 22	126.15	156.27	23.9
L 23	126.18	117.25	-7.1
L 24	109.18	121.06	10.9
L 25	95.80	98.35	2.7
L 26	115.25	99.43	-13.7
L 27	117.87	125.75	6.7
L 28	122.17	115.90	-5.1
L 29	100.23	107.04	6.8
AVERAGE	**109.91**	**101.65**	**-8.1**
SD	**13.87**	**26.56**	**19.4**

Table A5: Margin of Stability in AP Direction During Quiet Standing

	Pre		**Post**		**Percent Change**	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	6.04	6.60	5.47	6.51	-0.10	-0.01
DS 2	6.13	6.95	6.43	8.09	0.05	0.14
DS 3	6.45	5.84	5.47	6.17	-0.18	0.05
DS 4	4.03	6.40	5.36	4.97	0.25	-0.29
DS 5	6.86	10.54	5.34	10.05	-0.28	-0.05
DS 6	5.98	9.25	9.62	11.22	0.38	0.18
DS 7	20.77	22.28	24.12	33.14	0.14	0.33
DS 8	4.80	5.20	6.22	6.95	0.23	0.25
DS 9	16.74	20.39	8.01	15.42	-1.09	-0.32
DS 10	5.29	7.47	5.68	8.11	0.07	0.08
DS 11	5.88	7.07	5.79	8.41	-0.01	0.16
DS 12	4.46	4.73	7.35	6.46	0.39	0.27
DS 13	6.02	7.08	6.24	7.19	0.17	0.01
DS 14	4.97	5.91	6.52	6.71	0.24	0.12
DS 15	6.20	7.02	6.23	6.27	0.00	-0.12
AVERAGE	**8.18**	**8.85**	**7.66**	**9.71**	**0.02**	**0.05**
L 16	4.29	3.98	7.42	7.13	0.42	0.44
L 17	4.91	5.04	8.73	6.89	0.44	0.27
L 18	5.02	10.08	7.01	8.25	0.28	-0.22
L 19	4.54	5.90	5.83	9.64	0.22	0.39
L 20	6.94	8.46	11.95	9.73	0.42	0.13
L 21	5.57	4.26	4.76	4.73	-0.17	0.10
L 22	6.58	9.04	5.10	9.01	-0.29	0.00
L 23	8.27	7.26	9.66	7.46	0.14	0.03
L 24	8.37	8.20	12.31	14.18	0.32	0.42
L 25	5.62	4.62	9.36	4.74	0.40	0.03
L 26	10.63	10.51	10.67	18.16	0.00	0.42
L 27	9.46	9.13	10.77	6.70	0.12	-0.36
L 28	4.05	8.12	5.17	6.71	0.21	-0.21
L 29	5.36	4.97	6.29	6.92	0.15	0.28
L 30	8.22	7.42	8.98	10.43	0.08	0.29
AVERAGE	**6.52**	**7.13**	**8.27**	**8.71**	**0.18**	**0.13**

Table A6: Margin of Stability in ML Direction During Quiet Standing

	Pre		Post		Percent Change	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	1.59	2.13	2.92	3.10	0.46	0.31
DS 2	1.63	1.32	3.89	4.47	0.58	0.70
DS 3	1.38	1.35	1.43	4.22	0.04	0.68
DS 4	1.93	2.20	1.99	2.11	0.03	-0.04
DS 5	2.51	2.30	3.55	1.81	0.29	-0.27
DS 6	2.49	2.45	4.97	5.10	0.50	0.52
DS 7	6.81	4.48	7.79	6.21	0.12	0.28
DS 8	1.57	1.54	2.27	1.76	0.31	0.12
DS 9	5.24	7.64	5.32	4.34	0.02	-0.76
DS 10	1.86	2.02	2.43	2.51	0.23	0.20
DS 11	2.83	2.74	2.48	2.19	-0.14	-0.25
DS 12	2.43	1.59	3.09	2.27	0.22	0.30
DS 13	1.96	2.03	1.21	1.39	-0.62	-1.11
DS 14	2.02	3.03	2.47	3.03	0.19	0.00
DS 15	2.13	2.03	2.66	2.02	0.20	-0.01
AVERAGE	**2.56**	**2.65**	**3.23**	**3.10**	**0.16**	**0.04**
L 16	1.10	1.63	1.70	1.61	0.35	-0.02
L 17	1.44	1.33	4.02	2.05	0.64	0.35
L 18	2.60	2.53	3.26	2.70	0.20	0.06
L 19	2.09	1.46	1.76	1.84	-0.19	0.21
L 20	3.43	3.21	5.13	8.23	0.33	0.61
L 21	2.15	1.49	1.58	1.62	-0.36	0.08
L 22	2.76	2.16	2.36	2.80	-0.17	0.23
L 23	6.05	3.76	3.68	3.62	-0.64	-0.04
L 24	3.99	2.90	5.84	4.80	0.32	0.40
L 25	2.13	1.69	4.72	2.11	0.55	0.20
L 26	4.18	3.05	15.72	11.71	0.73	0.74
L 27	2.15	2.14	3.97	3.13	0.46	0.32
L 28	1.72	1.71	1.79	2.61	0.04	0.35
L 29	1.64	1.31	4.05	1.68	0.59	0.22
L 30	3.89	3.72	4.80	3.50	0.19	-0.06
AVERAGE	**2.76**	**2.27**	**4.29**	**3.60**	**0.20**	**0.24**

Table A7: Percent Change of Margin of Stability in AP Across Steps 1-2

	S1		S2	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	-0.12	-0.03	-0.14	-0.16
DS 2	-0.01	0.17	-0.20	-0.02
DS 3	-0.20	0.16	-0.32	-0.17
DS 4	0.03	0.22	-0.08	0.19
DS 5	-0.10	-0.14	0.08	0.31
DS 6	0.02	-0.05	-0.06	-0.01
DS 7	-0.13	-0.31	-0.54	-0.11
DS 8	-0.01	0.09	0.09	-0.07
DS 9	0.00	0.15	-0.21	0.17
DS 10	-0.10	-0.03	-0.26	-0.07
DS 11	-0.21	-0.07	-0.11	-0.09
DS 12	0.01	0.11	0.05	0.01
DS 13	0.01	-0.01	0.04	0.04
DS 14	-0.10	0.00	-0.08	-0.06
DS 15	-0.16	-0.04	-0.28	-0.13
AVERAGE	**0.07**	**-0.01**	**0.14**	**0.01**
L 16	0.05	0.06	0.07	0.40
L 17	0.26	0.00	0.37	-0.55
L 18	-0.04	0.04	0.03	0.04
L 19	-0.07	0.05	-0.08	-0.08
L 20	0.00	0.06	0.17	-0.17
L 21	-0.11	0.06	-0.16	-0.01
L 22	0.11	0.03	0.22	0.22
L 23	0.05	-0.51	-0.24	0.04
L 24	0.12	0.02	-0.09	0.04
L 25	0.02	0.11	-0.05	-0.03
L 26	-0.01	0.02	0.00	0.03
L 27	0.19	0.13	0.18	-0.01
L 28	-0.07	0.15	-0.13	-0.07
L 29	-0.13	0.17	-0.03	-0.18
L 30	0.13	-0.02	0.10	0.15
AVERAGE	**0.03**	**0.02**	**0.02**	**-0.01**

Table A8: Pre and Post Test Values of Margin of Stability in AP Across Steps 1-2

	S1				S2			
	Pre		Post		Pre		Post	
Participant	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**
DS 1	96.82	77.96	86.78	75.79	66.94	56.67	58.72	48.76
DS 2	92.75	72.59	91.62	87.20	70.20	49.45	58.39	48.62
DS 3	108.33	86.72	90.63	102.79	64.85	63.42	49.10	54.23
DS 4	81.31	59.01	84.04	76.13	51.58	34.35	47.64	42.52
DS 5	83.62	75.42	75.70	65.97	43.98	53.92	47.68	78.01
DS 6	93.43	81.08	95.74	77.24	65.88	47.27	61.98	46.74
DS 7	69.73	52.36	61.82	39.91	60.83	35.16	39.49	31.79
DS 8	99.33	73.44	98.67	80.97	56.52	39.67	61.98	37.21
DS 9	113.43	96.83	112.91	113.52	90.80	40.73	75.20	49.08
DS 10	84.45	71.38	76.99	69.49	61.66	44.66	49.03	41.73
DS 11	75.95	52.67	62.79	49.39	54.50	39.87	49.05	36.62
DS 12	87.89	74.56	88.80	83.45	55.43	61.41	58.13	61.81
DS 13	101.04	78.69	102.33	77.95	63.69	42.39	66.09	44.29
DS 14	88.08	69.86	80.09	69.86	77.23	50.05	71.80	47.13
DS 15	103.97	77.69	89.91	74.42	79.32	38.69	62.18	34.18
AVERAGE	**92.01**	**73.35**	**86.59**	**76.27**	**64.23**	**46.51**	**57.10**	**46.85**
L 16	109.52	88.47	115.72	93.89	61.79	28.55	66.73	47.65
L 17	94.91	114.18	127.86	114.51	54.25	101.56	86.63	65.37
L 18	80.41	63.49	77.08	66.20	47.00	44.93	48.62	46.99
L 19	93.12	79.51	87.16	83.85	45.33	38.60	41.90	35.85
L 20	93.02	71.64	93.00	75.85	41.67	46.35	50.03	39.69
L 21	87.03	69.88	78.09	74.59	48.54	43.19	41.74	42.58
L 22	105.62	82.47	118.44	84.75	64.42	48.74	82.21	62.55
L 23	51.64	83.99	54.50	55.64	39.27	31.44	31.61	32.73
L 24	92.93	79.41	105.35	80.98	59.46	37.92	54.61	39.31
L 25	71.74	68.22	73.43	76.76	35.89	36.37	34.06	35.38
L 26	95.36	86.09	94.02	88.05	71.57	59.82	71.39	61.53
L 27	71.25	63.47	88.41	73.10	55.05	47.69	67.18	47.32
L 28	129.95	101.50	121.52	118.78	88.92	58.28	79.02	54.30
L 29	73.89	54.20	65.38	65.57	43.25	44.22	41.87	37.62
L 30	58.92	52.80	67.39	51.67	58.01	49.38	64.46	57.91
AVERAGE	**87.29**	**77.29**	**91.16**	**80.28**	**54.30**	**47.80**	**57.47**	**47.12**

Table A9: Percent Change of Margin of Stability in ML Across Steps 1-2

	S1		S2	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	0.12	0.05	0.42	0.17
DS 2	0.15	0.27	-0.04	0.42
DS 3	0.03	-0.44	-0.40	0.23
DS 4	0.04	-0.04	0.14	-0.14
DS 5	0.12	-0.38	-0.38	-0.02
DS 6	-0.02	-0.05	0.04	-0.06
DS 7	0.14	0.04	-0.04	-0.01
DS 8	-0.03	-0.47	-0.06	-0.29
DS 9	0.20	-0.09	-0.02	0.21
DS 10	0.01	-0.29	0.10	0.12
DS 11	0.07	-0.15	-0.15	-0.06
DS 12	0.13	-0.20	0.03	0.21
DS 13	-0.17	-0.24	-0.19	-0.18
DS 14	0.23	-0.44	-0.45	0.14
DS 15	0.08	-0.04	0.06	0.01
AVERAGE	**0.07**	**-0.17**	**-0.06**	**0.05**
L 16	-0.08	-0.05	-0.10	-0.15
L 17	-0.43	0.16	-0.24	0.12
L 18	0.19	0.34	-0.59	-0.59
L 19	-0.12	-0.29	-0.08	0.34
L 20	-0.30	0.00	-0.41	-0.56
L 21	-0.21	-0.35	0.01	-0.14
L 22	-0.24	-0.13	-0.30	0.12
L 23	0.08	-0.56	0.13	-0.27
L 24	-0.12	-0.20	0.02	0.17
L 25	0.14	0.16	-0.13	0.13
L 26	0.01	-0.15	-0.23	0.26
L 27	0.09	-0.08	-2.12	-0.67
L 28	-0.05	-0.26	-0.26	-0.32
L 29	-0.07	-0.14	-0.15	-0.04
L 30	-0.08	0.04	0.10	0.32
AVERAGE	**-0.08**	**-0.10**	**-0.29**	**-0.09**

Table A10: Pre and Post Test Values of Margin of Stability in ML Across Steps 1-2

	S1				S2			
	Pre		Post		Pre		Post	
Participant	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**
DS 1	19.13	21.60	21.74	22.81	1.61	2.83	2.80	3.43
DS 2	19.23	17.61	22.72	24.16	5.64	4.72	5.44	8.12
DS 3	14.97	21.94	15.47	15.23	6.77	5.80	4.83	7.55
DS 4	19.05	20.38	19.88	19.60	12.16	13.86	14.15	12.21
DS 5	15.52	26.71	17.54	19.38	10.88	8.78	7.91	8.65
DS 6	12.65	12.94	12.43	12.30	15.36	12.39	16.04	11.65
DS 7	15.28	16.98	17.82	17.64	7.53	10.90	7.23	10.80
DS 8	14.03	18.85	13.57	12.83	5.53	8.94	5.22	6.92
DS 9	17.00	20.82	21.21	19.02	11.12	11.97	10.88	15.18
DS 10	18.05	17.57	18.24	13.66	10.80	13.24	12.04	14.96
DS 11	17.68	17.54	19.04	15.26	7.92	19.55	6.91	18.53
DS 12	13.28	18.08	15.28	15.03	10.01	10.00	10.29	12.60
DS 13	13.77	16.16	11.81	12.99	8.31	5.08	6.99	4.32
DS 14	15.84	21.73	20.50	15.05	13.43	9.83	9.28	11.42
DS 15	18.35	19.11	19.92	18.31	10.80	11.92	11.48	12.05
AVERAGE	**16.25**	**19.20**	**17.81**	**16.88**	**9.19**	**9.99**	**8.77**	**10.56**
L 16	12.67	14.85	11.71	14.08	10.20	12.43	9.28	10.79
L 17	22.57	16.39	15.77	19.52	10.16	10.70	8.22	12.14
L 18	10.20	10.87	12.59	16.39	10.22	11.56	6.43	7.28
L 19	14.32	17.15	12.73	13.31	11.08	8.16	10.25	12.38
L 20	19.29	15.07	14.83	15.05	9.53	18.69	6.77	12.00
L 21	19.16	22.90	15.80	16.93	9.27	10.89	9.33	9.55
L 22	17.40	15.23	14.06	13.53	12.54	16.05	9.63	18.15
L 23	15.18	19.19	16.50	12.32	4.93	9.84	5.67	7.75
L 24	17.40	19.03	15.50	15.92	11.07	11.63	11.34	14.06
L 25	14.79	15.05	17.27	17.89	9.44	6.02	8.38	6.94
L 26	15.61	17.25	15.76	14.95	11.31	7.63	9.23	10.25
L 27	18.87	22.29	20.75	20.56	10.28	12.14	3.29	7.26
L 28	19.31	20.86	18.35	16.61	8.44	8.82	6.71	6.66
L 29	12.93	16.57	12.03	14.60	11.31	10.75	9.85	10.36
L 30	19.20	20.28	17.73	21.16	14.82	15.45	16.47	22.57
AVERAGE	**16.59**	**17.53**	**15.43**	**16.19**	**10.31**	**11.38**	**8.72**	**11.21**

Table A11: Percent Change of Step Length

	S1		S2	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	-0.03	-0.04	-0.03	-0.02
DS 2	-0.04	0.22	-0.02	0.19
DS 3	-0.02	0.04	-0.02	0.05
DS 4	0.03	0.07	0.01	0.08
DS 5	-0.10	-0.02	-0.06	-0.03
DS 6	0.01	-0.01	-0.06	-0.01
DS 7	-0.11	-0.22	-0.03	0.00
DS 8	0.00	0.05	-0.02	-0.04
DS 9	-0.02	0.09	-0.09	-0.01
DS 10	-0.10	0.03	0.00	-0.03
DS 11	-0.07	-0.02	0.00	0.00
DS 12	0.04	0.01	0.02	0.03
DS 13	-0.01	0.04	0.02	0.00
DS 14	-0.16	-0.13	0.01	0.01
DS 15	0.03	0.11	-0.04	-0.04
AVERAGE	**-3.66%**	**1.36%**	**-2.11%**	**1.15%**
L 16	0.02	0.00	0.01	0.02
L 17	0.06	0.02	0.07	-0.04
L 18	-0.03	-0.07	0.03	0.05
L 19	-0.05	-0.06	0.03	0.06
L 20	0.00	0.01	-0.01	0.08
L 21	-0.01	-0.02	-0.01	0.01
L 22	0.04	-0.03	0.01	0.06
L 23	0.00	-0.18	-0.01	-0.01
L 24	0.08	0.02	0.06	0.08
L 25	-0.01	0.01	0.02	0.05
L 26	0.02	0.03	0.00	0.03
L 27	0.02	0.05	0.04	0.04
L 28	-0.01	0.01	-0.03	0.03
L 29	-0.08	0.09	-0.02	0.06
L 30	-0.01	-0.07	0.03	0.00
AVERAGE	**0.28%**	**-1.32%**	**1.42%**	**3.29%**

Table A12: Pre and Post Test Values of Step Length

	S1				S2			
	Pre		Post		Pre		Post	
Participant	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**
DS 1	704.40	654.04	683.40	626.77	748.00	675.56	729.61	661.81
DS 2	766.50	652.52	739.11	839.13	832.07	635.04	814.53	780.25
DS 3	789.16	756.39	772.21	789.24	798.32	760.73	781.02	798.60
DS 4	627.12	565.29	649.78	607.37	657.51	549.93	662.87	598.25
DS 5	680.14	580.71	619.95	567.69	744.00	632.84	699.84	615.19
DS 6	706.21	711.20	714.98	704.45	725.41	658.52	686.53	649.70
DS 7	613.56	568.36	550.92	465.06	686.16	585.97	664.39	586.49
DS 8	701.99	674.17	702.34	708.24	722.77	671.19	707.82	646.02
DS 9	745.33	747.73	734.16	818.22	858.81	735.37	785.33	724.99
DS 10	681.22	619.26	617.28	636.02	728.47	672.91	728.56	650.37
DS 11	620.58	565.45	579.79	552.17	651.49	545.73	649.91	546.59
DS 12	636.67	646.57	662.68	653.68	665.13	600.30	679.39	621.68
DS 13	673.92	639.53	664.73	664.21	713.02	649.24	729.25	651.51
DS 14	601.58	574.41	517.15	508.26	642.84	566.92	647.18	571.41
DS 15	637.46	589.05	658.10	664.82	668.18	543.91	644.56	523.52
AVERAGE	**679.05**	**636.31**	**657.77**	**653.69**	**722.81**	**632.28**	**707.39**	**641.76**
L 16	765.97	770.12	781.68	770.49	725.58	654.29	729.89	666.27
L 17	708.96	741.04	756.12	753.87	741.54	792.85	797.53	760.46
L 18	637.37	630.97	618.73	591.96	616.00	580.29	631.95	610.41
L 19	715.73	725.06	680.53	686.70	672.73	622.70	694.08	659.57
L 20	735.28	694.98	732.66	700.47	757.56	634.61	752.38	692.58
L 21	728.65	686.05	721.31	672.42	735.36	701.67	729.38	706.79
L 22	694.14	732.31	723.78	711.25	690.06	632.21	698.55	673.52
L 23	454.28	597.88	453.83	504.76	593.82	555.50	586.67	549.76
L 24	671.08	684.61	731.27	698.74	707.93	623.07	752.11	678.65
L 25	750.19	733.92	744.23	740.02	704.91	672.77	721.73	705.58
L 26	637.92	611.28	653.09	627.71	727.14	686.35	723.53	707.22
L 27	610.84	586.99	621.50	619.88	709.08	658.42	741.79	682.59
L 28	732.45	690.72	722.35	694.57	788.89	691.49	763.32	709.64
L 29	711.53	607.69	661.65	664.75	690.69	626.57	679.40	664.19
L 30	464.87	433.03	460.11	406.21	566.54	520.07	581.46	517.92
AVERAGE	**667.95**	**661.78**	**670.86**	**656.25**	**695.19**	**643.52**	**705.58**	**665.68**

Table A13: Percent Change of Step Length Variability

	S1		S2	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	0.43	-0.40	-1.15	-1.08
DS 2	-0.29	-0.42	0.23	0.16
DS 3	-0.67	-0.50	0.65	-1.94
DS 4	-0.47	0.40	-1.06	-1.75
DS 5	-1.19	0.19	0.11	-1.14
DS 6	-0.22	-1.92	0.18	-0.36
DS 7	0.59	-0.52	-0.02	-0.74
DS 8	0.43	0.27	-0.52	-0.66
DS 9	0.50	0.52	-0.07	-0.16
DS 10	0.11	0.26	-0.21	-0.56
DS 11	0.43	0.53	-0.12	0.38
DS 12	0.57	-1.18	-2.44	-0.71
DS 13	0.48	0.01	0.32	0.03
DS 14	-0.54	0.65	0.20	-1.14
DS 15	0.25	0.29	-0.78	-0.25
AVERAGE	**2.7%**	**-12.17%**	**-31.31%**	**-66.29%**
L 16	-1.13	-1.19	-3.23	-0.02
L 17	-0.31	0.20	-1.60	0.39
L 18	0.11	0.04	0.35	-0.40
L 19	-0.74	-1.47	-2.78	-0.07
L 20	-0.40	-0.42	-0.56	-0.24
L 21	-0.01	-0.56	0.02	-1.19
L 22	0.16	-1.79	-0.08	0.06
L 23	-0.61	-0.76	0.17	-1.48
L 24	0.20	-0.56	0.35	-0.91
L 25	0.53	-0.66	-0.18	0.35
L 26	0.55	0.30	-0.13	0.46
L 27	-0.54	-0.09	0.37	-0.37
L 28	-1.45	-1.07	-0.48	-0.15
L 29	0.61	0.05	0.19	-0.41
L 30	0.50	0.52	-0.50	0.26
AVERAGE	**-17.05%**	**-49.69%**	**-54.01%**	**-24.88%**

Table A14: Pre and Post Test Values of Step Length Variability

	S1				S2			
	Pre		Post		Pre		Post	
Participant	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**
DS 1	20.58	57.60	36.09	41.23	33.30	55.57	15.47	26.68
DS 2	33.59	62.68	25.95	44.22	27.88	32.23	36.17	38.19
DS 3	34.34	37.10	20.59	24.70	20.30	38.46	57.62	13.08
DS 4	41.00	22.02	27.87	36.81	48.95	56.60	23.75	20.58
DS 5	24.52	53.77	11.21	66.24	31.01	84.20	34.67	39.26
DS 6	36.85	34.31	30.29	11.74	29.11	31.18	35.44	23.00
DS 7	25.27	33.63	61.23	22.10	49.36	41.67	48.17	23.90
DS 8	7.88	35.23	13.86	48.00	22.97	30.96	15.11	18.59
DS 9	23.07	23.88	45.77	49.50	61.97	56.55	57.68	48.60
DS 10	19.83	34.04	22.26	45.86	27.59	26.83	22.88	17.15
DS 11	31.33	38.89	55.45	83.23	23.37	24.69	20.83	40.10
DS 12	16.40	40.87	38.29	18.78	28.73	43.10	8.34	25.19
DS 13	14.93	29.91	28.55	30.27	21.73	20.81	32.17	21.39
DS 14	73.68	24.56	47.80	70.02	43.21	48.96	54.19	22.90
DS 15	39.88	24.16	52.84	33.84	22.58	28.42	12.68	22.72
AVERAGE	**29.54**	**36.84**	**34.54**	**41.77**	**32.80**	**41.35**	**31.68**	**26.76**
L 16	37.94	40.99	17.79	18.71	29.73	26.67	7.02	26.04
L 17	43.50	37.62	33.08	46.73	36.16	14.75	13.91	24.05
L 18	23.79	30.02	26.65	31.38	19.63	43.36	30.17	30.86
L 19	32.87	41.75	18.88	16.93	27.24	55.89	7.21	52.09
L 20	33.09	77.79	23.60	54.89	37.23	46.89	23.81	37.78
L 21	31.68	33.66	31.31	21.53	21.40	40.41	21.79	18.44
L 22	16.02	69.03	18.98	24.77	25.92	22.19	23.97	23.65
L 23	49.06	100.83	30.40	57.33	26.14	21.90	31.34	8.85
L 24	27.91	64.61	34.70	41.52	35.17	89.65	53.83	46.86
L 25	9.80	36.33	20.92	21.92	20.36	21.77	17.26	33.29
L 26	14.66	26.70	32.45	37.94	35.67	16.58	31.67	30.52
L 27	32.72	41.13	21.26	37.83	19.55	37.18	31.16	27.09
L 28	17.19	42.22	7.01	20.37	35.49	24.40	24.01	21.28
L 29	18.97	60.86	49.27	64.04	19.67	65.78	24.42	46.80
L 30	18.13	20.26	36.13	42.14	24.99	23.32	16.61	31.70
AVERAGE	**27.16**	**48.25**	**26.83**	**35.87**	**27.62**	**36.71**	**23.88**	**30.62**

Table A15: Percent Change Step Width

	S1		S2	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	-0.41	-0.84	-0.09	0.29
DS 2	0.01	0.28	-0.05	0.37
DS 3	-0.29	-0.29	-0.71	0.26
DS 4	0.25	-0.09	0.20	-0.15
DS 5	-0.14	0.13	-0.23	0.33
DS 6	0.08	-0.17	0.11	0.06
DS 7	-0.23	-0.23	-0.17	-0.04
DS 8	-0.09	-0.24	-0.23	-0.15
DS 9	-0.17	-0.51	-0.09	0.20
DS 10	-0.10	-0.45	-0.01	0.19
DS 11	0.22	-0.16	0.10	0.13
DS 12	0.42	0.02	0.06	0.13
DS 13	0.14	0.09	-0.14	-0.34
DS 14	-0.21	-1.03	-0.31	0.01
DS 15	0.11	0.22	0.09	0.19
AVERAGE	**-2.74%**	**-21.86%**	**-9.81%**	**9.89%**
L 16	-0.07	-0.02	-0.07	-0.22
L 17	-0.45	-0.16	-0.01	0.05
L 18	-0.05	0.25	-0.12	-0.43
L 19	0.09	0.15	-0.01	0.22
L 20	-0.59	0.48	-0.50	-0.47
L 21	-0.16	-0.52	-0.28	-0.30
L 22	-0.32	-0.18	-0.01	0.08
L 23	-0.12	-0.47	0.02	-0.43
L 24	-0.02	0.27	0.19	0.22
L 25	0.09	0.02	-0.05	-0.11
L 26	-0.21	0.08	-0.23	0.20
L 27	0.04	0.05	-0.54	-0.29
L 28	-0.15	-1.36	-0.32	-0.17
L 29	-0.01	-0.19	-0.10	-0.11
L 30	-0.18	0.14	-0.05	0.10
AVERAGE	**-14.08%**	**-9.73%**	**-13.85%**	**-11.16%**

Table A16: Pre and Post Test Values of Step Width

	S1				S2			
	Pre		Post		Pre		Post	
Participant	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**
DS 1	63.50	59.32	45.05	32.26	56.89	85.26	51.97	120.81
DS 2	146.65	107.39	148.72	148.52	133.67	95.55	127.69	151.43
DS 3	118.58	156.01	92.26	120.84	130.70	91.17	76.48	123.33
DS 4	83.54	95.59	111.17	87.57	141.91	205.25	178.33	178.37
DS 5	82.63	76.92	72.37	88.28	161.19	100.55	130.99	151.16
DS 6	114.04	87.10	124.14	74.19	231.34	218.61	259.91	232.87
DS 7	124.86	138.32	101.47	112.83	164.71	176.15	140.61	169.66
DS 8	98.12	139.73	89.95	112.89	154.21	190.08	125.22	164.77
DS 9	174.28	164.18	148.83	108.91	157.71	177.85	144.24	222.00
DS 10	138.52	121.96	126.07	83.86	183.77	206.73	181.49	256.05
DS 11	89.13	69.75	113.56	60.17	127.17	216.79	141.18	247.90
DS 12	46.72	85.70	80.59	87.79	152.80	177.26	162.58	204.25
DS 13	62.15	68.05	71.99	74.82	134.54	142.76	118.29	106.25
DS 14	124.48	142.62	102.76	70.12	186.99	175.63	142.48	176.85
DS 15	127.50	104.08	143.52	132.70	169.86	179.27	187.67	222.18
AVERAGE	**106.31**	**107.78**	**104.83**	**93.05**	**152.50**	**162.59**	**144.61**	**181.86**
L 16	105.86	115.81	99.03	113.84	197.40	216.51	183.89	177.39
L 17	141.69	121.85	97.45	104.88	167.29	187.42	165.22	197.64
L 18	103.03	102.40	97.86	137.33	140.55	190.64	125.81	133.10
L 19	87.96	92.93	96.35	109.58	174.88	172.93	173.54	221.08
L 20	114.66	36.01	72.18	69.26	164.82	245.81	109.96	167.46
L 21	90.10	134.44	77.65	88.63	152.10	178.59	118.74	137.83
L 22	102.48	90.54	77.87	76.54	206.18	252.90	205.02	274.56
L 23	78.06	62.62	69.64	42.72	105.79	147.35	108.02	102.69
L 24	109.51	86.01	107.65	117.51	139.47	156.41	171.76	199.41
L 25	100.71	75.15	110.31	76.74	140.15	133.63	133.06	120.57
L 26	107.07	96.64	88.64	104.76	189.77	153.57	153.77	192.73
L 27	101.09	102.14	104.89	107.11	143.33	173.52	93.17	134.24
L 28	83.30	81.52	72.39	34.47	168.85	178.67	128.10	152.32
L 29	102.16	126.39	101.40	105.95	198.19	182.02	180.91	163.74
L 30	119.58	110.03	101.55	128.55	237.06	247.01	225.73	273.21
AVERAGE	**103.15**	**95.63**	**91.66**	**94.53**	**168.39**	**187.80**	**151.78**	**176.53**

Table A17: Percent Change Step Width Variability

	S1		S2	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	-0.10	-1.65	-0.29	-0.11
DS 2	0.30	-0.03	0.08	0.58
DS 3	0.32	-0.19	-0.25	0.32
DS 4	-0.34	0.60	0.59	0.73
DS 5	-1.98	-0.64	0.28	-0.58
DS 6	0.24	0.42	0.14	-0.69
DS 7	0.17	-1.62	0.34	-0.07
DS 8	-1.20	-0.98	-0.76	0.14
DS 9	0.55	0.17	0.06	0.07
DS 10	0.52	0.53	-0.69	-0.55
DS 11	0.43	0.69	0.32	0.06
DS 12	0.45	-0.54	-0.63	-0.63
DS 13	-0.22	-0.02	0.06	-0.36
DS 14	0.16	0.07	-1.17	0.31
DS 15	0.53	0.13	0.13	-0.83
AVERAGE	**-1.19%**	**-20.55%**	**-12.0%**	**-10.68%**
L 16	0.07	-1.46	0.53	0.13
L 17	-1.33	0.40	-3.00	0.27
L 18	0.23	0.40	-0.05	-0.24
L 19	0.07	-0.10	-7.94	-0.97
L 20	-0.46	0.55	0.16	0.43
L 21	-0.41	0.17	0.56	-0.96
L 22	-0.20	-0.11	0.12	0.67
L 23	0.43	-0.63	-0.06	-2.12
L 24	0.39	0.47	-0.05	0.36
L 25	0.33	0.42	-0.09	0.54
L 26	-0.21	-0.42	-1.54	-1.17
L 27	0.42	0.15	0.25	0.52
L 28	-1.66	-1.05	-0.13	0.15
L 29	0.57	0.36	-0.06	-0.05
L 30	0.42	0.34	-0.20	-0.51
AVERAGE	**-9.03%**	**-3.43%**	**-76.60%**	**-19.62%**

Table A18: Pre and Post Test Values of Step Width Variability

	S1				S2			
	Pre		Post		Pre		Post	
Participant	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**	**EO**	**EC**
DS 1	18.69	49.87	16.94	18.79	24.66	30.89	19.13	27.75
DS 2	12.61	26.91	17.90	26.02	27.80	24.81	30.18	59.14
DS 3	25.07	21.45	37.03	17.96	35.31	39.92	28.20	58.72
DS 4	23.55	11.73	17.63	29.08	16.95	17.03	41.03	63.77
DS 5	16.62	42.99	5.57	26.17	25.57	82.05	35.57	52.06
DS 6	12.66	23.62	16.58	40.58	29.37	38.26	34.13	22.65
DS 7	14.72	38.27	17.70	14.63	18.52	26.26	27.95	24.53
DS 8	32.99	58.59	14.99	29.60	35.57	29.86	20.16	34.68
DS 9	11.17	61.33	24.83	73.49	44.20	45.81	46.95	49.30
DS 10	11.15	13.64	23.12	29.12	39.26	19.63	23.27	12.68
DS 11	15.33	14.60	26.87	46.69	35.55	35.55	52.61	38.02
DS 12	8.62	31.80	15.76	20.59	19.94	44.67	12.20	27.46
DS 13	9.97	22.89	8.20	22.45	13.82	45.91	14.67	33.74
DS 14	13.29	34.50	15.77	37.05	32.96	24.41	15.20	35.32
DS 15	5.86	42.44	12.46	49.02	21.09	27.95	24.30	15.24
AVERAGE	**15.49**	**32.98**	**18.09**	**32.08**	**28.04**	**35.54**	**28.37**	**37.00**
L 16	9.36	25.06	10.06	10.20	11.48	24.83	24.18	28.57
L 17	39.92	17.01	17.15	28.12	49.05	30.84	12.25	42.15
L 18	18.16	14.20	23.62	23.70	25.79	60.11	24.57	48.54
L 19	15.76	37.48	16.93	33.98	63.24	68.87	7.07	34.89
L 20	13.40	21.57	9.15	48.24	29.41	27.50	34.98	48.05
L 21	15.11	21.71	10.69	26.03	13.74	48.46	31.42	24.72
L 22	16.22	37.21	13.56	33.66	20.06	17.78	22.69	54.23
L 23	21.44	56.69	37.72	34.80	19.57	60.09	18.53	19.27
L 24	15.37	21.14	25.09	40.20	24.35	32.49	23.16	50.84
L 25	8.87	26.50	13.22	45.51	23.80	34.08	21.94	74.05
L 26	23.82	23.62	19.64	16.66	41.23	55.85	16.24	25.73
L 27	17.31	15.53	29.91	18.27	25.41	23.38	33.80	49.09
L 28	20.08	33.17	7.54	16.16	21.05	33.68	18.70	39.77
L 29	10.04	24.43	23.18	38.13	24.32	27.61	23.04	26.42
L 30	14.32	20.90	24.59	31.45	22.18	30.72	18.52	20.29
AVERAGE	**17.28**	**26.41**	**18.80**	**29.67**	**27.64**	**38.42**	**22.07**	**39.11**

Table A19: ML COM Variability (mm)

	Pre		Post		Percent Change	
Participant	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed	Eyes Open	Eyes Closed
DS 1	72.29	73.16	88.37	76.71	0.18	0.05
DS 2	57.28	54.37	64.40	66.64	0.11	0.18
DS 3	47.42	87.80	49.77	44.62	0.05	-0.97
DS 4	40.65	41.91	39.87	45.60	-0.02	0.08
DS 5	32.16	58.09	36.65	49.73	0.12	-0.17
DS 6	31.12	34.32	31.84	34.60	0.02	0.01
DS 7	41.39	44.20	39.48	38.75	-0.05	-0.14
DS 8	36.40	45.30	40.89	44.28	0.11	-0.02
DS 9	53.47	62.65	59.31	52.47	0.10	-0.19
DS 10	50.93	45.08	45.03	39.08	-0.13	-0.15
DS 11	48.11	40.29	53.15	42.65	0.09	0.06
DS 12	24.11	40.45	29.22	33.84	0.17	-0.20
DS 13	21.03	33.97	23.23	35.54	0.09	0.04
DS 14	37.44	54.33	39.73	30.29	0.06	-0.79
DS 15	42.30	43.99	44.20	41.90	0.04	-0.05
AVERAGE	**42.41**	**50.66**	**45.68**	**45.11**	**0.06**	**-0.15**
L 16	28.34	32.86	26.50	34.22	-0.07	0.04
L 17	58.75	37.52	34.03	39.09	-0.73	0.04
L 18	22.46	29.45	22.96	36.86	0.02	0.20
L 19	30.06	39.42	29.23	37.02	-0.03	-0.06
L 20	49.76	39.16	45.67	40.52	-0.09	0.03
L 21	37.90	52.46	42.02	43.04	0.10	-0.22
L 22	32.84	36.04	26.74	42.91	-0.23	0.16
L 23	34.65	34.62	33.02	26.31	-0.05	-0.32
L 24	40.61	44.94	34.86	40.93	-0.16	-0.10
L 25	38.28	39.45	44.70	49.05	0.14	0.20
L 26	35.51	44.38	34.79	33.12	-0.02	-0.34
L 27	50.52	53.20	57.68	49.90	0.12	-0.07
L 28	34.19	40.35	35.02	30.54	0.02	-0.32
L 29	30.55	42.82	27.75	38.44	-0.10	-0.11
L 30	39.16	41.27	38.27	49.30	-0.02	0.16
AVERAGE	**37.57**	**40.53**	**35.55**	**39.42**	**-0.07**	**-0.05**

Table A20: Percent Change Centre of Pressure Displacement in AP and ML

	dCOP ML		dCOP AP	
Participant	EO	EC	EO	EC
DS 1	0.59	-2.37	0.23	-1.26
DS 2	0.68	0.36	0.08	-0.13
DS 3	0.40	0.19	0.01	0.02
DS 4	0.02	0.31	0.33	-0.29
DS 5	0.54	0.13	0.10	0.08
DS 6	-0.46	0.41	0.10	0.19
DS 7	-0.22	0.20	0.09	0.34
DS 8	-1.36	0.70	-0.63	0.41
DS 9	-0.14	-0.27	-0.82	-0.47
DS 10	-0.18	-0.88	-0.09	0.29
DS 11	0.60	-0.02	-0.21	0.23
DS 12	0.56	0.08	0.17	0.12
DS 13	-3.72	-3.78	0.07	-0.08
DS 14	0.18	-0.01	0.24	0.15
DS 15	0.39	0.38	0.58	0.86
AVERAGE	**-0.14**	**-0.30**	**0.02**	**0.03**
SD	**1.13**	**1.21**	**0.35**	**0.47**
L 16	0.21	0.11	0.22	0.32
L 17	0.41	0.24	0.43	0.12
L 18	0.45	0.12	0.45	-0.01
L 19	-0.16	0.59	0.16	0.36
L 20	0.31	0.60	0.30	0.15
L 21	-0.03	0.08	-0.05	0.09
L 22	0.16	0.47	-0.20	0.03
L 23	-0.23	0.45	-0.08	0.19
L 24	0.10	-0.59	0.28	0.21
L 25	-0.10	0.16	0.01	-0.02
L 26	0.44	0.83	-0.55	0.36
L 27	0.65	-0.67	0.14	-0.25
L 28	-0.17	0.59	0.20	0.13
L 29	0.60	0.64	0.39	0.11
L 30	0.16	-0.25	0.11	0.16
AVERAGE	**0.19**	**0.22**	**0.12**	**0.13**
SD	**0.29**	**0.45**	**0.27**	**0.16**

Table A21: Percent Change Centre of Pressure Velocities in AP and ML

	vCOP ML		**vCOP AP**	
Participant	EO	EC	EO	EC
DS 1	0.32	0.08	0.20	0.01
DS 2	0.23	0.15	0.17	0.15
DS 3	0.22	0.12	0.11	-0.01
DS 4	-0.16	0.55	0.14	0.08
DS 5	0.60	0.14	0.26	-0.03
DS 6	-1.12	0.57	0.08	0.18
DS 7	-1.01	0.16	0.21	0.18
DS 8	-2.40	0.71	-0.03	0.27
DS 9	-0.47	0.00	-2.61	-0.31
DS 10	0.59	-1.39	0.31	0.34
DS 11	0.50	0.32	-1.12	0.43
DS 12	0.56	0.22	0.10	0.11
DS 13	-3.54	-4.64	-0.19	-0.50
DS 14	0.24	0.03	0.33	0.24
DS 15	0.12	0.35	0.05	0.21
AVERAGE	**-0.35**	**-0.18**	**-0.13**	**0.09**
SD	**1.21**	**1.32**	**0.77**	**0.24**
L 16	0.02	-0.13	0.00	-0.18
L 17	0.13	-0.05	0.19	-0.20
L 18	0.49	0.24	0.55	0.01
L 19	-0.05	0.44	-0.06	0.01
L 20	0.12	0.31	0.04	0.24
L 21	0.00	0.18	-0.09	0.13
L 22	0.01	0.48	-0.03	0.11
L 23	0.08	0.53	0.02	0.20
L 24	0.49	-0.48	-0.05	-0.21
L 25	0.07	-0.06	-0.07	-0.11
L 26	-0.14	0.83	-0.91	0.19
L 27	0.25	-0.85	0.01	-0.29
L 28	-0.74	0.62	0.16	0.35
L 29	0.35	0.42	0.60	-0.01
L 30	0.10	-0.53	0.08	-0.20
AVERAGE	**0.08**	**0.13**	**0.03**	**0.00**
SD	**0.29**	**0.47**	**0.33**	**0.20**

Appendix B: Informed Consent Form

WILFRID LAURIER UNIVERSITY
INFORMED CONSENT STATEMENT

Title of project: Neural mechanisms of balance and gait adaptations after downslope walking

Primary investigator: Nikki Aitcheson-Huehn, BSc, Master's Student
Email: aitc8260@mylaurier.ca
Supervisors: Dr. Michael Cinelli (mcinelli@wlu.ca) and Dr. Jayne Kalmar (jkalmar@wlu.ca),
Associate Professors of Kinesiology & Physical Education

INFORMATION

You are invited to participate in a research study. This research study will take place in full at the Athletic Centre (AC1-56) at Wilfrid Laurier University. The purpose of this story is to (a) record changes in balance and gait that occur after walking downslope walking on a treadmill, and (b) identify the neural mechanism that makes motor neurons less excitable after downslope walking. Approximately 30 participants will be tested from WLU's student population between the ages of 18 and 26. To participate in this study, it is important that you do not have a diagnosed neurological disorder or musculoskeletal disease, no musculoskeletal injury within the past 90 days, no history of shin pain during exercise, or regularly participate in ballet or gymnastics (≥ 3x per week).

You will be asked to fill out a health history questionnaire prior to completing the protocol. During the protocol, you will be randomly assigned to walk on a treadmill that is either level with the ground or angled down to create the downslope. Before and after you walk on the treadmill, we will assess the excitability of the nerves that control a muscle on the back of your leg (the soleus). We will do this by measuring a reflex (H reflex) in response to nerve stimulation. To do this, we will apply a series of electrical pulses; the size of the pulse will gradually increase until it is strong enough to cause a brief contraction (a twitch) of your calf muscles. We will also apply a series of pairs of electrical pulses (one to a nerve behind your knee and one to a nerve on the side of your leg) that help us measure reflex pathway that helps to inhibit muscle activity. You will receive 10 pairs of pulses. This should take approximately 20 minutes. Next, you will be asked to stand quietly on a force plate for 10s, at which point you will be instructed to take a step with your right leg. There will be 10 trials of quiet standing and gait initiation (taking a step): 5 with eyes open and 5 with eyes closed (alternating trials). The visual condition will alternate with each trial. In total, the protocol will take less than 120 minutes to complete.

Participant's Initials: _____

RISKS

There are minimal risks in this study; you may get tired walking on the treadmill for 30-minutes. If tiredness occurs, the treadmill speed will be decreased. Depending on your fitness level, it is possible that the treadmill walking will induce mild muscle soreness that could last for 1 or 2 days after exercise. This soreness should not feel different than exercise-induced soreness that one feels after a gym workout. Additionally, you might experience slight discomfort or anxiety when your nerve is stimulated during collection of the spinal excitability measures. In this case, we can implement longer breaks between stimuli and you may ask to stop the experiment and withdraw from the study at any time if you do not wish to proceed.

BENEFITS

Downslope walking has been indicated to improve balance and gait in clinical populations, thus you may have temporary benefits to your balance control. Regardless, you will assist with understanding the persisting effects of downslope walking and their neural mechanism(s) to support recommendation of downslope walking in the clinic.

CONFIDENTIALITY

Identification of participants will be kept anonymous with a coding system understood by only the primary investigator and supervisors. The coding system will be used through data collection and only aggregate data will be published. All personal information about the participants and their test results will be kept separate to ensure protection of privacy; physical documentation will be kept in a locked cabinet and electronic data will be kept on a password protected computer in the laboratory. The collected information will only be accessible to the primary investigator and supervisors and will be erased and shredded after 7 years.

CONTACT

If you have questions at any time about the study or the procedures, (or you experience adverse effects as a result of participating in this study) you may contact the researchers, Nikki Aitcheson-Huehn (aitc8260@mylaurier.ca), Dr. Michael Cinelli (mcinelli@wlu.ca, 519-884-0710 ext. 4775), or Dr. Jayne Kalmar (jkalmar@wlu.ca, 519-884-0710 ext. 3334). This project has been reviewed and approved by the University Research Ethics Board (which receives funding from the Research Support Fund). If you feel you have not been treated according to the descriptions in this form, or your rights as a participant in research have been violated during the course of this project, you may contact Dr. Jayne Kalmar, Chair, University Research Ethics Board, Wilfrid Laurier University, 519-884-1970 ext. 3131 or jkalmar@wlu.ca.

Participant's Initials: _____

PARTICIPATION

Your participation in this study is voluntary; you may decline to participate without penalty. If you decide to participate, you may withdraw from the study at any time without penalty and without loss of benefits to which you are otherwise entitled. If you withdraw from the study, every attempt will be made to remove your data from the study, and have it destroyed. You have the right to omit any question(s)/procedure(s) you choose.

FEEDBACK AND PUBLICATION

Results will be available on the Lifespan Psychomotor Behavioural Lab website upon study completion. Results will also be presented at conferences and published in a relevant journal.

Participant's Initials: _____

CONSENT

I have read and understood the above information. I have received a copy of this form. I agree to participate in this study.

Participant's signature _____ Date: _____

Investigator's signature _____ Date: _____

Appendix C: Questionnaire for Young Adults

(a) Age (month/day/year): _____

(b) Gender: _____

(c) On average, how many days per week are you physically active: _____

(d) Would you consider yourself to engage in low, moderate, or high levels of physical activity: Low Moderate High

(e) Have you or do you regularly participate in ballet or gymnastics? Yes No

(f) How often do you exercise on a treadmill: Weekly Bi-Weekly Monthly N/A

(g) Do you have any neurological diseases or disorders: Yes No

(h) Do you have any musculoskeletal diseases or disorders: Yes No

(i) Have you had any musculoskeletal injuries in the past 12 months: Yes No

Appendix D: Borg Rating of Perceived Exertion (RPE)

Number	Level of Exertion
6	No exertion
7	
7.5	Extremely light
8	
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard (heavy)
16	
17	Very hard
18	
19	Extremely hard
20	Maximal exertion

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