POSTURAL THREAT-INDUCED MODULATION OF STRETCH REFLEX PATHWAYS IN STATIC AND DYNAMIC POSTURAL CONTOL

by

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Abstract

There are clear changes to human static and dynamic postural control in situations of elevated postural threat (e.g. standing at the edge of an elevated platform). One possible explanation for these changes is that the amount of afferent information from muscle spindles in the ankle musculature is altered by postural threat. Two experiments have been conducted to explore postural threat-induced changes to soleus spinal stretch reflex function during static control of posture (Study 1), and in response to dynamic postural disturbances (Study 2). In Study 1, soleus Hoffmann (H-) and tendon stretch (T-) reflexes were used to explore changes in reflex amplitude while subjects stood quietly in conditions of low (ground level) and high (3.2m above ground) postural threat. Height-induced postural threat was associated with larger T-reflexes and higher arousal, these effects occurred without systematic changes in H-reflex amplitudes or background muscle activation. We interpret these findings as indirect evidence for arousal-mediated changes in muscle spindle sensitivity. In Study 2, emotionally-charged pictures were used to explore the effects of arousal on H- and T-reflexes, as well as whole body postural perturbations. The pictures failed to elicit significant changes in physiological arousal, H- or T-reflexes, or perturbation response parameters. However, the threat of postural perturbation caused parallel increases in T-reflexes, physiological arousal, and perceived anxiety. Therefore, we conclude that arousal-induced changes in stretch reflexes are not context specific, but rather a generalized response to postural threat. Furthermore, these results together provide substantial evidence in support of independent modulation of muscle spindle sensitivity in humans.

Preface

The protocols used in these studies were reviewed by The University of British Columbia Clinical Research Ethics Board (UBC CREB# H06-70316; see Appendix A). All subjects provided written informed consent prior to participation in these studies and every effort has been made to ensure that the subjects are not identified in this thesis.

None of the studies contained in this thesis have been submitted for publication at the time of thesis submission.

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Chapter 1: General Introduction

The ultimate goal when standing is to not fall down, or in other words, maintain balance. There are two general types of balance control required to achieve this goal. When standing quietly the body needs to counteract the effects of gravity to maintain stance. Sometimes however, the body is perturbed by acute external forces, other than gravity, and it must respond dynamically to avoid a fall. At the moment, the leading model for static, or quiet, balance control is the inverted pendulum. In the inverted pendulum model the body is represented as a point mass fixed to a rigid segment that constantly sways about a single axis of rotation (the ankles) [Winter, Patla, Prince, Ishac, & Gielo-Perczak, 1998]. The position of the point mass, which represents the body's centre of mass (COM), is coupled to and controlled by the changing distribution of forces exerted by the feet onto the ground [Winter, Prince, Frank, Powell, & Zabjek, 1996; Winter et al., 1998]. The mean location of these forces is known as the centre of pressure (COP), which can be interpreted as the net result of all forces involved in balance control [Winter et al., 1996]. The position of the COP is dictated by the location of the COM and the relative stiffness of the ankle musculature and other soft tissues spanning the joint. One inverted pendulum-based theory of postural control dictates that the ankle muscles can be modelled as tuned springs, where the stiffness of the springs dictates how much the pendulum sways [Winter et al., 1998]. However, some argue that the passive stiffness generated from the ankle musculature is not sufficient to maintain balance [Loram, Maganaris, & Lakie, 2007]. Therefore, active muscle tone must also be generated to maintain balance. How stable people are, or how much they sway is controlled by changing muscle tone. As tone is increased, the muscles stiffen and sway is restricted.

Postural perturbations force the body to respond with dynamic postural responses to maintain balance. Dynamic postural responses serve to return and stabilize the COM within the bounds of stability. These responses can be broken into three distinct phases: stretch reflexes, balance correcting responses, and stabilization responses [Carpenter, Allum, & Honegger, 1999a;Carpenter, Frank, Adkin, Paton, & Allum, 2004a]. Spinal stretch reflexes (40-90 ms post-perturbation) are the direct result of muscle stretch induced by the perturbation and do not necessarily contribute to recovery of balance (e.g. stretch reflexes in ankle muscles are counter-productive to regaining balance after a support surface rotation [Allum & Honegger, 1992]). Automatic balance correcting responses are the first corrective responses to the perturbation and typically begin 90-120 ms after a perturbation [Diener, Dichgans, Bootz, & Bacher, 1984; Allum, Huwiler, & Honegger, 1996]; they primarily serve to bring the COM back over the base of support. Interestingly, the latencies of these responses are considered too short to be volitional and too long to be simple spinal reflexes [Nashner & Cordo, 1981], suggesting that they are triggered in a supra-spinal loop. Finally, stabilization responses (>350 ms post-perturbation) serve to stabilize the COM in the new mechanical constraints of the post-perturbation environment. Due to their late onset latencies, stabilisation responses are thought to be under more volitional control [Carpenter et al., 1999a].

In an experimental setting, measures of COP, estimated COM location, and ankle muscle activity can give insight into how posture is controlled, a field generally known as posturography. Quiet sway can be quantified by tracking the COM through space, by monitoring COP, and by comparing COM and COP to estimate sway stiffness [Winter et al., 1998; Winter, Patla, Rietdyk, & Ishac, 2001]. Electromyography (EMG) can be used to track muscle activation levels, and comparisons between COM, COP and EMG can be used to relate muscle activation to measures of sway. Dependent measures extracted from these signals, such

as frequency, variability and mean position, can give insights into how sway is controlled. The above techniques can also be used to describe dynamic postural control, such as the response to an induced perturbation [*c.f.* Carpenter et al., 1999a]. One can describe movement strategies in terms of displacement amplitude and velocity with body kinematics (e.g. COM), as well generated reaction forces and segment dynamics with kinetic profiles (e.g. forces that comprise COP) of the response. EMG can also be used to quantify latency and amplitude of muscle responses. It can also be used to study the coordinated pattern of muscle activity within a postural response. These measures taken together can be used to define the postural strategies triggered by the central nervous system to prevent falls.

There is a growing base of literature detailing the effects of fear of falling on postural control. For example, when older adults are afraid of falling, or perceive the risk of injury from falling to be higher, they demonstrate less postural stability than age matched controls [Maki, Holliday, & Topper, 1991]. Recent studies have directly manipulated postural threat with changes in support surface height to observe the effects of fear of falling on postural control. When young healthy adults are exposed to a high postural threat they shift their COP away from the edge, increase their COP sway frequency, and decrease the variability in amplitude; effectively becoming more stable [Carpenter, Frank, & Silcher, 1999b; Carpenter, Frank, Silcher, & Peysar, 2001a; Carpenter, Adkin, Brawley, & Frank, 2006; Adkin, Frank, Carpenter, & Peysar, 2000; Brown, Sleik, Polych, & Gage, 2002; Brown, Polych, & Doan, 2006; Davis, Campbell, Adkin, & Carpenter, 2009; Huffman, Horslen, Carpenter, & Adkin, 2009]. Interestingly, this effect is only seen when normal vision is available. When subjects stand with their eyes closed the effects of threat on sway frequency and variability are abolished [Carpenter et al., 1999b]. Carpenter et al. [1999b] concluded that subjects were exercising a stiffer control of their COM at high threat in order to decrease the risk of falling over the edge, a hypothesis

that was later confirmed with whole body kinematic data [Carpenter et al., 2001a]. Carpenter et al. [2001a] further demonstrated that postural threat induced leaning and stiffening responses occur with concurrent increases in tibialis anterior (TA) and decreases in SOL (SOL) muscle activity. In the same paradigm, Adkin et al. [2000] demonstrated that the effects of postural threat on quiet standing are dependent on the degree of threat; subjects tend to gradually adopt a stiffer control of their posture as the relative threat is increased. Taken together, these studies clearly indicate that postural threat influences how people stand quietly, and that the strength of this effect is dictated by the degree of threat.

Although it is important to understand how people control their posture when standing quietly, perturbing posture can also be a useful method for investigating postural control. Since there are clearly adaptations to the ways people control their balance when standing still in a high postural threat scenario, it is perhaps not surprising that there are also changes to how they respond to perturbations. People tend to restrict forward body displacement when perturbed towards the edge of an elevated platform [Brown & Frank, 1997; Carpenter et al., 2004a]. Furthermore, people respond with larger balance correcting responses in the muscles responsible for recovering balance, independent of perturbation direction [Carpenter et al., 2004a]. The fact that balance correcting responses are larger, independent of perturbation direction is an interesting finding. Presumably, a perturbation purely in the medial-lateral plane should be no more threatening in the high than in the low threat condition when the source of the postural threat lies in front of the subject. Therefore, medial-lateral balance correcting response should be the same across conditions of postural threat. Since medial-lateral responses change [Carpenter et al., 2004a], it suggests that the changes are not perturbation-specific (i.e. larger responses when perturbed towards the threat, unchanged responses in all other directions), suggesting that there is likely some overarching effect of the threat on postural control. The

global change in response might reflect either: an amplification of incoming sensory inputs, such as a more intense visual, vestibular or proprioceptive signal in response to the same perturbation; or a change in the response parameters that are cued by the sensory stimuli.

The above investigations have produced a convincing base of evidence to suggest that there is an effect of postural threat on postural control. However, these studies fail to explain why and how this adaptation occurs. Since these early studies, much of the postural threat literature has focussed on identifying the underlying psychological, physiological and/or neurophysiological mechanisms that are responsible for inducing the changes to postural control.

Three findings of particular interest have come from pursuits of the psychological impacts of postural threat on postural control. First, the effects of postural threat on postural control are largest when vision is available [Carpenter et al., 1999b; Davis et al., 2009]. Therefore, it would seem that people process the available visual information and interpret it, either consciously or unconsciously, as threatening. This would suggest that postural responses to threat are grounded in a cognitive or psychological experience of the threat. In fact, it has been demonstrated that there are several psychological consequences of standing in a high postural threat scenario, including: decreases in balance confidence and perceived stability, as well as increases in fear and anxiety [Adkin, Frank, Carpenter, & Peysar, 2002; Carpenter et al., 2006; Davis et al., 2009; Huffman et al., 2009].

Secondly, Huffman et al. [2009] attempted to link conscious motor processing and movement self-consciousness, both indicators of conscious control of movement, to the biomechanical factors of sway frequency, variability, and leaning. They found that conscious motor processing and movement self-consciousness were both correlated with leaning away

from the edge, but sway frequency and variability were not linked with either factor. This result suggests that the different aspects of the postural responses to threat are perhaps not all controlled by the same mechanisms. While sway frequency and variability, the indicators of stiffness, are likely controlled by sub-conscious processes, leaning may be a result of a more conscious level of postural control.

The third important finding from psychological investigations of postural threat is that not everyone responds in the same way to postural threat. Davis et al. [2009] attempted to extend the scaling effect of postural threat, previously reported by Adkin et al. [Adkin et al., 2000], from a maximum surface height of 1.6 m to 3.2 m. All subjects demonstrated the typical frequency and leaning responses to postural threat. However, Davis et al. [2009] demonstrated a significant fear of falling by surface height interaction on sway amplitude variability. They found that their subjects adopted one of two postural reactions in the highest threat scenario: they either decreased (non-fearful), or they increased their sway variability (fearful). As such, people who were fearful increased sway variability and ultimately became more unstable. Alternatively, people who were merely anxious at the highest threat position decreased sway variability, and therefore, increased sway stiffness. This means that postural threat induced stiffness can be overridden by genuine fear, implying that the stiffening response in young healthy adults is anxiety, not fear based. Based on the results of these investigations of the psychological effects of postural threat, the following conclusions can be drawn: postural threat can influence anxiety, self efficacy and perceptions of movement; sway stiffness is linked to anxiety but can be overridden by fear; and, in contrast to leaning, sway stiffness is not associated with a change in conscious control of posture.

Postural threat induced anxiety, as a construct, has three basic components: a cognitive component, where the individual becomes aware of the consequences of the threat; a valence or

pleasantness component, the experience is generally unpleasant; and an arousal component, the person becomes physiologically aroused [Neiss, 1988]. It is not clear how these factors might interact to induce the threat response, or even if they are all involved in mediating it. Of the three, physiological arousal has consistently been linked to postural threat [Adkin et al., 2002; Brown et al., 2002; Carpenter et al., 2006; Brown et al., 2006; Davis et al., 2009; Huffman et al., 2009]. It is not clear if, or how arousal might influence postural control, as direct links between the autonomic and somatic systems have not been fully established. However, there is at least some indirect evidence to suggest that the vestibular and proprioceptive systems might be influenced by autonomic state.

The vestibular system might be an avenue for arousal to influence postural control. Balaban and Jacob [2001] and Balaban [2002] argue that anxiety and autonomic arousal are both linked with vestibular function. The vestibular nuclei have reciprocal projections to the parabrachial nucleus, and are also subject to noradrenergic influence from the locus coeruleus and possibly serotonergic influences from the dorsal raphe nucleus [Balaban, 2002]. This means that the vestibular nuclei should be excited when sympathetic activity is increased. Therefore, with arousal, people should be more able to perceive and respond to head and body motion. Theoretically, this should increase sway frequency and decrease variability, making quiet sway stiffer. In a dynamic paradigm, the elevated excitability state should translate into larger perturbation-induced potentials at the vestibular nuclei, which should in turn translate to larger balance correcting response volleys to the vestibulospinal, vestibulocollic, reticulospinal and possibly transcortical pathways [Marsden, Playford, & Day, 2005], increasing the magnitude of balance correcting responses.

There is also a body of literature that suggests that the proprioceptive system can be influenced by autonomic arousal. Achilles tendon stretch reflexes are facilitated by emotionally

[Bonnet, Bradley, Lang, & Requin, 1995] or sexually arousing images [Both, Boxtel, Stekelenburg, Everaerd, & Laan, 2005], as well as static handgrip and ischemia [Hjortskov, Skotte, Hye-Knudsen, & Fallentin, 2005; Kamibayashi et al., 2009], all of which are known to drive sympathetic arousal. Matre and Knardahl [2003] demonstrated that when plasma noradrenalin was increased by glucose ingestion, ankle movement detection thresholds were decreased, suggesting a change in proprioceptive acuity. Furthermore, resting muscle spindle efferent discharge increases with behaviours that are thought to drive sympathetic activity, including isometric and isotonic contractions, Jendrassik's manoeuvre, and mental computation [Ribot, Roll, & Vedel, 1986; Ribot-Ciscar, Rossi-Durand, & Roll, 2000]. However, the literature does not decisively point to an effect of sympathetic activity on proprioceptive function. Passatore et al. [1996] demonstrated that cervical sympathetic nerve stimulation in precollicularly decerebrate rabbits decreased afferent firing rates in response to masseter vibration. Similar results were seen in anaesthetized cats when cervical sympathetic nerve stimulation was paired with sinusoidal oscillations in trapezius and splenius muscle lengths [Hellström, Roatta, Thunberg, Passatore, & Djupsjöbacka, 2005]. Finally, Macefield, Sverrisdottir and Wallin [2003] were unable to demonstrate a conclusive effect of breath-holding, which drives sympathetic activity, on human peroneal nerve muscle spindle afferent firing rates.

If arousal does alter proprioceptive function by either increasing acuity or amplifying lower limb stretch reflexes, then one would expect an increase in sway stiffness during quiet standing. An increase in proprioceptive acuity would mean that less sway-induced ankle muscle stretch would be required to generate an afferent signal. Therefore, assuming the efferent firing threshold has not changed, less stretch would be required to generate a spinal stretch reflex. This would result in more frequent contractions, which would increase muscle tone, which would

decrease sway amplitude and increase sway frequency, or cause a stiffening response. The same result could be achieved by priming the efferent motor neurons to respond to afferent signals, if the efferent firing threshold is decreased then it would require fewer afferent impulses to cause the motor neuron to fire. Again, this would result in more frequent muscle contractions, which could have the effect of decreasing amplitude variability and increasing frequency of sway. Furthermore, in response to a perturbation, a change in proprioceptive acuity would translate into a larger perturbation-induced afferent volley. This larger volley should increase spinal reflexive responses, as well as proprioception-based supra-spinal automatic balance correcting responses.

Based on the current state of the postural threat literature, two important questions arise concerning both static and dynamic postural control. First, is the stiffer control of sway that is typically seen when standing quietly an effect of a gain change in the lower-limb stretch reflex pathways? In Study 1 I have addressed this issue by testing both Hoffmann and tendon-stretch reflexes in situations of low and high height-induced postural threat. My second question is: do the effects of anxiety and arousal on spinal reflex pathways also translate into changes to dynamic postural control? In Study 2 I used emotionally charged pictures to study the effects of arousal on Hoffmann and tendon-stretch reflexes, as well as dynamic responses to support surface tilts to see if links could be established between spinal reflexes and dynamic postural control.

Chapter 2: Study 1 – Static Postural Control

2.1. Introduction

Soleus (SOL) Hoffmann (H-) reflexes have been demonstrated to be modulated when people stand in conditions that are associated with an increase in postural threat. Specifically, Sibley, Carpenter, Perry, & Frank [2007] demonstrated that H-reflex amplitudes were smaller when people stood at the edge of an elevated platform with their eyes open. Furthermore, their subjects did not demonstrate H-reflex inhibition when they stood with their eyes closed at the edge, or when they stood in the centre of the elevated platform. Sibley's [2007] results parallel observations made in experiments where slightly different postural threats were imposed. For example, Llewellyn et al. [1990] demonstrated that SOL H-reflexes are attenuated when people walk on a narrow elevated walkway compared to a ground level treadmill. And McIlroy et al. [2003] demonstrated that SOL H-reflexes were smaller when subjects were informed that a pendulum they were balancing with their feet might be perturbed. Although this is not a situation where the consequences of a fall are elevated, it can be considered a postural threat because the likelihood of a loss of "balance" is increased. In all cases the changes in H-reflexes were independent from changes in stimulus intensity or tonic muscle activity that might otherwise explain the reflex modulation.

There are four ways that SOL H-reflexes can change in amplitude without a change in stimulation intensity. These include: (1) reciprocal inhibition caused by contraction of an antagonist muscle (e.g. tibialis anterior) [Stein, Estabrooks, McGie, Roth, & Jones, 2007; Zehr, 2002]; (2) descending facilitation relating to agonist contraction (i.e. voluntary contraction of SOL) [Stein et al., 2007]; (3) descending presynaptic inhibition (PSI) [Zehr, 2002]; and (4) homosynaptic post-activation depression (HPAD) [Curtis & Eccles, 1960; Stein et al., 2007].

These influences can act in isolation or exert a cumulative effect, which can be inferred through different experimental techniques.

It is unlikely that either reciprocal inhibition or descending facilitation is responsible for the decrease in H-reflex amplitude induced by postural threat. Sibley et al. [2007] only found an effect of threat on H-reflexes when subjects stood with their eyes open, despite similar changes in muscle activity in both visual conditions. If reciprocal inhibition and/or a drop in descending agonist facilitation were to blame for the change in reflex amplitude, then the change should have been consistent across visual conditions.

PSI has not yet been ruled out as the mechanism through which H-reflexes are inhibited in a high postural threat paradigm. PSI results from the excitation of inhibitory interneurons in the spinal cord that act on the presynaptic terminals of afferents [Zehr, 2002]. There are several potential sources that could cause PSI, such as descending corticospinal or rubrospinal projections [Krakauer & Ghez, 2000], and reciprocal projections from antagonist afferents (here, labelled reciprocal inhibition) [Zehr, 2002]. For the sake of clarity, in this paper PSI will be treated as a descending effect, distinct from reciprocal inhibition, and is assumed to originate rostral to the lower motor neuron pool.

In an H-reflex paradigm, PSI would ultimately suppress the size of the reflex by reducing the amount of neurotransmitter released by the stimulus pulse. In the Sibley et al. [2007] study, the subjects may have responded to the threat by using PSI to inhibit all lower limb reflexes to shift from reflexive to a more volitional control of posture [*c.f.* Huffman et al., 2009]. Alternatively, they may have selectively inhibited the SOL lower motor neuron pool, perhaps to reduce the destabilizing effect of the H-reflexes. Since H-reflexes cause small perturbations in a specific limb, it is plausible that with the added threat of falling from height the subjects in both

the Sibley et al. [2007] and Llewellyn et al. [1990] studies opted to inhibit the spindle afferents ascending from the SOL that, within the context of the experiment, may cause them to be unstable. Unfortunately, the available data do not allow for confirmation or rejection of either hypothesis.

The last mechanism that could possibly account for H-reflex attenuation with postural threat is HPAD. HPAD is thought to be an artefact of excessive tonic afferent firing, where neurotransmitter stores become depleted at the site of the presynaptic terminal [(Curtis & Eccles, 1960; Hultborn et al., 1996; Trimble, Du, Brunt, & Thompson, 2000]. In effect, this depletion of available neurotransmitter causes synaptic transmission to be less potent with successive action potentials. Theoretically, one could demonstrate HPAD by isolating a 1a-afferent and a corresponding α-motor neuron from all other influences and manipulate the firing frequency of the afferent while recording the firing frequency of the efferent. Direct evidence for HPAD could be garnered if the resultant frequency curve is bell-shaped. Obviously, this protocol is not possible to test in living human subjects, though HPAD can be induced experimentally with paired H-reflexes. When two H-reflexes are paired in close succession, the first reflex can have a depressive effect on the second [Stein et al., 2007]. By using a paired stimulation technique where the stimulation intensities and inter-stimulus intervals are kept constant across experimental conditions, one can test for a change in HPAD by comparing the relative depression of the second reflex across conditions. If HPAD is responsible for the change in H-reflexes previously reported with postural threat [Sibley et al., 2007; Llewellyn et al., 1990; McIlroy et al., 2003], then the above method should reveal the effect of postural threat on HPAD.

Of the mechanisms that can influence H-reflexes outlined above, HPAD is worth further discussion because of its indefinite implications on human neurophysiology. If HPAD is

induced when people become anxious in response to a postural threat, then it implies that for some reason afferent pathways are being stimulated at frequencies higher than which they can effectively transmit [Curtis & Eccles, 1960]. A change in spindle afferent firing frequency is not incredibly novel, it happens anytime a muscle spindle is stretched. The tension in muscle spindles is controlled by the γ-motor neuron or fusimotor system, which is distinct from the α-motor neuron system which activates extrafusal muscle fibres [Pearson & Gordon, 2000]. Typically, the fusimotor system works in parallel with the α-motor system to ensure that muscle spindle fibres contract when the muscle contracts and relax when the muscle is elongating or at rest [Pearson & Gordon, 2000]. This effectively ensures that the muscle spindles and surrounding muscle fibres maintain a consistent relative tension, ensuring the spindles are primed to detect stretch at any muscle length. This process is called alpha-gamma coupling.

In an elevated postural threat scenario the SOL is not elongated, it is shortened because people tend to adopt a posterior lean away from the edge, so there should not be an increase in tonic spindle firing rate associated with tonic muscle stretch. Furthermore, SOL is generally less electrically active in a high postural threat situation [Carpenter et al., 2001a], which means that the α-motor system is less active[1] and therefore the fusimotor system should be more quiescent as well. This means that the intrafusal fibres should, if anything, be relatively slack and less sensitive to muscle stretch. Combined, these effects should lead to a drop in afferent traffic from muscle spindles, and thus reduce the likelihood of an HPAD effect. However, this logic relies on the assumption that fusimotor activity cannot change independently from α-motor activity.

[1] It is difficult to relate changes in surface EMG with changes in muscle activity when a muscle has changed length because the number of fibres and their relative depth with respect to the recording range of the electrodes has changed. Although, when a muscle has contracted there should be more fibres within the recording range of the electrodes and if the same motor units are being activated at the new length, then a greater proportion of their fibres should be within the recording range. Therefore, if EMG activity decreases when muscle length has also decreased, it is safe to assume that fewer motor units are being activated at the new length.

There is however some evidence to suggest that mammals can modulate fusimotor activity independently from α-motor activity. Based on studies of single spindle primary afferent recordings, Prochazka et al. [1988] argue that muscle spindles can 'wind-up' to suit different behavioural contexts. For example, a cat that is seated and preparing to move will demonstrate some γ-static activity with almost no γ-dynamic activity. Where a cat that is walking across a narrow elevated beam (in a state of elevated postural threat), would have high γ-static and γ-dynamic activity [Prochazka et al., 1988]. Presumably, this spindle 'wind-up' provides the cat with a richer proprioceptive experience and allows them to react more readily to maintain their balance.

Although there is as of yet no direct evidence that humans can alter fusimotor activity, in theory it should be possible to experimentally infer a change in muscle spindle 'wind-up' in humans. One can dissociate changes in reflex excitability that occur at the spinal level from those that occur outside of the central nervous system by comparing electrically evoked H-reflexes with mechanically evoked tendon-stretch (T-) reflexes. An H-reflex is elicited by a current applied to the afferent axons ascending to the spinal cord. Therefore, H-reflexes can only be modified by factors acting on the spinal cord at the level of the lower motor neuron pool. A T-reflex is elicited by stretching the muscle-tendon unit and the muscle spindles imbedded therein. Therefore, a T-reflex is subject to the same modulation at the spinal level as an H-reflex, but it is also subject to changes in the states of the muscle spindles from which it was elicited. By comparing H-reflexes and T-reflexes elicited through the same neural pathways, it should be possible to dissociate changes in motor neuron pool state from receptor sensitivity. For example, if an experimental manipulation causes attenuation in both H- and T-reflexes equally, then it is likely that the attenuation is occurring because of spinal level inhibition. Alternatively, if H-reflexes are unchanged and T-reflexes are augmented by the experimental

manipulation, then it is likely that the T-reflex augmentation is the result of a change at the level of the muscle spindle, such as spindle 'wind-up'. It is difficult to predict the effects of HPAD on T-reflexes. Theoretically, T-reflexes should be inhibited by HPAD. Although, if HPAD is the result of increased afferent traffic related to spindle 'wind-up', then a single T-reflex would likely be larger, despite HPAD. From the above scenarios, it is clear that both H- and T-reflexes must be tested simultaneously when searching for mechanisms that might influence postural control through spinal reflex pathways

The purpose of this study was to uncover the mechanisms through which height-related anxiety attenuates lower limb spinal reflexes in a static postural control paradigm. The study was designed to address three specific research questions: 1) Can I confirm that SOL H-reflexes are attenuated with postural threat? 2) Are SOL T-reflexes also inhibited by postural threat? 3) Is afferent over-activity, or HPAD, responsible for the attenuation of SOL H-reflexes previously reported with high postural threat? Three related hypotheses were tested: first, I hypothesized that I would confirm the findings of Sibley et al. [2007] and demonstrate an inhibitory effect of threat on SOL H-reflexes. Second, considering the evidence for spindle 'wind-up' in animal models [Prochazka et al., 1988]; I hypothesized that my human subjects would demonstrate larger T-reflexes when standing in a high versus low postural threat situation. And third, since I expected T-reflex facilitation from 'wind-up' and H-reflex attenuation, I hypothesized that HPAD would be the cause of H-reflex attenuation.

2.2. Methods

2.2.1. Subjects

Thirty-two young healthy adults recruited from the university community by word of mouth consented to participate in this study, of which, 20 (8 female) completed the protocol (mean ± SE; 24.7 ± 0.71 years). No subjects reported any known neurological, vestibular or orthopaedic impairments that might impair their ability to maintain quiet stance. Furthermore, no subjects reported an extreme fear of heights at the outset of the study. This study was approved by the University of British Columbia Clinical Research Ethics Board (see Appendix A), and all subjects provided written informed consent prior to their participation in the study.

2.2.2. Protocol

Postural Threat

Subjects were asked to stand quietly (i.e. stand with their feet side-by-side and not move, but not necessarily stand as still as possible, see Zok et al. [2008] for distinctions) on a hydraulic lift in two conditions of postural threat. In the low threat condition (LOW) subjects stood 0.8m above ground and 0.6 m from the edge of the support surface. In the high threat condition (HIGH) subjects stood 3.2 m above ground and at the edge of the support surface (Figure 1-1). As an effect of presentation order on postural responses to threat has previously been reported [Adkin et al., 2000], the LOW condition was always presented before the HIGH condition. Each condition consisted of two 150 sec standing trials with a two-minute standing break in between. Subjects continued to stand in the same positions during the breaks between trials. However, while the lift ascended from the LOW to the HIGH position subjects were allowed to lean backwards onto a chair-back, allowing them to lean away from the edge and rest their legs, while still keeping their foot position constant. Prior to each trial subjects completed a pre-test

balance confidence questionnaire and after each trial they completed the psychosocial questionnaires outlined below.

Stimulation Protocol

Five H-reflex paired stimulations (1 stimulation = 2 reflexes) and five single T-reflex stimulations were elicited in a pseudo-random order in each trial. H-reflex pairs and single T-reflexes were interspersed with a variable 10 to 30sec interval to ensure the reflexes were not unintentionally influenced by HPAD [Stein et al., 2007] or expectation. Prior to starting the experiment, subjects underwent an M-wave recruitment curve while standing in a control condition at the LOW height. Single, 1 ms long, 150 V electrical stimulations to the right tibial nerve, spaced 7 sec apart and of increasing amperage (S48 Stimulator, SIU5 Stimulation Isolation Unit, CCU1 Constant Current Unit; Grass Technologies, USA) were used to generate the M-wave recruitment curve. H-reflexes were induced with 1ms long, 150V square-wave electrical pulses with a stimulation intensity set to induce an M-wave between 10% and 15% of the individual's maximum M-wave amplitude (M_{max}). This intensity was chosen to ensure susceptibility to modulation (facilitation and/or inhibition) [Hultborn, Meunier, Morin, & Pierrot-Deseilligny, 1987; Katz, Meunier, & Pierrot-Deseilligny, 1988)], and is comparable in intensity with previous investigations of SOL H-reflex modulation [Sibley et al., 2007; Llewellyn et al., 1990; McIlroy et al., 2003]. H-reflexes were elicited in pairs 200ms apart to induce HPAD effects on the second reflex [Stein et al., 2007]. The 200 ms interval was chosen based on pilot investigations which demonstrated 200 ms to be an interval that reliably produced an HPAD effect, but was not strong enough to completely extinguish the second reflex. Tendon stretch (T-) reflexes were elicited in the right SOL by single mechanical taps directed perpendicular to the long axis of the Achilles' tendon with a computer controlled linear motor

(motor: LinMot PS01 – 23x80, controller: LinMot E2000-AT, software: LinMot v.1.3.12; NTI Ltd, USA).

Bracing

There is evidence to suggest that SOL length [Tokuno, Carpenter, Thorstensson, Garland, & Cresswell, 2007; Tokuno, Garland, Carpenter, Thorstensson, & Cresswell, 2008; Tokuno, Taube, & Cresswell, 2009] and both SOL and TA contraction intensity [Stein et al., 2007] can influence SOL stretch or H-reflex amplitude. Since leaning away from the edge [Carpenter et al., 1999b; Carpenter et al., 2001a; Adkin et al., 2000; Sibley et al., 2007] and changes in background muscle activity [Carpenter et al., 2001a; Sibley et al., 2007] are typically seen when subjects stand at the edge of a high surface, these factors were controlled to ensure that changes in SOL reflex sensitivity can reliably be attributed to changes in postural threat. To control leaning in this study, I braced my subjects about the ankles in both conditions of postural threat (Figure 1-2). Pilot investigations suggest that standing braced about the ankles not only controls ankle angle, but also reduces the changes in tonic shank muscle activity typically seen with increased postural threat [Carpenter et al., 2001a; Sibley et al., 2007]. The subjects were instructed to stand "as normal as possible" in the braces, and not to lean into, or rely on the braces for support.

2.2.3. Measures

Electromyography

EMG (Telemyo 2400 RG2, Noraxon, USA) was collected from the right SOL and TA with belly-belly preparations as well as a SOL belly-tendon preparation. The signals were amplified 500x and band-pass filtered between 10 and 1500 Hz online. H-reflexes were measured as the peak-to-peak amplitude of the first H-wave in each pair in the SOL belly-

tendon EMG signal (Figure 1-3 Part A). SOL belly-tendon preparations were used because they do not capture as much high frequency EMG signal as a belly-belly preparation [Fuglevand, Winter, Patla, & Stashuk, 1992], thus improving the odds of discerning a reflex response from background muscle activity. The peak-to-peak amplitude of the second H-wave in each pair was measured and subtracted from the first to calculate the HPAD effect (Figure 1-4 Part A). H-reflex and HPAD amplitudes were then averaged across the two 150 sec trials performed in each condition to get mean scores for each level of postural threat. The peak-to-peak M-wave amplitude for each H-reflex was also measured as a check of stimulation intensity and location. Reflexes were excluded from examination if they exhibited an M-wave amplitude outside of the target stimulation range (10-15% M_{max}).

T-reflexes were measured as the peak-to-peak amplitude of the twitch EMG potential that occurred 30 to 50 ms after the mechanical tendon tap in the SOL belly-tendon signal (Figure 1-5 Part A). T-reflex potentials had to be visually discernable from background activity to be included in the final analysis. T-reflex amplitudes were averaged across trials for each condition for each subject. For the sake of comparison with past postural threat studies, background muscle activity was calculated as the root mean square error of the SOL and TA belly-belly EMG signals 100 ms prior to the stimulus artefact of each reflex. These values were also averaged across trials for each condition. Since it is common for individuals to increase lower limb background activity with increased postural threat [Carpenter et al., 2001a; Sibley et al., 2007], and although the ankle braces were intended to limit this effect, I excluded an H- or T-reflex if the background activity preceding it was more than twice the amplitude of the mean background activity of the LOW condition. Had I not imposed this criterion, I would not have been able to argue that the electro-mechanical state of the muscle was consistent across stimulations [*c.f.* Stein et al., 2007].

Electrodermal Activity

Galvanic skin conductance was used to quantify electrodermal activity (EDA) as an indicator of autonomic arousal. EDA was measured from the thenar and hypothenar eminences of the non-dominant hand at 1000 Hz (model 2502, CED, UK) and averaged over each 150 sec trial. Trials were then averaged across height conditions to quantify the effect of postural threat.

Psychosocial Measures

As a measure of self-efficacy, subjects rated their confidence that they would be able to maintain their balance and avoid a fall prior to each trial. After each trial they then rated their experienced fear [*c.f.* Davis et al., 2009] and perceived stability [Schieppati, Tacchini, Nardone, Tarantola, & Corna, 1999] as well as completed a state anxiety scale [Adkin et al., 2002; modified from: Smith, Smoll, & Schutz, 1990]. The state anxiety scale is intended to quantify subjective perceptions of three aspects of state anxiety related to the postural threat: somatic anxiety, worry, and concentration [Adkin et al., 2002].

2.2.4. Statistical Analyses

The dependent measures of interest in this study were: H-reflex amplitude, HPAD magnitude, T-reflex amplitude, background EMG for SOL and TA, GSC, and the psychosocial scores. The change in dependent measures across conditions of postural threat was tested with within-subjects t-tests for statistical significance. The criterion for statistical significance for all tests was set to $\alpha = 0.05$.

2.3. Results

2.3.1. Subject Exclusions

Of the 20 subjects who completed the study, I excluded three subjects because they exhibited background TA activity that was at least twice as high in the HIGH condition as the LOW. Therefore, seventeen subjects were included in the data set presented here. Of those included, 14 subjects had reliable H-reflex data (consistent M-wave amplitude between 10-15% M_{max}), and 15 subjects had reliable T-reflex data; 12 subjects were included in both the H- and T-reflex data pools. Almost all subjects demonstrated some change in M-wave amplitude from first to second stimulation in an HPAD pair. If the second M-wave in a pair fell outside of the 10-15% M_{max} range, then the HPAD effect from that pair was not included in the final analysis. 13 of the 14 subjects included in the H-reflex analysis were included in the HPAD analysis. Due to changes in the protocol with the progression of the study, GSC was only collected from 14 of the 17 subjects, and psychosocial questionnaires were only completed by 10 subjects.

2.3.2. Arousal, Anxiety and Psychosocial Measures

Participants in this study were less confident in their ability to maintain balance for the duration of the balance task in the HIGH compared to the LOW condition ($t_9 = 3.213$, $p = 0.011$, $\eta^2 = 0.534$). During the trials, participants were significantly more aroused in the HIGH condition than the LOW condition, as indicated by an increase in mean EDA ($t_{13} = -2.277$, $p = 0.040$, $\eta^2 = 0.285$). Subjects also experienced more anxiety ($t_9 = -2.769$, $p = 0.022$, $\eta^2 = 0.460$), more fear ($t_9 = -3.591$, $p = 0.006$, $\eta^2 = 0.589$) and felt less stable ($t_9 = 2.818$, $p = 0.020$, $\eta^2 = 0.469$) at the HIGH compared to the LOW height.

2.3.3. Reflex Amplitudes and Background EMG

On average, H-reflexes were 10.6% smaller in the HIGH compared to LOW height condition, though this effect was not statistically significant (Figure 1-3 Part B; $t_{13} = 1.254$, $p = 0.232$, $\eta^2 = 0.108$). Despite the average decrease in H-reflex amplitude, only half of the subjects (7 of 14) actually demonstrated a decrease in H-reflex amplitude in the HIGH condition, the rest of the subjects' H-reflexes were either slightly higher or the same across threat conditions (Figure 1-3 Part C). There was no significant change in M-wave amplitude (Figure 1-3 Part B; $t_{13} = -0.467$, $p = 0.648$, $\eta^2 = 0.016$), suggesting that any change in H-reflex amplitude was not due to changes in stimulation intensity across conditions. Furthermore, there was no change in the calculated HPAD effect across conditions of postural threat (Figure 1-4 Part B; $t_{12} = 0.465$, $p = 0.650$, $\eta^2 = 0.021$).

As depicted in Figure 1-5 Part B, T-reflex peak-to-peak amplitudes were significantly increased with height. T-reflex amplitudes were on average 40.7% larger in the HIGH compared to the LOW threat condition ($t_{14} = -3.343$, $p = 0.005$, $\eta^2 = 0.444$), with 14 of the 15 subjects showing an increase (Part C). Finally, as shown in Figure 1-6, there does not appear to be an effect of threat on either TA ($t_{16} = 0.619$, $p = 0.547$, $\eta^2 = 0.028$) or SOL ($t_{16} = -0.495$, $p = 0.628$, $\eta^2 = 0.015$) background EMG.

2.4. Discussion

With this experiment I have confirmed my hypothesis that postural threat would increase T-reflex amplitudes. However, while H-reflexes were on average inhibited in the HIGH condition, I cannot support my hypothesis for threat-induced H-reflex inhibition with these data. Since any potentiation at the synapse should affect both H- and T-reflexes equally, I suggest that the facilatory drive acting on T-reflexes must come from a peripheral source. The most likely peripheral source for T-reflex facilitation is a change in muscle spindle sensitivity. An increase in muscle spindle sensitivity, or spindle 'wind-up' [Prochazka et al., 1988], would mean that a constant mechanical stretch of the muscle would cause a larger afferent volley to the spinal cord. Such a change in spindle sensitivity would not necessarily be evident at the level of the lower motor neuron pool; therefore, H-reflexes should not be directly influenced by it.

In theory, elevated muscle spindle sensitivity and normal sway-induced loading should cause HPAD. Higher Ia afferent neuron firing rates caused by increased spindle sensitivity could lead to a depletion of neurotransmitter stores at the synapse (HPAD). However, I was not able to support my hypothesis that the lower motor neuron pool would be inhibited by HPAD with increased postural threat. There was no significant effect of postural threat on calculated HPAD. Furthermore, H-reflexes, which should be susceptible to HPAD, were not inhibited with threat.

The failure to demonstrate a significant change in H-reflex amplitude makes this study difficult to place within the context of the current postural threat literature. Typically, H-reflexes are inhibited with threat or balance task difficulty [Sibley et al., 2007; Llewellyn et al., 1990; McIlroy et al., 2003]. It is plausible that my failure to replicate these results is due to a methodological deviation from previous experiments. For example, it is possible that I failed to demonstrate a change in HPAD in this study because I had my subjects stand braced about the ankles. Depletion of neurotransmitter facilitated by increased muscle spindle sensitivity would

be dependent on the muscle being repeatedly stretched enough to cause a high spindle firing rate. By bracing my subjects, I effectively limited sway about the ankles, possibly preventing the repeated stretching required to cause the spindle to fire at higher rates and deplete transmitter stores. This could also be the reason why I failed to demonstrate SOL H-reflex inhibition in this study. If postural threat-induced H-reflex inhibition is an artefact of HPAD, then by eliminating HPAD in my experimental manipulation of threat I may have removed the mechanism required to reproduce the inhibition reported in previous studies [Sibley et al., 2007; Llewellyn et al., 1990; McIlroy et al., 2003].

There is one potential confound to the HPAD hypothesis for H-reflex inhibition described above: Sibley et al. [2007] only demonstrated an inhibitory effect of postural threat on H-reflexes when vision was available. When Sibley et al. [2007] had their subjects stand in the high threat condition with their eyes closed their subjects did not demonstrate H-reflex inhibition, despite comparable changes in background EMG across visual conditions. However, Carpenter et al. [1999b] demonstrated that the effects of postural threat on sway frequency and variability are abolished when subjects stand with their eyes closed. This means, that there was possibly a considerable difference in the frequency at which muscle spindles were being stretched across visual conditions in the Sibley et al. [2007] study. This potential change in frequency of stretch could have created different states of HPAD across visual conditions, and therefore, smaller H-reflexes in the eyes-open condition of the Sibley et al. [2007] study.

Alternatively, I may not have demonstrated H-reflex inhibition in this study because I prevented my subject from leaning backwards by bracing them at the ankles. H-reflexes tend to be smaller when subjects lean backwards, compared to forwards [Tokuno et al., 2007; Tokuno et al., 2008; Tokuno et al., 2009]. Also, people tend to lean backwards when standing at height [Carpenter et al., 1999b; Carpenter et al., 2001a]. However, unlike sway frequency and

amplitude, posterior leaning is not influenced by the availability of vision when standing at height [Carpenter et al., 1999b], and yet Sibley et al. [2007] only demonstrated H-reflex inhibition when vision was available. As such, it would seem that postural threat-induced H-reflex inhibition is not linked to posterior leaning, and therefore my failure to reproduce previous effects was not an artefact of sway restriction.

Another discrepancy between this and other studies of postural threat-induced H-reflex modulation is the surface height used to induce the threat. I used a higher surface height to induce threat (3.2 m) than others have used (1.6 m [Sibley et al., 2007], and 0.3 m [Llewellyn et al., 1990]). A change in elevation from 1.6 m to 3.2 m can cause significant differences in arousal, anxiety and balance confidence, as well as a robust fear response in some people [Davis et al., 2009]. Furthermore, when fearful people are put into a high postural threat scenario, they tend to sway more [Davis et al., 2009]. Therefore, it is possible that the participants' experience in this experiment was considerably different than in previous H-reflex studies. However, it is not clear why these theoretical differences between studies would cause the inhibitory effect of threat on SOL H-reflexes to disappear. The subjects in this study reported the increases in arousal, anxiety and fear; and decreases in balance confidence and perceived stability that one would typically expect from an exposure to height-induced postural threat [Adkin et al., 2002; Carpenter et al., 2006; Davis et al., 2009; Huffman et al., 2009].

The fact that the psychosocial and arousal impacts of my threat manipulation are, if anything, more intense than the manipulations used in previous experiments makes my failure to replicate SOL H-reflex inhibition all the more surprising. Postural threat-induced H-reflex inhibition is thought to be a response to awareness to the threat [Sibley et al., 2007]. It is thought that PSI is used to mute reflexes so that they do not destabilize the person and thereby increase

their risk of being harmed [Llewellyn et al., 1990; Sibley et al., 2007]. This begs the question: why would the subjects in this study opt not to induce PSI and inhibit their reflexes?

Sibley et al. [2007] suggested that any of HPAD, PSI and vestibular influences might inhibit H-reflexes. Aside from HPAD for the reasons previously mentioned, it seems unlikely that any of these inhibitory influences would be absent in my paradigm if present in others. That is, unless the ankle braces served to stabilize the subjects enough to prevent the need for these other influences to act on the lower motor neuron pool. Anecdotally, this does not seem likely as the H-reflex stimulations were still strong enough to cause destabilization, despite the braces. Furthermore, the braces were difficult to step with, which one might expect to provide more incentive to limit destabilization because recovery would be more difficult than in a free standing situation. Therefore, it seems unlikely that the braces provided enough stability to significantly alter reflex modulation across heights, even though I cannot confirm this with the available data.

Alternatively, the braces may have elicited some tonic neurophysiological influence on the lower motor neuron pool that would not have been present had the subjects not been braced. Two possible inputs come to mind: elevated Ib reciprocal inhibition and modulation from cutaneous afferents. It is probable that the subjects in this study still had to activate the calf musculature to maintain upright standing [*c.f.* Loram et al., 2007], and possible that by restricting sway the dynamic contractions typically used in sway became more isometric in nature. Isometric contractions increase golgi tendon organ (and Ib afferent) firing rates [Edin & Vallbo, 1990], and therefore increase Ib reciprocal inhibition. It is possible that the braces inhibited the lower motor neuron pool to an extent where the potential height effects were either overridden or masked. However, this seems unlikely because I was able to elicit a measurable HPAD effect in this experiment, clearly demonstrating that I had not reached a "floor effect" of

reflex inhibition. Furthermore, the braces would have provided new cutaneous inputs from just below the knee (where they were attached to the shank), and could have caused plantar cutaneous feedback that was different from free standing. Plantar cutaneous stimulation is known to modulate forced dorsiflexion stretch reflex and H-reflex amplitudes [Sayenko et al., 2007; Sayenko et al., 2009], and these effects are not necessarily the same across stimulation methods [Sayenko et al., 2009]. What is not clear is how cutaneous inputs related to the bracing may have modulated the reflexes studied in this experiment? It seems unlikely that cutaneous inputs would have changed across conditions of postural threat, and it does not appear that I reached a limit in the extent to which spinal reflexes could have been modified (as demonstrated by T-reflex facilitation and induced HPAD), therefore it seems unlikely that cutaneous inputs from the braces can be used to explain my results. However, in future studies it would be beneficial to have both braced and un-braced trials to determine if the braces are in fact modulating reflexes beyond the effects of postural threat.

Finally, my failure to replicate the reflex inhibition reported in previous studies does not appear to be an issue of statistical power. 14 subjects were included in this analysis, where 15 were used by Sibley et al. [2007], 10 by McIlroy et al. [2003], and 5 by Llewellyn et al. [1990]. All of these studies have been from the young healthy adult population, and assuming random sampling, should have similar statistical effects if all studies were dealing with the same physiological phenomenon. Therefore, it seems all the more likely that my failure to replicate is an artefact of methodological variation, and not a sampling error.

The principle finding of this study is that SOL T-reflexes are modulated independently from H-reflexes and tonic muscle activity in situations of elevated postural threat. I conclude that the most likely cause for this effect is an increase in muscle spindle sensitivity. The fact that I was not able to demonstrate a systematic change in H-reflex amplitude, yet had such

prominent changes in T-reflexes, if anything, serves to bolster this conclusion. The change in muscle spindle sensitivity without apparent changes in tonic muscle activity suggests that muscle spindles are becoming more sensitive, likely by either γ-motor neuron activation (without significant changes in α-motor neuron activity) , or direct sympathetic drive. To date there is no direct evidence to suggest that humans are capable of modulating γ-drive, and hence spindle sensitivity, independently from α-motor neuron drive. Nor has there been conclusive evidence presented for sympathetic facilitation of muscle spindles in humans.

There is a growing body of literature that links increased T-reflex amplitudes with arousal [Bonnet et al., 1995; Both et al., 2005; Hjortskov et al., 2005; Kamibayashi et al., 2009]. And, as in this study, arousal seems to influence T-reflexes independently from H-reflexes [Kamibayashi et al., 2009]. There is indirect evidence to suggest that muscle spindle sensitivity is influenced by arousal in humans [Ribot-Ciscar et al., 2000; Ribot et al., 1986; Matre & Knardahl, 2003] and cats [Prochazka, Hulliger, Zangger, & Appenteng, 1985; Prochazka et al., 1988]. Furthermore, Barker and Saito [1981] used electron microscopy to reveal direct links between sympathetic nerves and a fraction of cat diaphragm muscle spindles. As such, it is possible that the changes in T-reflex amplitudes observed in this study are related to increased muscle spindle sensitivity caused by threat-induced arousal.

There are certain limitations to my ability to infer a change in muscle spindle sensitivity from these results. It has been suggested that comparing H- and T-reflexes is confounded by the nature of the H-reflex stimulation. The electrical stimulation of 1a afferents to generate an H-reflex should also stimulate Ib afferents. These Ib afferents should then stimulate Ib inhibitory interneurons which would suppress the H-reflex [Ghanim et al., 2009]. T-reflexes should not be susceptible to this inhibition as the stimuli do not evoke strong Ib afferent volleys. Although I acknowledge this possibility, it seems unlikely that the excitatory postsynaptic potential of the

first H-reflex in each pair (which was used to measure H-reflex amplitude) would be influenced by inhibition from a disynaptic pathway with a slower conduction velocity that was stimulated at the same time, as the inhibitory postsynaptic potential should arrive well after the excitatory potential [Pearson & Gordon, 2000]. This phenomenon may have influenced my HPAD effects by increasing the postsynaptic inhibition for the second reflex. However, I am not aware of a way of calculating the effects of Ib inhibition in an HPAD paradigm.

Finally, it is possible that the discrepancy between H- and T-reflex amplitudes with increased postural threat is due to the shape of the afferent volley caused by the respective stimuli. H-reflexes are square-wave volleys that in this case last 1 ms, where T-reflexes are diffuse, rounded and can last 5 ms [Morita, Petersen, Christensen, Sinkjaer, & Nielsen, 1998]. It is possible that the effects of HPAD or PSI are less intense on T-reflexes because of their dispersion and that if they were as square and short as H-reflexes, they would be inhibited as well. However, my results demonstrate that T-reflexes are facilitated independently of H-reflexes, and furthermore, that H-reflexes are not systematically influenced by postural threat. If H-reflexes were suppressed and T-reflexes did not change then one could attribute the discrepancy to the unequal impact of PSI or HPAD, but these results suggest that some peripheral mechanism is driving T-reflex facilitation independent of central inhibition, if at all present. As such, despite the above concerns, I feel that these results reflect an increase in muscle spindle sensitivity that dwarfs any inhibitory effects of postural threat on the lower motor neuron pool.

2.4.1. Conclusions

In conclusion, despite failing to confirm my hypothesis that H-reflexes would be attenuated with increased postural threat, I have demonstrated that T-reflexes are significantly

facilitated when standing under threatening conditions. I interpret this discrepancy as evidence for peripheral modulation of spinal reflexes with increased anxiety and arousal associated with postural threat. Specifically, I have attributed this facilitation of T-reflexes to heightened muscle spindle sensitivity, which appears to be occurring independent from changes in tonic muscle activation. Finally, I can neither support nor reject the hypothesis that HPAD is the cause of postural threat-induced H-reflex modulation. Although I did not demonstrate a significant change in HPAD, I also did not demonstrate a change in H-reflex amplitudes, therefore I cannot rule out the possibility that HPAD, when present, can inhibit H-reflexes in a high postural threat scenario. These data do not support the theory that PSI, induced when aware of a threat, is responsible for reflex inhibition with increased postural threat because the subjects in this study were, if anything, exposed to a stronger postural threat stimulus than that used in previous studies [Sibley et al., 2007; Llewellyn et al., 1990]. Therefore, these subjects should have demonstrated at least as much, if not more reflex inhibition than that observed in previous studies. Finally, these data support the theory that stretch reflexes are facilitated with arousal, or at least threat-induced anxiety, and that a change in muscle spindle sensitivity is most likely the mechanism through which the facilitation is achieved.

Chapter 3: Study 2 – Dynamic Postural Control

3.1. Introduction

The results from Study 1 suggest that postural threat can induce changes to lower limb stretch reflexes. The increased T-reflex and relatively constant H-reflex amplitudes observed when subjects stood at an elevated surface height suggest that threat facilitates stretch reflexes outside of the lower motor neuron pool, possibly at the level of the muscle spindle. Studies with slightly different manipulations of postural threat than that used in Study 1 have suggested that H-reflexes are inhibited in high postural threat scenarios [Sibley et al., 2007; Llewellyn et al., 1990; McIlroy et al., 2003]. It is not clear why the central nervous system would want to counteract the effects of increased spindle sensitivity (i.e. spindle 'wind-up') under threatening conditions. However, it has been suggested that in situations of either increased postural difficulty, or threat, people shift from spinal to supra-spinal control of posture, thus allowing the body to generate larger proprioception-induced afferent volleys to supra-spinal structures [Llewellyn et al., 1990] without generating larger, potentially destabilizing spinal reflexes [Llewellyn et al., 1990; Sibley et al., 2007].

The proprioceptive system is thought to be involved in triggering postural responses to perturbations [Carpenter et al., 1999a]. Therefore, it might be expected that threat-induced changes to proprioceptive function would result in changes to dynamic postural responses. For example, since T-reflexes are increased with postural threat, one would expect to see larger perturbation-induced spinal stretch reflexes with increased threat. Also, it has been suggested that perturbation induced proprioceptive inputs travel to supraspinal centres to trigger postural responses [Nashner & Cordo, 1981; Diener et al., 1984; Jacobs et al., 2008]. As such, postural threat could also have the potential to amplify automatic balance correcting responses, which are

thought to originate supra-spinally, and increase perturbation-induced cortical potentials. Of these three hypotheses, two are indirectly supported by the available literature.

There are two studies that have examined postural responses in detail in situations of increased postural threat. First, Brown and Frank [1997] demonstrated that forward COM displacements were reduced when subjects were pushed forwards while standing at the edge of an elevated platform. Later, Carpenter et al. [2004a] presented subjects with support surface tilts in eight directions in conditions of low and high postural threat to investigate the effects of threat on different perturbation response parameters. They reproduced the effects of threat on COM displacement observed by Brown and Frank [1997]. In addition, Carpenter et al. [2004a] found that while stretch reflexes were not influenced, balance correcting responses were amplified with increased postural threat. If these observations are to be attributed to proprioceptive mechanisms, a plausible explanation would be that muscle spindle sensitivity is increased in the high threat condition. The spindle 'wind-up' would lead to a larger afferent volley to spinal and supra-spinal response triggering centres (e.g. cortex and/or brainstem), which would lead to larger responses. However, the lack of change in spinal stretch reflex amplitudes would imply that the lower motor neuron pool is inhibited in the high threat condition, therefore, muting the effects of larger afferent volleys at the level of the spinal cord. In general, these effects would lead to a shift towards more supra-spinal control of dynamic postural responses in high threat situations.

Studies of the effects of postural threat on perturbation-induced cortical potentials lend further support to the notion of amplified proprioceptive volleys to the cortex in situations of elevated postural threat. Adkin, Campbell, Chua, & Carpenter [2008] demonstrated increased N1 cortical potentials in response to unexpected forward pushes while standing at the edge of an elevated platform. They posited that their findings may indirectly support the hypothesis of

Llewellyn et al. [1990] that threat causes spindle wind-up to increase the amount of afferent information available for the cortex to process the perturbation.

The ability to relate observed changes in dynamic postural responses to underlying proprioceptive mechanisms is hindered by two potentially confounding factors. First, height-induced postural threat is a direction specific stimulus that also elicits global bodily changes. Therefore it is difficult to dissociate the effects of stimulus location from those of the generalized changes in physiological and emotional state in a postural threat paradigm. Typically, the subject stands with their toes at the edge of an elevated platform in a high threat condition in postural threat studies [c.f. Carpenter et al., 1999 b; Carpenter et al., 2001a; Carpenter et al., 2004a; Adkin et al., 2000; Adkin et al., 2008]. This means that the threat is specifically located in front of the subject and that all other directions are (relatively) threat free. Altered kinematic responses to perturbation in the high threat condition of the Brown and Frank [1997] study are difficult to extend to other situations because the subject was always pushed towards the threat. Their data do not speak to whether their subjects would have responded in the same way had they been pushed away from the threat. Carpenter et al. [2004a] tried to address this limitation by keeping the location of the threat constant and changing the direction of perturbation. The perturbations used by Carpenter et al. [2004a] to induce stretch reflexes in SOL were directed away from the edge. Therefore, it is possible that the discrepancy between the stretch reflex results of Study 1 and Carpenter et al. [2004a] is due to the fact that the perturbations had the effect of pushing the subject away from the edge and thus potentially reduced the threat. However, Carpenter et al. [2004a] found that people changed their balance correcting responses to all perturbation directions in the high threat condition. I interpret this finding as evidence that threat induces neuro-muscular changes to postural control that are not necessarily linked to threat location. Further support for this hypothesis could be garnered by

reproducing the results of Carpenter et al. [2004a] in a direction aspecific paradigm with a stimulus that induces the same neuro-muscular changes as height-induced threat.

The second limitation to postural perturbation studies with increased postural threat is that the extent to which the perturbations themselves may induce changes in threat or arousal is not clear. There are two potential confounds that may arise with perturbations: a ceiling effect on stretch reflex facilitation, or a floor effect on inhibition. Perturbations are known to cause a significant change in autonomic arousal that can last up to and beyond 10 sec after a perturbation [Sibley, Mochizuki, Esposito, Camilleri, & McIlroy, 2008]. Therefore, it is possible that the peak excitatory effects of arousal on stretch reflexes were reached as a cumulative effect of the perturbations in the low threat condition in the Carpenter et al. [2004a] study. If there is a limit to the extent arousal can increase stretch reflexes, then the reflex amplitude would not be further increased with the added arousal due to the threat of standing at the edge of an elevated platform. Alternatively, since stretch reflexes are counter-productive to regaining balance after a support surface pitch perturbation [Allum & Honegger, 1992], it is possible that the threat of being perturbed was sufficient to suppress stretch reflexes to their maximum extent. Therefore, once in the high threat condition the subjects in the Carpenter et al. [2004a] study would simply not have been able to further suppress their stretch reflexes with the added motivation of height-induced threat. Either way, it is not clear how experience and expectancy might influence postural responses to perturbations.

At this time, there is no available evidence to directly link threat-induced changes in spinal stretch reflex sensitivity with changes in dynamic postural responses. Therefore, the overall goal of this study was to compare arousal induced changes in reflex sensitivity with perturbation response parameters within the same subjects. This was addressed with a paradigm meant to elicit arousal without the potential confounds of either a direction to be avoided, or the

generalized fear and anxiety associated with height-induced threat. Emotionally charged pleasant pictures have been demonstrated to elicit changes in arousal [Lang, Greenwald, Bradley, & Hamm, 1993; Ribeiro, Teixeira-Silva, Pompéia, & Bueno, 2007; Horslen & Carpenter, unpublished observations] and cause changes in static postural control that mirror the effects of postural threat (i.e. an increase in COP frequency comparable to that typically seen with postural threat) [Horslen & Carpenter, unpublished observations]. As such, pleasant pictures were used in this study to manipulate arousal, instead of postural threat.

There were three specific aims to this study. First, I intended to replicate the results of Carpenter et al. [2004a] with a direction aspecific arousal manipulation to understand the links between arousal-induced stretch reflex changes and dynamic postural responses. I hypothesized that arousal would cause an increase in the amplitude of the stretch reflex and balance correcting responses, as 'wind-up' would cause a larger proprioceptive afferent volley to postural response triggering centres. The second specific aim of this study was to determine the effect of arousal on SOL T-reflex amplitude, and to determine if this effect is dependent on the threat of a perturbation. I hypothesized that T-reflexes would be amplified with arousal, and would be further facilitated when the threat of perturbation was present. Finally, I also explored the effects of arousal and threat of perturbation on H-reflex amplitude. I hypothesized that H-reflexes would not be significantly affected by arousal, yet they would be diminished in the presence of perturbations [McIlroy et al., 2003]. By comparing the results these three aims, I hoped to directly relate changes in lower limb proprioceptive function with perturbation response parameters within the same subjects.

3.2. Methods

3.2.1. Subjects

Sixteen young healthy males between the ages of 19 and 40 (mean ± SE; 23.4 ± 1.23 years) participated in this study. None of the subjects reported a known neurological, vestibular or orthopaedic impairment that would have affected their ability to recover from a postural perturbation. All subjects gave written informed consent prior to participation in the study. The methods used in this study were reviewed and approved by the University of British Columbia Clinical Research Ethics Board (see Appendix A).

3.2.2. Materials

Emotionally charged pictures from the International Affective Picture System (IAPS) database [Lang, Bradley, & Cuthbert, 2008] were used to manipulate arousal in this study. Pictures were projected onto a white screen ~3.7 m in front of the subject, the projected image was 1 m high and 1.3 m wide, the base of the projected image was 1.3 m above the tilting platform on which the subjects stood. 140 pictures were selected (divided into four groups of 35; see Appendix B) with the intention of maintaining a consistent normative valence (level of pleasantness) and eliciting two distinct levels of arousal (Low and High Arousal). Since its inception, IAPS has been gathering normative ratings of arousal and valence for each picture in their database based on the Self-Assessment Manikin (SAM) scale [Bradley & Lang, 1994]; they provide these ratings to researches who subscribe to the system. The SAM scales are essentially a series of 1-9 Likert scales used to rate different theoretical dimensions of emotion. In brief, low values indicate either unpleasantness or low arousal and high values indicate pleasantness or high arousal. The SAM scale will be further discussed in the measures section of this paper. The normative IAPS ratings for the pictures used in this study are : Low Arousal

(group 1) valence 6.53 ± 0.05, arousal 3.52 ± 0.07; Low Arousal (group 2) valence 6.60 ± 0.08, arousal 3.55 ± 0.07; High Arousal (group 1) valence 7.44 ± 0.06, arousal 6.90 ± 0.06; High Arousal (group 2) valence 7.40 ± 0.08, arousal 7.06 ± 0.06. Two picture groups were created for each level of arousal because two experimental conditions were conducted at each level of arousal (perturbations: present, absent), and there is evidence that the emotional effects of a picture are muted on the second exposure to it [Bonnet et al., 1995].

I isolated my sample to males because IAPS normative rankings indicated that the largest arousal range between Low and High Arousal pictures within a consistent valence band could be achieved with pleasant pictures presented to males [Lang et al., 2008], thereby increasing my chances of eliciting a large change in arousal across experimental conditions. Furthermore, pleasant pictures are also known to induce a broader range of T-reflex magnitudes from low to high arousal in seated subjects [Bonnet et al., 1995].

Although pictures can be considered a location-specific stimulus, arousing pleasant pictures do not elicit observable approach or withdrawal behaviours in postural control (i.e. leaning) [Azevedo et al., 2005; Facchinetti, Imbiriba, Azevedo, Vargas, & Volchan, 2006; Horslen & Carpenter, unpublished observations]. As discussed earlier, it is difficult to dissociate the effects of leaning away from the edge in a height-induced postural threat paradigm, from the effects of generalized changes in physiological and emotional state related to the height. Leaning is known to cause changes in perturbation evoked stretch reflex latencies and amplitudes [Diener, Bootz, Dichgans, & Bruzek, 1983], T-reflex amplitudes [Lin, Brown, & Walsh, 1997] and H-reflex amplitudes [Tokuno et al., 2007; Tokuno et al., 2008; Tokuno et al., 2009]. Therefore, by choosing a stimulus to drive arousal that does not induce a lean, I was able to focus on the effects of arousal on dynamic postural control and proprioceptive function, without the potential confounds of leaning away from an edge.

3.2.3. Protocol

Experimental Conditions

Since this experiment was intended to determine how arousal influences both stretch reflexes and balance correcting responses to rotational perturbations in the sagittal plane, I chose to focus my analysis on SOL and compare arousal induced changes in reflex sensitivity with perturbation response parameters within subjects. SOL was chosen because it is a primary balance correcting muscle for anterior-posterior postural perturbations and is also readily available for H- and T-reflex testing. In this study, subjects received a series of perturbations and H- and T-reflexes while exposed to either the Low or High Arousal picture conditions. Furthermore, I was concerned that the threat of perturbation might be sufficient to modulate SOL stretch (T- or H-) reflexes. To accommodate this question, I also exposed subjects to the Low and High Arousal conditions while inducing H- and T-reflexes without the rotational perturbations. Therefore, each subject experienced four experimental trials with two levels of picture induced arousal: Low and High Arousal with perturbations, and Low and High Arousal without perturbations. The order of presentation was counterbalanced across subjects.

Stimulations

H- and T-reflexes were elicited in the same manner, and with the same equipment as in Study 1. Although the results from Study 1 suggested that HPAD was not a factor in soleus reflex modulation with postural threat, the same paired-pulse paradigm was used to reveal the role, if any, of HPAD in dynamic postural control. However, these results will not be analyzed here, as they do not pertain to the specific aims of this study. To be included in the final analysis an H-reflex had to be elicited by a stimulation that elicited an M-wave between 10 to 15% of M_{max}. The peak force of each T-reflex tap to the Achilles' tendon was also recorded (Isotron

Dynamic Force Sensor, Endevco, USA; A/D sampling: 1000 HZ). For each subject a force inclusion range was set to the mean ± 2 SD of the trial with the least tap force variability. To be included in the trial mean, a T-reflex must have been elicited by a tap with a peak force within the inclusion range. There were 15 paired H-reflex and 15 T-reflex stimulations in each of the perturbations absent conditions; and 10 paired H-reflex, 10 T-reflex, and 12 support surface tilts in each of the perturbations present conditions. A minimum of 5 reflexes (H- or T-) within the target stimulation range were required for a trial to be included in the final analysis [Sibley et al., 2007].

Support surface rotations were used as postural perturbations in this study because they elicit stretch and balance correcting responses in separate muscle groups [Allum & Honegger, 1992; Allum, Honegger, & Schicks, 1993], allowing for independent analysis of each phase of the response. It is important to differentiate between the relative phases of responses to perturbations because it has been established that these phases can be modulated independently [Carpenter et al., 2004a]. Each subject responded to six dorsiflexor (toes-up) and six plantar flexor (toes-down) support surface rotations. Two different perturbation directions were used in this study so that the relative response phases could be elicited in different muscle groups. For instance, a toes-up perturbation elicits a stretch reflex in SOL and a balance correcting response in TA, yet a toes-down perturbation stretches TA and requires a balance correcting response from SOL.

Postural perturbations were elicited with support surface rotations in the sagittal plane (7°, 140 ms-long, 50°/sec); the axis of rotation was aligned with the subjects' lateral maleoli. The subjects stood on a 33 cm^2, 43cm high tilting platform, surrounded by a 2.4 x 1.2 m level stage that served to eliminate any threat due to platform elevation (Figure 2-1). Furthermore, 1.5 cm high adjustable heel cups were used to keep subjects' foot position constant throughout the

experiment. To minimize potential first-exposure effects [Keshner, Allum, & Pfaltz, 1987]; the first perturbation (of six) for each direction in each trial was excluded from the final analysis. If for any reason the EMG data from a perturbation were not reliable (e.g. data transmission or recording artefacts), the perturbation was subsequently removed from the analysis. Data from at least four of the remaining five perturbations in each direction was required for a trial to be included in the final analysis. An experimenter was positioned behind the subjects at all times to assist in the event of a loss of balance, none of the subjects lost their balance because of a perturbation.

Trials were designed such that stimulus type and timing would not be predictable, thus ensuring that subjects were not able to anticipate the appropriate postural response to a perturbation [c.f. Burleigh & Horak, 1996], or habituate to the perturbation stimuli [Keshner et al., 1987]. Perturbation and spinal reflex (H- and T-) stimulations were always separated by at least 7 sec to ensure HPAD would not unintentionally influence the responses [c.f. Stein et al., 2007], nor would arousal be artificially elevated from the dynamic response to the previous stimulus [c.f. Sibley et al., 2008].

3.2.4. Measures

Electromyography - Collection

EMG (Telemyo 2400 RG2, Noraxon, USA) was used to record muscle activation with belly-belly preparations (~2 cm apart) on: soleus (SOL), tibialis anterior (TA), rectus femoris, biceps femoris, rectus abdominus, paraspinals, and deltoid. All recordings were taken from the right side, sampled at 3000 Hz, amplified 500x and band-pass filtered between 10 and 1500 Hz online then A/D sampled at 1000 Hz (Power 1401, CED, UK). Resting muscle activity and responses to perturbations were recorded from these muscles.

Electromyography - Reflexes

As in Study 1, separate SOL belly-tendon preparations were also used to measure reflex responses to H- and T-reflex stimuli (Figure 2-2); these data were A/D sampled at 3000 Hz to improve resolution of the reflex waveforms. H-reflex amplitudes were measured as the peak-to-peak amplitude of the H-wave of the first H-reflex in each 200 ms pair. The amplitude was then converted to and reported as a percentage of the individual's maximum M-wave amplitude (% M_{max}) by dividing it by the maximum M-wave peak-to-peak amplitude and then multiplying it by 100.

Electromyography - Perturbations

To calculate the EMG responses to perturbations, the EMG signals were high-pass filtered at 30 Hz and low-pass filtered at 100 Hz offline with digital filters (Spike2, CED, UK), then rectified (Figure 2-3). The onset of EMG activity for each muscle was determined with an algorithm that picked the point where the EMG remained above threshold (mean plus 2 standard deviations of a 500 ms background activity period) for at least 12 of 15 ms (i.e. could not fall below threshold for more than 3-ms). Onset latencies were visually confirmed by the same experimenter for all cases.

Amplitudes for the stretch reflex and balance correcting response phases [Carpenter et al., 2004a] of the response to each perturbation were calculated from the onsets of the EMG activity. Stretch reflex amplitudes were calculated as a 40 ms integrated area starting at the onset of the stretch reflex. Balance correcting responses were calculated as a 100 ms integrated area starting at the onset of the balance correcting response in the non-stretched muscle. The amplitudes of both the stretch reflex and balance correcting responses were normalized to background activity in the respective muscles by subtracting integrated areas of EMG of

comparable lengths (40 ms for stretch, 100 ms for balance correcting responses) starting 750 ms prior to the onset of the perturbation.

Electromyography – Background Muscle Activity

Background muscle activity was calculated prior to each stimulus (H-reflexes, T-reflexes and perturbations) as the root mean square (RMS) error amplitude of the raw EMG signal over a 100 ms period starting 500 ms prior to stimulus onset. These data were then averaged within a trial to get a trial-wide mean. As stated earlier, there were a total of 30 stimulations in each trial in the Perturbations Absent conditions, and 32 stimulations in each trial in the Perturbations Present conditions, all stimulations within a trial were included in the trial average.

Kinematics

Kinematic data were used to ensure a consistent ankle angle was maintained between stimulations [Tokuno et al., 2007; Tokuno et al., 2008; Tokuno et al., 2009]. Kinematic data from the right shank and foot (Optotrak Certus, NDI, Canada) were recorded at a sampling rate of 3000 Hz and used to calculate real-time ankle angle in both the sagittal and frontal planes. Mean ankle angles were calculated with a one-minute quiet standing trial conducted prior to the first experimental trial. Ankle angle had to be within 2 SD of the mean in both planes for a perturbation, H-, or T-reflex stimulus to be elicited during the experimental trials.

Electrodermal Activity

EDA was measured in the same manner and with the same equipment as in Study 1. EDA was calculated for each stimulus as the average conductance level (low-pass filtered at 5 Hz) of a 1 sec window ending 500 ms before stimulus onset. These values were then averaged together within a trial to get the trial-wide mean EDA level. I also calculated dynamic electrodermal

responses (EDRs) to postural perturbations. Two measures were used to quantify EDRs: EDR amplitude, and time to peak EDR (Figure 2-4 A). EDR amplitude was calculated as the difference between the EDR peak and the EDA level preceding the perturbation. Time to peak EDR was calculated as the time between perturbation onset and EDR peak [Sibley et al., 2008]. A change in amplitude of at least 0.5 μS was required for an EDR to be included in the analysis [Sibley et al., 2008]. As with EMG responses to perturbations, the first EDR was excluded from analysis and a minimum of four of the five remaining EDRs had to be usable for a subject to be included in the final analysis.

Psychosocial Measures

Subjective ratings of emotional valence and arousal were collected immediately after each trial with the Self-Assessment Manikin (SAM) scale [Bradley & Lang, 1994]. The SAM scale measures subjective ratings of pleasantness and emotional arousal or intensity on separate nine-point Likert scales. The higher the ratings, the more pleasant or arousing a stimulus is perceived. These ratings were used to measure the emotional effects of the picture stimuli presented in each trial. Furthermore, to facilitate comparison across studies, subjects were asked to rate their perceived fear, stability, and state anxiety with the same questionnaires used in Study 1.

3.2.5. Statistical Analyses

There were two primary independent variables in this study: arousal state (Low and High), and threat of perturbation (absent, present). The dependent variables in this study were: H-reflex amplitude, T-reflex amplitude, SOL background activity, TA background activity, SAM Arousal, SAM valence, EDA, perceived stability, fear, and state anxiety. I also measured amplitudes and latencies for SOL and TA perturbation-induced stretch reflexes and balance correcting responses, as well as EDR amplitude and time to peak. All of these variables except

for those specifically relating to perturbation responses were tested for statistically significant effects in 2 (arousal state) x 2 (threat of perturbation) repeated measures ANOVAs. The dependent measures of perturbation-induced muscle activity were tested in repeated measures t-tests to explore the effects of arousal on perturbation response. EDR amplitude and time to peak were only measured as responses to perturbations; unlike the measures of muscle activity these values are comparable across perturbation direction. Therefore, I used 2 (arousal condition) x 2 (perturbation direction: toes-up, toes-down) repeated measures ANOVAs to explore the possibility of arousal x direction interactions on either EDR amplitude or time to peak. To my knowledge, with the exception of SAM valence and SAM arousal, there is no reason to consider any of the measures in this study as inter-dependent. Therefore, the criterion for statistical significance for the all tests was set to $\alpha = 0.05$, except for SAM valence and arousal. For these variables, a Bonferroni correction for multiple tests was used where α was corrected to 0.025. Effect sizes are reported for all statistical tests, partial eta squared (η_p^2) was used to quantify effects for all ANOVAs, eta squared (η^2) is used to report effects for all repeated measures t-tests [Pallant, 2007]. Although these tests are calculated differently, the values generated are generally considered comparable [Huck, 2008]. Statistics software was used to calculate all statistical tests (SPSS 18, SPSS, Inc., USA) with the exception of η^2 which was calculated manually.

3.3. Results

3.3.1. Subject Exclusions

Of the 16 subjects to complete this study, one subject could not tolerate the H-reflex stimulations and therefore did not provide H-reflex data. Two more subjects were excluded from the H-reflex analysis because they did not have enough reflexes (minimum five) within the target stimulation intensity range (10-15% M_{max}); therefore, 13 subjects were included in the final H-reflex analysis. Five subjects were excluded from the T-reflex analysis because of fluctuations in stimulation intensity between conditions. One subject was excluded from the final perturbation analysis due to equipment malfunction. Also, one subject was excluded from the EDA analysis because EDA data was missing from one of the four trials. Three additional subjects were excluded from the EDR analysis because their EDRs were not sufficiently large enough in one or more conditions to pass the exclusion criteria. Finally, one subject did not answer the fear and perceived stability questionnaires.

3.3.2. Physiological and Emotional Arousal, Psychosocial Scores

Figure 2-5 demonstrates the effects of threat of perturbation and arousal condition on EDA, perceived fear, stability and anxiety. EDA levels were significantly higher during the trials where the threat of perturbation was present ($F_{1,14} = 25.72$, $p < 0.001$, $\eta_p^2 = 0.648$). EDA was not statistically influenced by arousal ($F_{1,14} = 0.027$, $p = 0.871$, $\eta_p^2 = 0.002$), and was not subject to an arousal x threat of perturbation interaction ($F_{1,14} = 0.58$, $p = 0.459$, $\eta_p^2 = 0.04$). After the trials, subjects reported that they experienced more anxiety ($F_{1,15} = 6.07$, $p = 0.026$, $\eta_p^2 = 0.288$) and felt less stable ($F_{1,14} = 12.72$, $p = 0.003$, $\eta_p^2 = 0.476$) when the threat of perturbation was present. Neither of these variables were influenced by arousal (Anxiety: $F_{1,15} = 1.365$, $p = 0.261$, $\eta_p^2 = 0.083$; Stability: $F_{1,14} = 0.758$, $p = 0.399$, $\eta_p^2 = 0.051$), or by an arousal x

threat of perturbation interaction (Anxiety: $F_{1,15} = 0.04$, $p = 0.845$, $\eta_p^2 = 0.003$; Stability: $F_{1,14} = 0.257$, $p = 0.62$, $\eta_p^2 = 0.018$). Experienced fear was influenced by a significant arousal x threat of perturbation interaction ($F_{1,14} = 4.707$, $p = 0.048$, $\eta_p^2 = 0.252$), as well as main effects of arousal ($F_{1,14} = 6.183$, $p = 0.026$, $\eta_p^2 = 0.306$) and threat of perturbation ($F_{1,14} = 12.34$, $p = 0.003$, $\eta_p^2 = 0.468$). Post-hoc analyses revealed that subjects were significantly more afraid in the High Arousal condition than in the Low Arousal condition when the threat of perturbation was present ($t_{14} = -2.60$, $p = 0.021$, $\eta_p^2 = 0.326$). However, there was no significant difference in fear across arousal conditions when the threat of perturbation was not present ($t_{14} < 0.001$, $p > 0.999$, $\eta_p^2 < 0.001$).

SAM ratings of emotional arousal related to the picture stimuli were significantly higher in the High compared to the Low Arousal conditions (Figure 2-6; $F_{1,15} = 113.07$, $p < 0.001$, $\eta_p^2 = 0.883$), though they were not influenced by threat of perturbation ($F_{1,15} = 1.521$, $p = 0.236$, $\eta_p^2 = 0.092$), nor by an arousal x threat of perturbation interaction ($F_{1,15} = 1.05$, $p = 0.323$, $\eta_p^2 = 0.065$). There was a trend to a main effect of arousal on SAM valence ratings ($F_{1,15} = 3.92$, $p = 0.066$, $\eta_p^2 = 0.207$), where valence was on average higher in the High compared to the Low Arousal conditions. SAM valence was not influenced by threat of perturbation ($F_{1,15} = 0.66$, $p = 0.429$, $\eta_p^2 = 0.042$), nor was it influenced by an arousal x threat of perturbation interaction ($F_{1,15} = 3.17$, $p = 0.095$, $\eta_p^2 = 0.175$).

3.3.3. Reflexes

T-reflex amplitudes were significantly larger when the threat of perturbation was present (Figure 2-7; $F_{1,10} = 7.15$, $p = 0.023$, $\eta_p^2 = 0.417$), yet there was no significant effect of arousal ($F_{1,10} = 0.005$, $p = 0.946$, $\eta_p^2 < 0.001$) or arousal x threat of perturbation interaction on T-reflex amplitudes ($F_{1,10} = 0.926$, $p = 0.359$, $\eta_p^2 = 0.085$). H-reflex amplitudes were not influenced by

either threat of perturbation (Figure 2-7; $F_{1,12} = 1.01$, $p = 0.334$, $\eta_p^2 = 0.078$) or arousal condition ($F_{1,12} = 1.14$, $p = 0.307$, $\eta_p^2 = 0.087$), nor were they influenced by an arousal x threat of perturbation interaction ($F_{1,12} = 2.05$, $p = 0.178$, $\eta_p^2 = 0.146$). There were no significant changes in M-wave amplitude across experimental conditions (Arousal: $F_{1,12} = 1.09$, $p = 0.318$, $\eta_p^2 = 0.083$; Threat of perturbation: $F_{1,12} = 0.091$, $p = 0.768$, $\eta_p^2 = 0.008$), nor were they influenced by an interaction ($F_{1,12} = 1.06$, $p = 0.324$, $\eta_p^2 = 0.081$).

3.3.4. Perturbations

The means and standard errors of all perturbation-based measures are contained in Table 2-1. There was no statistically significant effect of arousal on SOL stretch reflex amplitudes ($t_{14} = 0.707$, $p = 0.491$, $\eta^2 = 0.034$) or latencies ($t_{14} = 0.192$, $p = 0.850$, $\eta^2 = 0.003$) in response to a toes-up perturbation. TA stretch reflexes in response to a toes-down perturbation were also unaffected by arousal condition (amplitude: $t_{14} = -0.579$, $p = 0.572$, $\eta^2 = 0.023$; latency: $t_{14} = 1.07$, $p = 0.305$, $\eta^2 = 0.075$). There was no significant change in the TA balance correcting response to a toes-up perturbation across arousal conditions (amplitude: $t_{14} = 0.804$, $p = 0.435$, $\eta^2 = 0.044$; latency: $t_{14} = 1.23$, $p = 0.238$, $\eta^2 = 0.098$). Furthermore, SOL balance correcting responses to toes-down perturbations were not influenced by arousal condition (amplitude: $t_{14} = 1.21$, $p = 0.245$, $\eta^2 = 0.095$; latency: $t_{14} = 0.986$, $p = 0.341$, $\eta^2 = 0.065$).

There was a statistically significant main effect of direction on EDR amplitude ($F_{1,11} = 10.36$, $p = 0.008$, $\eta_p^2 = 0.485$), where EDRs were larger for toes-up perturbations than for toes-down perturbations (Figure 2-4 Part B). There was no significant effect of arousal ($F_{1,11} = 0.109$, $p = 0.747$, $\eta_p^2 = 0.010$), nor was there a significant arousal x perturbation direction interaction on EDR amplitude ($F_{1,11} = 0.746$, $p = 0.406$, $\eta_p^2 = 0.063$). There were no significant main effects of arousal (Figure 2-4 Part C; $F_{1,11} = 0.026$, $p = 0.875$, $\eta_p^2 = 0.002$) or direction ($F_{1,11} = 3.06$, $p =$

0.108, $\eta_p^2 = 0.218$) on EDR time to peak, nor was there a significant arousal x direction interaction ($F_{1,11} = 0.986$, $p = 0.342$, $\eta_p^2 = 0.082$).

3.3.5. Background Muscle Activity

There were no significant main effects of arousal (Figure 2-7; $F_{1,15} = 2.16$, $p = 0.162$, $\eta_p^2 = 0.126$) or threat of perturbation ($F_{1,15} = 0.12$, $p = 0.737$, $\eta_p^2 = 0.008$), nor was there an arousal x threat of perturbation interaction on SOL background activity ($F_{1,15} = 0.63$, $p = 0.438$, $\eta_p^2 = 0.041$). TA background activity was not influenced by threat of perturbation ($F_{1,15} = 1.35$, $p = 0.263$, $\eta_p^2 = 0.083$), however, there was a trend to a decrease in TA background activity in the High Arousal conditions compared to the Low Arousal conditions ($F_{1,15} = 3.47$, $p = 0.082$, $\eta_p^2 = 0.188$). There was no statistically significant arousal x threat of perturbation interaction on TA background activity ($F_{1,15} = 0.27$, $p = 0.612$, $\eta_p^2 = 0.018$).

3.4. Discussion

3.4.1. Arousal Manipulation

Despite a strong effect of picture content on emotional arousal score, the arousal manipulation used in this study failed to elicit a significant physiological response as indicated by tonic EDA levels or perturbation induced EDRs. As such, I was not able address my research questions concerning the effects of arousal on spinal reflexes or perturbation response characteristics in the manner I had intended. It is not clear why I was unable to induce changes in physiological arousal with the materials used in this study, since other studies have demonstrated changes in arousal when comparing pleasant-arousing pictures with pleasant-calming pictures [Ribeiro et al., 2007; Horslen & Carpenter, unpublished observations]. It is possible that the males in this study simply did not find the "arousing" pictures in the High Arousal condition very arousing, or that they had acclimatized to the picture content to an extent where physiological differences were not discernable (pictures presented in two, approximately five minute long blocks per condition).

The majority of the pictures in the High Arousal group were of erotic scenes containing female nudes or heterosexual couples with action sports pictures (e.g. skydiving, white-water rafting) mixed in. There are a number of potential reasons why the subjects in this study might not have demonstrated a change in arousal across conditions. First of all, the subjects may not have been sexually aroused by the pictures. I did not screen for sexual preference or feelings towards erotic materials in this study. As such, I cannot speak to how sensitive to, or how these subjects responded to the materials presented beyond a general assessment of perceived pleasantness and emotional arousal. Though sexual and uncensored, the pictures may not have been appropriate in nature, or sexually intense enough to elicit arousal [*c.f.* Both et al., 2005]. Alternatively, the environment in which the pictures were displayed may have influenced how

the subjects responded to the pictures. Furthermore, when contrasting the Low and High Arousal pictures, the subjects may have been inclined to rate the groups at opposite ends of the emotional arousal scale, creating an artificial gulf in emotional arousal scores which did not accurately reflect their true physiological response.

Finally, it is possible that I failed to demonstrate a change across arousal conditions because of the stimuli I used to evoke reflexes and postural responses. All of the conditions in this study contained unpredictable, destabilizing, and somewhat noxious stimulations. I demonstrated that people were more aroused when the threat of perturbation was present, and quantified the extent to which individual perturbations influenced arousal (with EDR amplitudes). It is likely that the H- and T-reflex stimuli also induced changes in arousal. Furthermore, pictures in the database are known to induce small changes in arousal [Ribeiro et al., 2007]. Therefore, it is plausible that the reflex stimuli themselves acted to elevate arousal to such an extent that the effects of picture content were masked.

3.4.2. Threat of Perturbation

The serendipitous finding that threat of perturbation is enough to cause physiological changes in arousal has allowed us to draw some interesting conclusions about arousal and spinal reflexes that I would otherwise not be able to make. Principally, I am able to support my hypotheses that T-reflexes would be facilitated by arousal and yet H-reflexes would not be significantly influenced. In Study 1, I proposed that T-reflexes were facilitated because of arousal-induced changes in muscle spindle sensitivity related to postural threat. Since threat of perturbation caused significant increases in anxiety and fear, as well as EDA, and since these effects parallel those typically seen when standing in a state of elevated postural threat [Adkin et al., 2002; Carpenter et al., 2006; Brown et al., 2006; Davis et al., 2009; Huffman et al., 2009], I

cannot dissociate the effects of arousal on T-reflex amplitudes from those of generalized postural threat. However, there is a body of non-postural threat literature that suggests T-reflex amplitude is facilitated by arousal [Bonnet et al., 1995; Both et al., 2005; Hjortskov et al., 2005; Kamibayashi et al., 2009], and as such I consider my findings to be supportive of that hypothesis.

I had expected from the outset that the threat of perturbation would induce spinal reflex changes common to postural threat [*c.f.* Sibley et al., 2007; Llewellyn et al., 1990; McIlroy et al., 2003]. However, I was not able to demonstrate the H-reflex inhibition I had hypothesized. It is not clear why I failed to confirm this hypothesis. There are major differences between this, and other studies of postural threat, namely: the location of the threat source, and clear bounds that define the threat (e.g. the edge of an elevated platform). Often, there are clear bounds between areas that are safe and areas to be avoided (i.e. the edge(s) of an elevated support surface), and these bounds present restrictions in the postural responses available to respond to a perturbation (forward stepping is restricted by the edge) [Brown & Frank, 1997; Carpenter et al., 1999b; Carpenter et al., 2001a; Sibley et al., 2007; Llewellyn et al., 1990]. It has been hypothesized that people inhibit the lower motor neuron pool when standing at height to mute involuntary destabilizing reactions that might push them over the edge [Llewellyn et al., 1990; Sibley et al., 2007]. In this study I positioned my subjects on top of the threat source (the tilting platform), and also removed restrictions to forward stepping (by surrounding the platform with a level stage). Therefore, the subjects in this study could not take action to avoid the source of the threat, yet they did have more postural strategies available to respond to a perturbation. Combined, these factors may have reduced the utility of lower motor neuron pool inhibition as a protective strategy. Also, by monitoring ankle angle I was able to ensure that subjects had not leaned in anticipation of being perturbed. I further reduced the likelihood that subjects were

leaning within the acceptable bounds by presenting perturbations in two unpredictable directions, which reduces the utility of leaning as a protective strategy [Carpenter et al., 2004a]. If H-reflex inhibition with postural threat is a consequence of leaning [*c.f.* Tokuno et al., 2007; Tokuno et al., 2008; Tokuno et al., 2009], then by eliminating the drive to lean I may have inadvertently removed the source of H-reflex inhibition.

The purpose of eliciting both spinal reflexes (H- and T-) and postural perturbations in this study was to enable us to compare arousal-induced changes in spinal reflex sensitivity with characteristics of dynamic postural responses. Unfortunately, my failure to manipulate arousal and the lack of change in perturbation response parameters precludes us from making these comparisons. Although I cannot draw new conclusions about the role of spinal stretch reflexes in dynamic postural control, I have gained new insight into the effects of dynamic posturography protocol on spinal stretch reflexes.

Since T-reflexes are amplified simply when the threat of perturbation is present, I must call into question the ecological validity of conclusions about stretch-induced proprioceptive information drawn from dynamic postural control paradigms. Increased muscle spindle sensitivity should increase the amplitudes of the Ia afferent volleys induced by the perturbation. Presumably, this modulation would not be in effect in a fully unexpected and unpractised, "natural" perturbation [Oude Nijhuis et al., 2009]. If dynamic postural responses are at all subject to the amplitude of stretch-induced proprioceptive volleys [Carpenter, Allum, & Honegger, 2001b], then the role of proprioceptive information in dynamic postural control may be inflated in dynamic posturography studies due to the changes in arousal that accompany this scenario.

This issue of a possibly distorted role for proprioceptive information in dynamic postural control is potentially exacerbated by the fact that many studies (including this one) ignore first trials. First trials are often ignored because the response elicited tends to be significantly different from subsequent trials [Keshner et al., 1987; Oude Nijhuis et al., 2009]. As such, there is an emerging trend to examine first exposure and "very first trials" separately from latter ones in an attempt to get a more ecologically valid view of dynamic postural control [Oude Nijhuis et al., 2009; Visser et al., 2010]. Unfortunately, I cannot comment on when the T-reflex modulation occurred in this study. My analysis included all T-reflexes, including those that occurred prior to the first perturbation. The subjects were also made aware of the threat of perturbation prior to their exposure to it. As such, it is possible that the subjects in this study may have been aroused in anticipation of the first perturbation. If this were the case, then it would be likely that they had already modulated their muscle spindle sensitivity prior to the first perturbation. As such, even fully unpractised very-first perturbations in an experiment, where the subject has been informed that they will be perturbed, may not be comparable to fully unexpected "natural" ones. Therefore, it might only be possible to elicit a true ecologically valid perturbation if the subject is deceived and, therefore, fully unaware that they will be perturbed.

3.4.3. Perturbation Direction Effects on EDRs

Another unexpected result to emerge from this study was that EDRs were larger for backward compared to forward perturbations. To my understanding, this is the first study to demonstrate an effect of perturbation direction on EDR amplitude. However, there is evidence to suggest that EDR amplitude is linked to perturbation characteristics, such as speed of perturbation and need to generate a motor response [Sibley, Mochizuki, & McIlroy, 2009]. Due to biomechanical factors such as a larger moment arm anterior to the ankles (pitch perturbations), as well as a smaller base of support posterior to the ankles within which to regain

balance, backward perturbations tend to be more destabilizing [Carpenter et al., 1999a] and are more likely cause a fall when compared to forward ones (young healthy adults: [Oude Nijhuis et al., 2009]; people with Parkinson's disease:[Carpenter, Allum, Honegger, Adkin, & Bloem, 2004b]). Since, backward perturbations induce faster trunk movements [Carpenter et al., 1999a] and require larger corrective responses to maintain stability than forward ones, one would predict that they would elicit larger EDRs [Sibley et al., 2009].

3.4.4. Considerations for Future Research

The major limitation to this study is that I failed to elicit an arousal response that was discernable across conditions. Unfortunately, I cannot discern whether my failure to detect a change is due to a lack of response to the arousing materials, or whether the effects of the materials were masked by arousal induced by the reflex stimulations. In retrospect, it would have been beneficial to have had two extra trials where subjects were exposed to Low and High Arousal pictures without reflex or perturbation stimuli. These added trials would have enabled us to dissociate the arousing effects of the pictures from those of the reflex stimulations. However, it is possible that I failed to demonstrate an arousal effect because the subjects in my study had been over exposed to the picture content. If this were the case, then added exposure to pictures in a control trial would only serve to strengthen this effect. Since pictures have proven unreliable, alternative methods of inducing arousal should be used in future studies of arousal and proprioceptive function in postural control. Alternative methods that have proven feasible for driving arousal and modifying T-reflexes in seated paradigms include: static handgrip, ischemia, and mental arithmetic [Hjortskov et al., 2005; Kamibayashi et al., 2009]. Alternatively, glucose ingestion [Matre & Knardahl, 2003] and pharmacological manipulations of arousal [Hill & Fischer, 1971] might prove useful, as they would likely last long enough to prevent acclimatization, allowing longer collection periods and more data from each subject.

3.4.5. Conclusions

In summation, I have demonstrated that arousal responses to postural perturbations are not consistent across perturbation direction. The role of the autonomic nervous system in postural responses is not yet clear [Sibley et al., 2009]. However, it is clear that autonomic responses to postural perturbations are dependent on perturbation characteristics. I have also garnered further support of the theory that T-reflexes are facilitated by arousal. This finding, in combination with the lack of change in H-reflex amplitude or background muscle activity, suggests that arousal increases muscle spindle sensitivity. Finally, I conclude that postural perturbations used in dynamic posturography pose a postural threat, and that subjects modulate stretch reflexes accordingly. I interpret this finding as evidence that researchers should use caution when extrapolating the role of proprioceptive information, as defined by traditional posturography, to fully unexpected, "natural" postural perturbations.

Chapter 4: Conclusion

I wanted to address two general research aims with this thesis. The first aim was to determine whether significant modulation of spinal stretch reflex pathways could be observed with high postural threat? Evidence for threat-induced modulation of stretch reflex pathways can be drawn from significant increases in T-reflex amplitudes with threat associated with standing on an elevated support surface (Study 1) and with threat of impending postural perturbation (Study 2). The second research aim was to determine whether anxiety and/or arousal mediated changes in stretch reflex function could translate into changes to dynamic postural control. Unfortunately, the arousal manipulation in Study 2 failed to elicit changes in either postural response parameters, or spinal reflex amplitudes. As such, I cannot offer a definitive answer to this question.

Taken as a whole, the most striking result of this thesis is that in both studies I demonstrated that humans can modulate soleus T-reflexes independently from H-reflexes. In fact, I consistently demonstrated concurrent increases in T-reflex amplitudes, baseline EDA, perceived anxiety, and fear; without changes in H-reflexes or background muscle activity. I suggest that changes in muscle spindle sensitivity must be responsible for T-reflex facilitation because modulation at the level of the lower motor neuron pool should influence both H- and T-reflexes similarly. I offer two possible ways to explain changes in muscle spindle sensitivity: (1) γ-motor neuron drive without α-motor neuron activation (α-γ decoupling), or (2) direct sympathetic stimulation of muscle spindles.

I feel that α-γ decoupling is the most feasible explanation for the changes in stretch reflex amplitudes I have observed. There is evidence that animals [Prochazka et al., 1985; Prochazka et al., 1988], as well as some limited evidence from humans [Ribot et al., 1986; Ribot-Ciscar et

al., 2000; Aniss, Diener, Hore, Burke, & Gandevia, 1990a; Aniss, Diener, Hore, Gandevia, & Burke, 1990b], to suggest that γ-motor neurons (which control intrafusal muscle fibre tension) can be activated independently from α-motor neurons (which control extrafusal skeletal muscle). Hypothetically, increased γ-drive with constant α-drive would result in a decrease in muscle spindle firing threshold without a change in background muscle activation, if the muscle is then stretched, then the reflex response would be larger.

The concept of α-γ decoupling in humans is somewhat controversial as some researchers have concluded that it is unlikely that humans selectively modulate γ-motor neuron activity [Gandevia & Burke, 1985; Gandevia, Wilson, Inglis, & Burke, 1997]. However, these conclusions are drawn from studies where subjects attempted to voluntarily activate fusimotor drive in muscles with minimal α-motor neuron activation [Gandevia & Burke, 1985; Gandevia et al., 1997]. In contrast, I have used a paradigm that elicits non-voluntary, sub-conscious behavioural changes [Huffman et al., 2009] and requires tonic muscle activation (in order to maintain upright stance). These conditions are highly similar to those used to demonstrate α-γ decoupling in cats [Prochazka et al., 1988].

An alternative explanation for my findings is that there is a modulatory link between sympathetic drive and muscle spindle sensitivity. As previously discussed, there is a growing body of evidence linking autonomic arousal with T-reflex amplitude [Both et al., 2005; Hjortskov et al., 2005; Kamibayashi et al., 2009] and proprioceptive function [Matre & Knardahl, 2003]. Barker and Saito [1981] have demonstrated that noradrenergic and cholinergic autonomic axons innervate a fraction of cat diaphragm muscle spindles. Moreover, Hunt [1960] demonstrated that sympathetic activity leads to lower spindle firing thresholds and larger stretch-induced afferent volleys in denervated cat postural muscles. Presumably the autonomic and somatic nervous systems act independently [Iversen, Iversen, & Saper, 2000], yet would

have a cumulative effect on muscle spindle sensitivity [Macefield et al., 2003]. As such, a change in spindle sensitivity could be achieved with either fusimotor drive or sympathetic activation. If both α- and γ-drives were held constant and sympathetic drive to the muscle spindles was increased, then one would expect amplified stretch reflexes without a significant change in background muscle activation.

Sympathetic drive to muscle spindles is still a somewhat controversial explanation for changes in stretch reflex amplitudes. For example, both Passatore et al. [1996] and Hellström et al. [2005] failed to demonstrate a link between cervical sympathetic nerve stimulation and afferent firing rates in reduced animal preparations. Furthermore, Macefield et al. [2003] were not able to demonstrate a change in human Ia afferent firing frequency when subjects induced arousal with prolonged breath holds. Presumably, such a change in arousal would influence T-reflex amplitude [Hjortskov et al., 2005; Kamibayashi et al., 2009]; however, T-reflexes were not tested in Macefield et al.'s [2003] study. It should be noted that Macefield et al.'s [2003] results are based on recordings from quiescent muscles. Therefore, it is unknown whether some underlying fusimotor or α-motor drive is required for the sympathetic influence on muscle spindle to take effect [Macefield et al., 2003].

Since it is not clear how, or if, sympathetic and fusimotor drive interact to modulate muscle spindle sensitivity, I propose an experiment to address the issue. To my knowledge, there is no accepted method to control fusimotor drive in humans. However, autonomic arousal can be pharmacologically controlled. I propose that H- and T-reflexes should be tested in a high postural threat scenario while subjects are in a pharmacologically induced hypo-aroused state. Ideally, the postural threat should induce fear and anxiety, without the sympathetic drive that normally accompanies these effects. If T-reflex amplitudes are increased with threat, despite constant sympathetic drive and without concurrent changes in H-reflexes or background muscle

activity, then increased fusimotor drive and α-γ decoupling could be the only feasible explanation for the hypothetical result. Alternatively, a negative or even a null result would not rule out a role for α-γ decoupling in stretch reflex modulation, it would however demonstrate that arousal is required for stretch reflex modulation with increased postural threat.

In both of the experiments presented in this thesis I tested H- and T-reflexes after manipulating conditions of postural threat. In Study 1, I manipulated threat by having my subjects stand at low and high support surface heights, where the risk of injury associated with a fall is increased and forward stepping was restricted. In Study 2, I used the threat of perturbation as a postural threat. In this case the risk of injury from a fall was the same across conditions, yet the likelihood of a fall was increased. H-reflexes are typically inhibited when people stand on elevated support surfaces [Sibley et al., 2007], are threatened with perturbation [McIlroy et al., 2003], or perform a novel balance task [Llewellyn et al., 1990]. Despite implementing similar conditions, I failed to demonstrate an inhibitory effect of postural threat in either of my paradigms. In Study 1 I discussed at length potential confounds that might explain my failure to replicate previous results. A major difference in protocol between previous studies and Study 1 was the use of ankle braces to restrict sway. Interestingly, when the ankle braces were removed from the protocol for Study 2, the results did not change. Therefore, it seems that my dismissal of the ankle braces as a confound was ultimately justified. Furthermore, this second failure to replicate an attenuation of H-reflexes in a condition of elevated postural threat calls into question the strength of the effects previously reported, as well as my interpretation of their relevancy to postural threat.

Implications for Postural Control

The general research aims for this thesis were developed in consideration of behavioural responses to postural threat that manifest in postural control. Specifically, my first general research question was intended to determine if height-induced postural threat induces changes in spinal stretch reflex function that could explain why people adopt stiffer postural sway at height [Carpenter et al., 1999b;Carpenter et al., 2001a]. I have concluded that the observed changes in T-reflex amplitudes are the result of increased muscle spindle sensitivity. An increase in muscle spindle sensitivity should translate into more frequent and larger sway-induced afferent volleys in unperturbed quiet stance. The increase in afferent flow to the spinal cord should translate into increased postural muscle activation, and ultimately, increase sway stiffness. Therefore, I can support increased muscle spindle sensitivity induced by postural threat as a viable explanation for increased sway stiffness.

The findings of this thesis leave many potential avenues for future postural control research. Despite the fact that I have clearly demonstrated the capacity for arousal induced changes in stretch reflex function, the effects of arousal on dynamic postural control remain unresolved. The effects of postural threat on dynamic postural responses are established [Brown & Frank, 1997; Carpenter et al., 2004a], and yet the role arousal plays in these effects is still not clear. It would be beneficial to re-address the research aims of Study 2 with a more potent arousal stimulus to determine how arousal influences dynamic postural control. As discussed in Study 2, the threat of perturbation is enough to elevate arousal. Therefore, it will be crucial to define the effects of arousal on dynamic postural control if laboratory findings are to be accurately translated to “natural”, fully unexpected perturbations.

There is an emerging trend in dynamic posturography to studying very first trials as more ecologically valid indicators of dynamic postural control [Oude Nijhuis et al., 2009; Visser et al., 2010]. However, in light of the findings presented here, it would be important to determine when threat-induced arousal occurs, whether threat-induced arousal habituates, and to define the effects of arousal on postural responses. First trial responses tend to be larger and less effective at restoring balance [Oude Nijhuis et al., 2009], and have been described as 'startle-like' [Hansen, Woollacott, & Debu, 1988] because they appear to contain protective startle responses overlaid onto balance corrective responses [Oude Nijhuis et al., 2009]. It is possible that these larger, 'startle-like' first trial responses are a consequence of elevated arousal, as arousal has been indirectly linked to startle response amplitude [Lang, Bradley, & Cuthbert, 1990]. Furthermore, it is unknown whether arousal diminishes with repeated exposure to postural perturbations. If there is a link between arousal and dynamic postural control, and if arousal habituates with exposure to postural perturbations, then dynamic postural response habituation [Keshner et al., 1987; Oude Nijhuis et al., 2009] might be linked to arousal.

Finally, the samples used in these studies were comprised of young healthy adults, yet one of the ultimate goals of posturography is to understand the causes of, and prevent falls in individuals with balance impairments [Visser, Carpenter, van der Kooij, & Bloem, 2008]. There are established links between fear of falling and balance performance in older adults [Maki et al., 1991] and persons with Parkinson's disease [Adkin, Frank, & Jog, 2003]. Furthermore, it has been established that postural threat induces both anxiety and physiological arousal in older adults [Carpenter et al., 2006; Brown et al., 2006], and that fear and arousal are linked in young healthy adults [Davis et al., 2009]. What remains unknown is the role that arousal and arousal-induced changes to proprioceptive function might play in the postural control deficits that occur in older adults and persons with Parkinson's disease.

I have demonstrated a strong link between postural threat-induced arousal and stretch reflexes with these studies. These findings have implied a level of proprioceptive control previously dismissed in humans. Specifically, I feel that these data suggest that muscle spindle sensitivity can change without underlying changes in lower motor neuron pool, or tonic muscle activation state. Ultimately, I feel that these data will provide a foundation from which to tackle many issues in postural control. From basic questions concerning interplay between the somatic and autonomic nervous systems, to global issues concerning the application of posturography; I feel that these studies can significantly contribute to our understanding of human postural control.

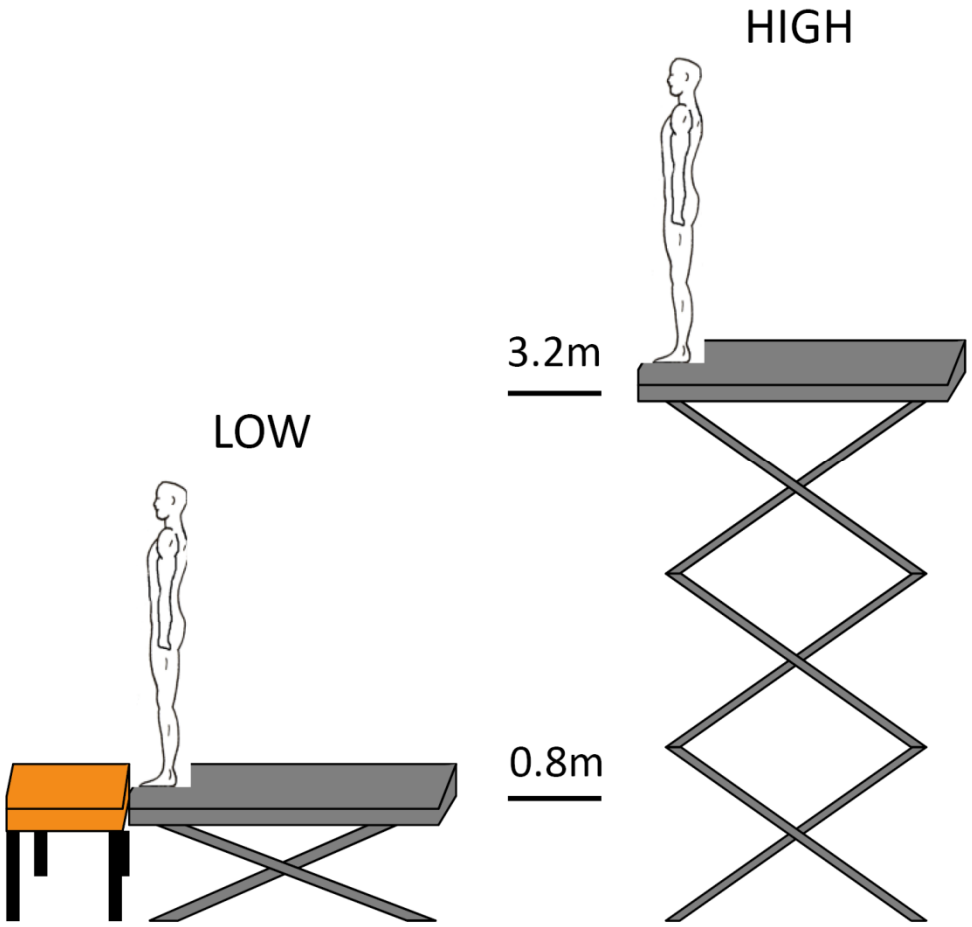

Figure 1- 1: The two conditions of postural threat used in Study 1. In the LOW condition a table was used to extend the support surface in front of the subject, the table was removed in the HIGH condition so that the distance between the subject and the floor was 3.2 m.

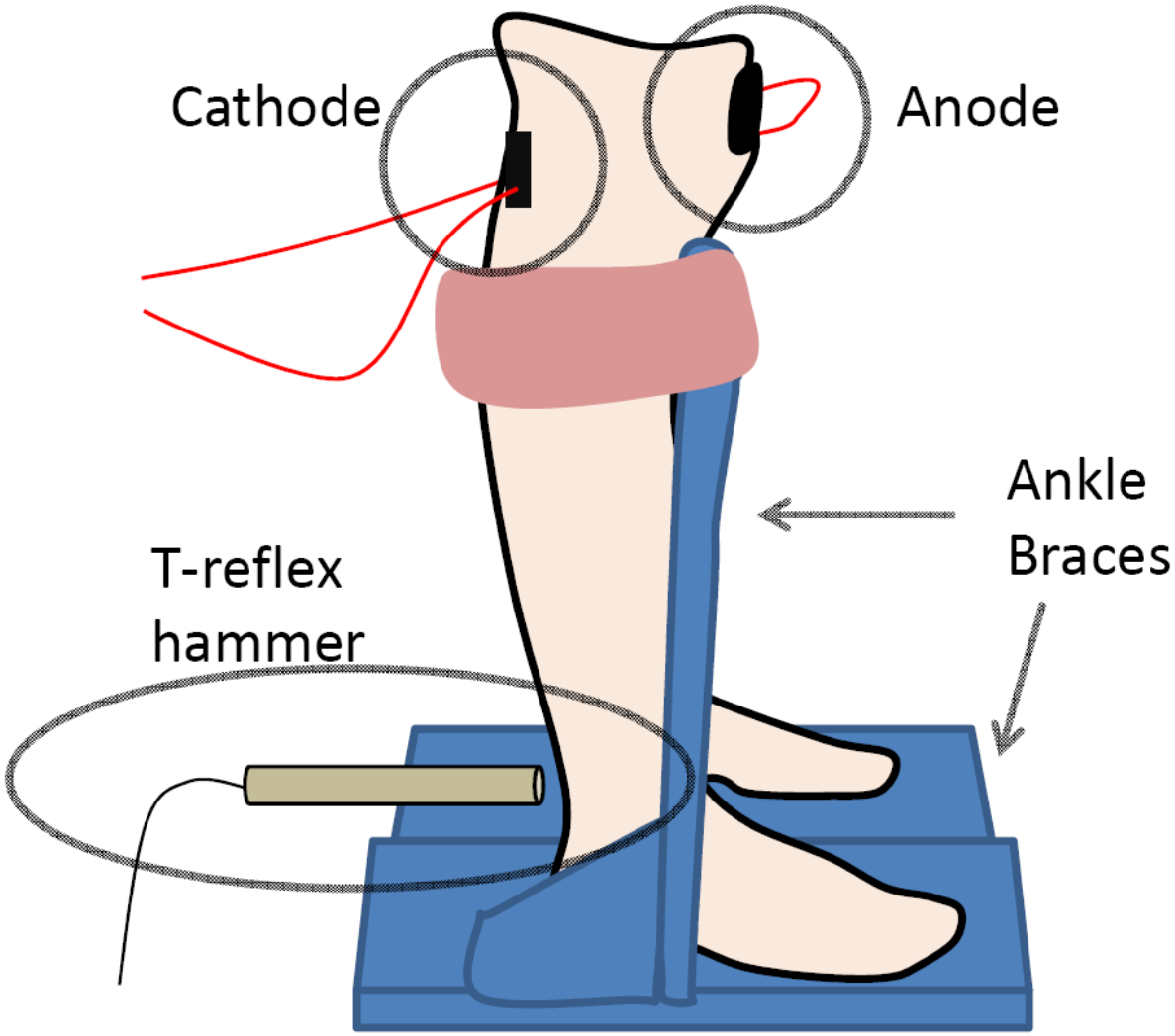

Figure 1- 2: The ankle bracing system used in Study 1. Also depicted are the H-reflex anode and cathode placements, as well as the T-reflex hammer location.

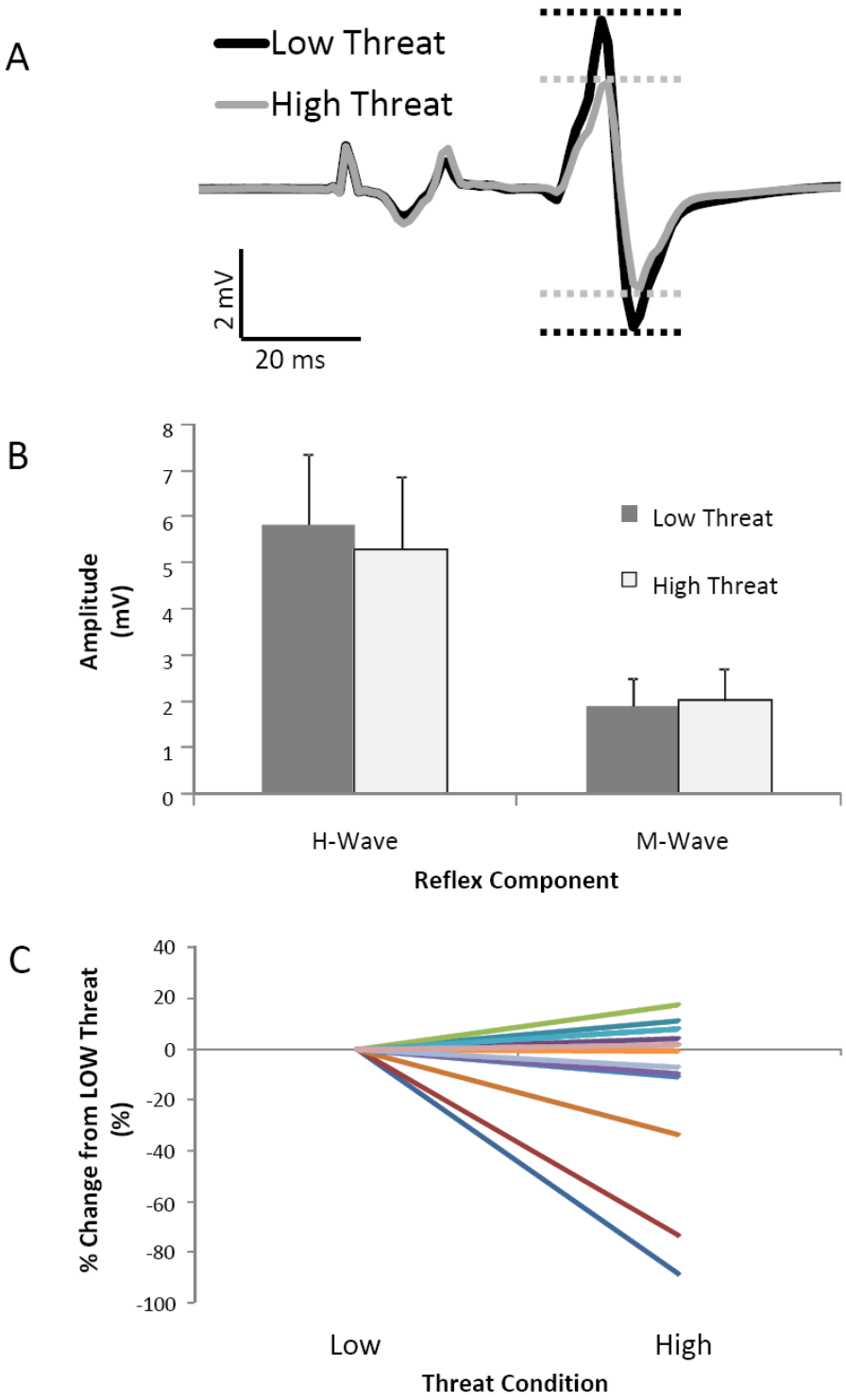

Figure 1- 3: (Part A) Single H-reflex traces from LOW and HIGH threat conditions from a representative subject. (Part B) Mean (+SE) H-reflex and M-wave amplitudes for LOW and HIGH postural threat conditions. (Part C) H-reflex percent change from LOW to HIGH threat condition is presented for each subject.

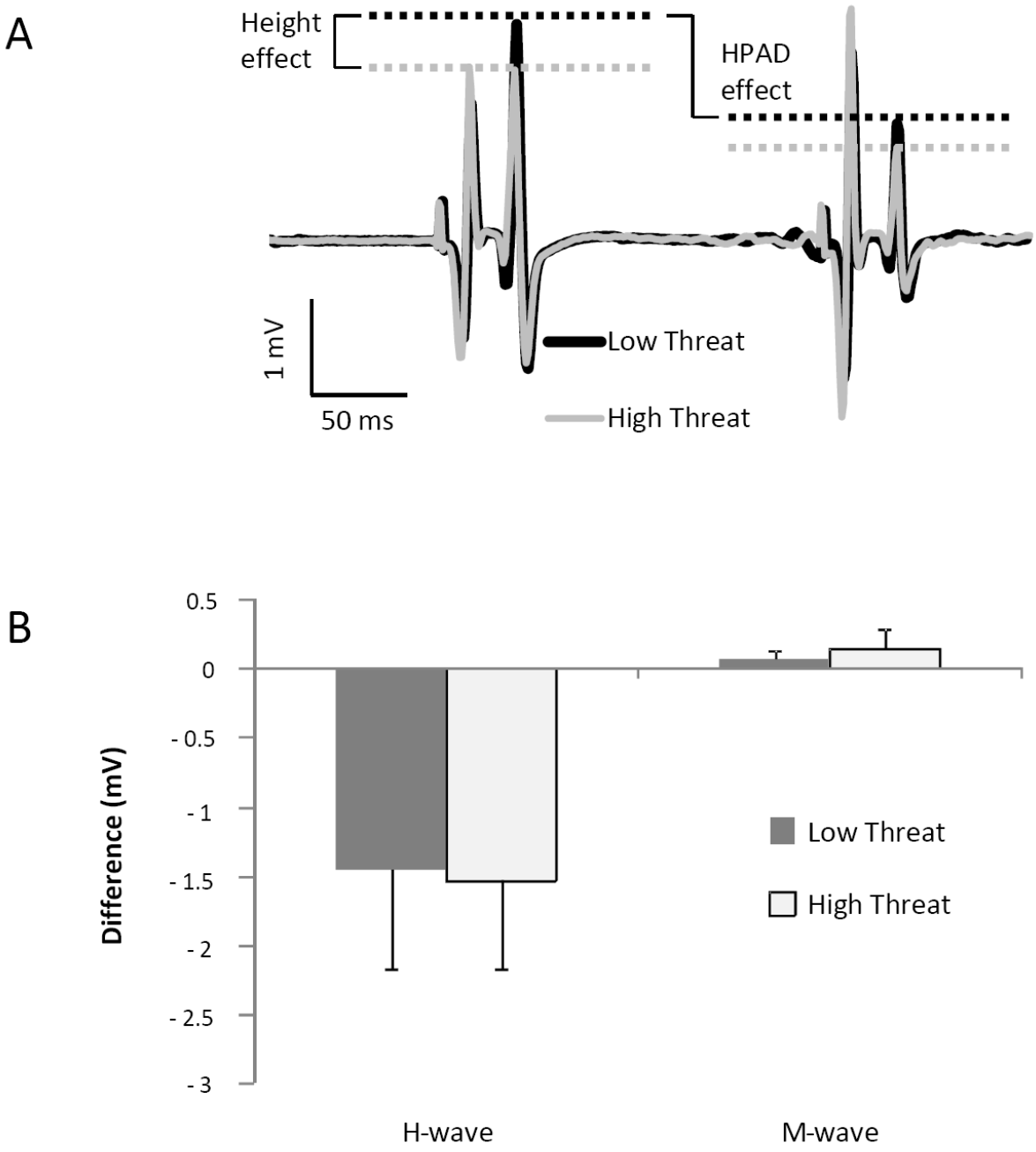

Figure 1- 4: (Part A) Paired H-reflex traces from LOW and HIGH conditions from a representative subject with HPAD effects highlighted. (Part B) Mean (+SE) calculated HPAD effects for LOW and HIGH conditions in Study 1. Also presented are the mean changes in M-wave amplitude from first to second stimulus for each of the threat conditions.

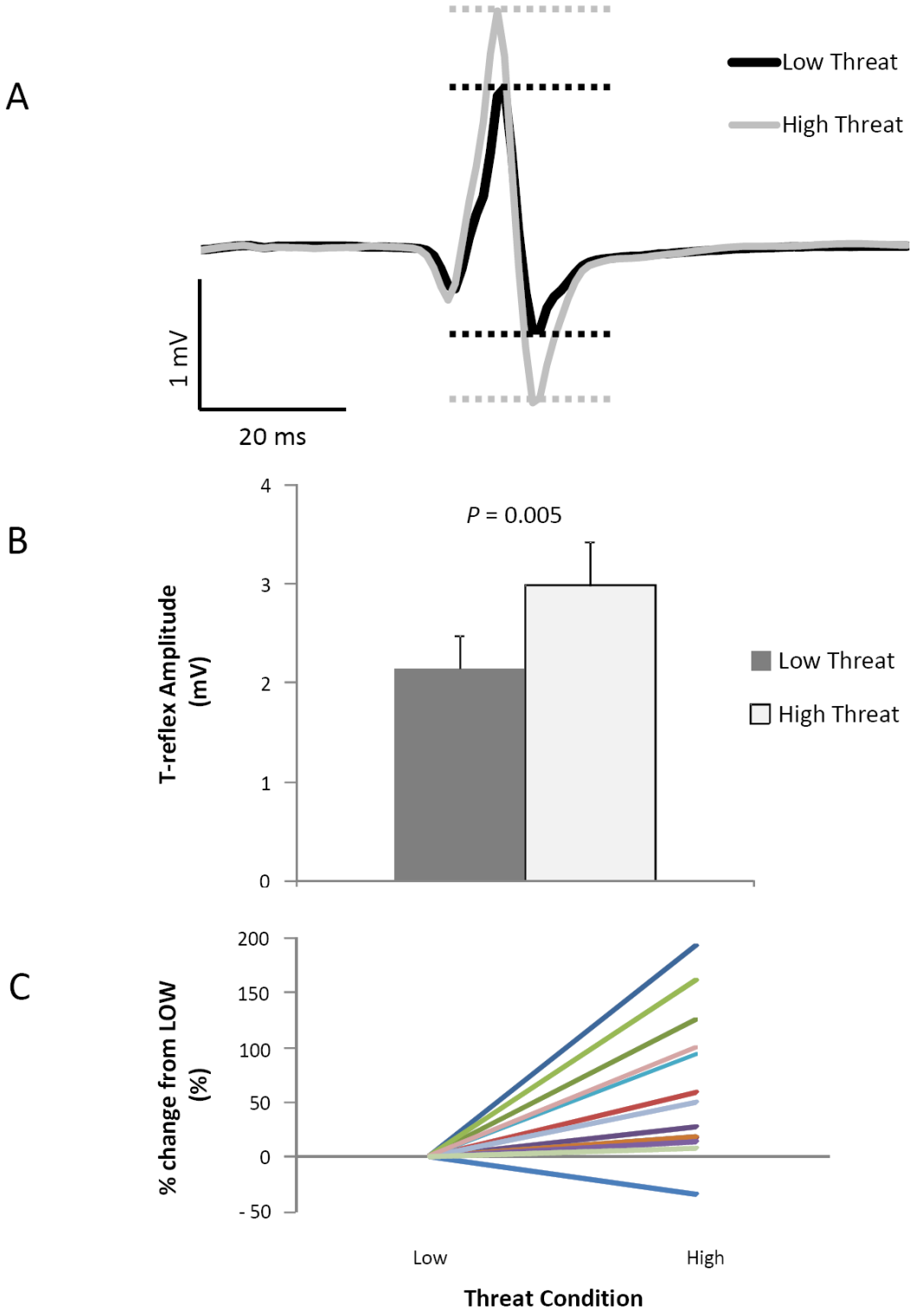

Figure 1- 5: (Part A) Single T-reflex traces from LOW and HIGH conditions from a representative subject. (Part B) Mean (+SE) T-reflex amplitudes for LOW and HIGH postural threat conditions. (Part C) Percent change from LOW to HIGH threat condition is presented for each subject.

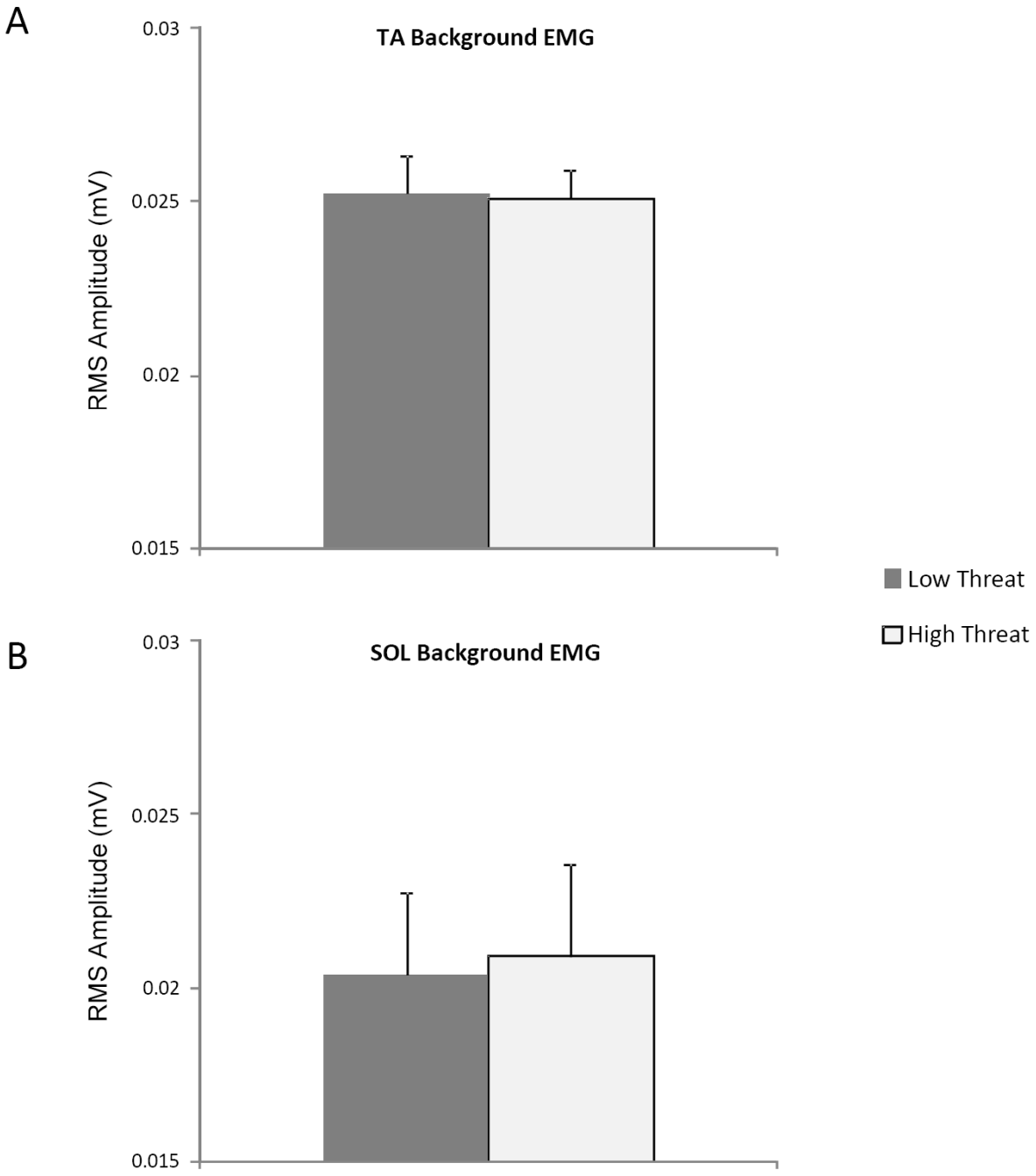

Figure 1- 6: Mean (+SE) pre-stimulus background EMG for TA (part A) and SOL (part B).

Figure 2- 1: Image of the experimental setup used in Study 2. The tilting platform (white square) was positioned in the middle of a 2.4 x 1.2 m stage and heel cups were positioned around the subjects' feet to ensure that they maintained a consistent foot placement. The emotionally-charged pictures were projected onto a screen positioned approximately 3.7 m in front of the subject, the projected image was 1 m high and 1.3 m wide, the base of the image was 1.3 m above the stage.

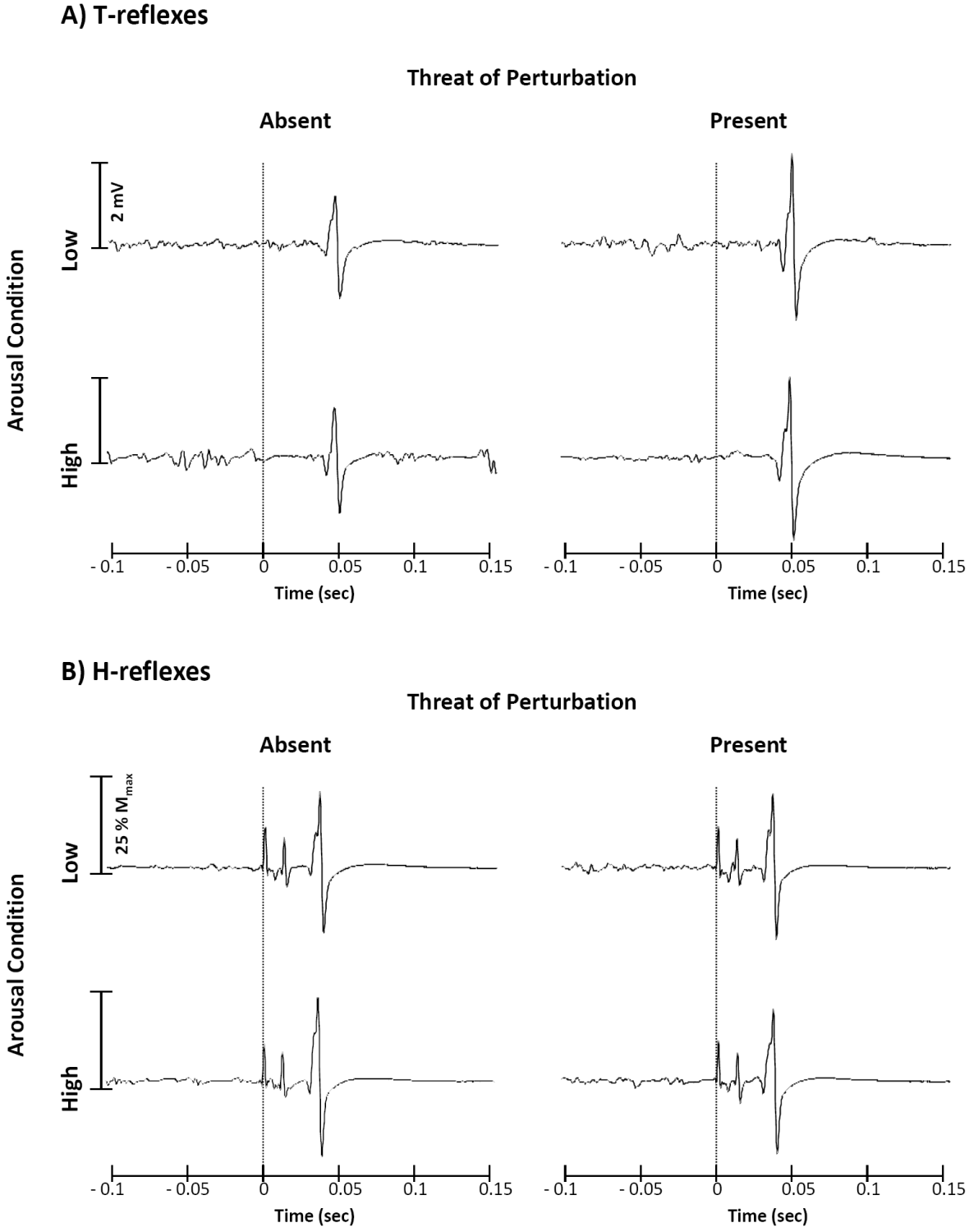

Figure 2- 2: Single T-reflex (Part A) and H-reflex (Part B) traces from a representative subject. The traces are grouped by arousal condition (horizontally) and threat of perturbation (vertically). Time zero indicates stimulus onset; also marked as a dashed vertical line in the T-reflex traces.

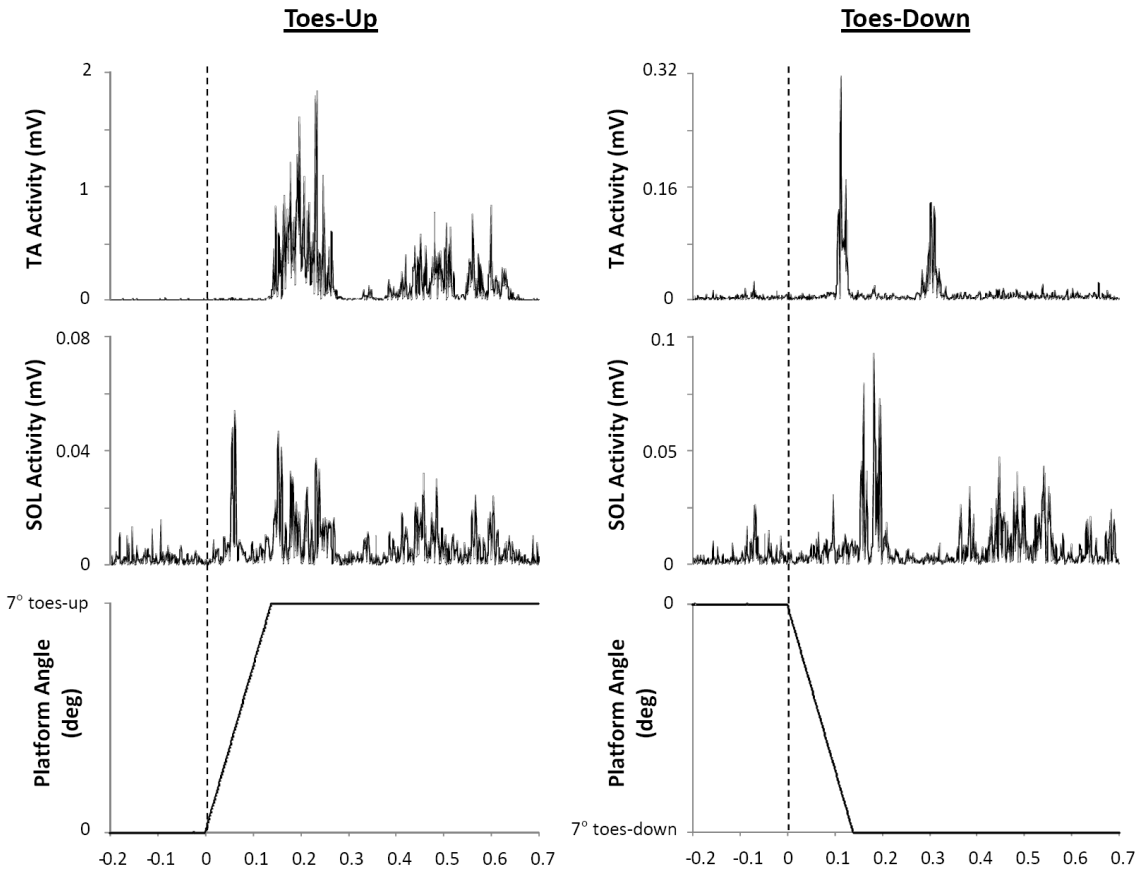

Figure 2- 3: EMG and platform angle traces from a single toes-up (left column) and toes-down (right column) perturbation from a representative subject. In the toes-up perturbation SOL is stretched and TA acts as a balance corrector, in the toes-down perturbation TA is stretch and SOL generates a balance correcting response. The dashed vertical lines indicate the onset of platform movement.

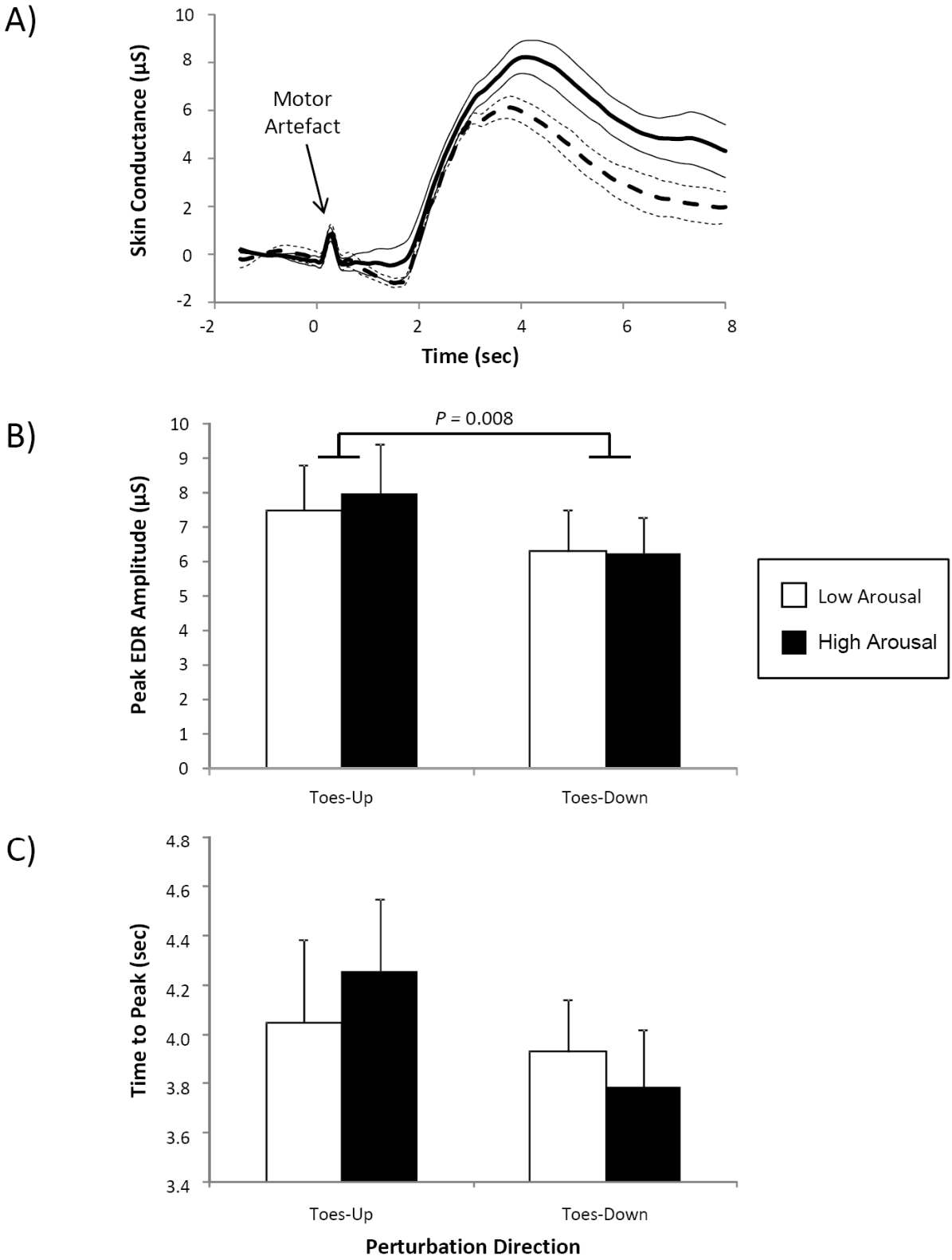

Figure 2- 4: (Part A) Single trial averages (±SE) of 5 toes-up (solid lines) and 5 toes-down (dashed lines) EDRs from a representative subject. For graphical purposes these data have been normalized to a 1 sec pre-perturbation baseline. Also depicted are group means (+SE) for peak EDR amplitude (Part B) and time to peak amplitude (Part C).

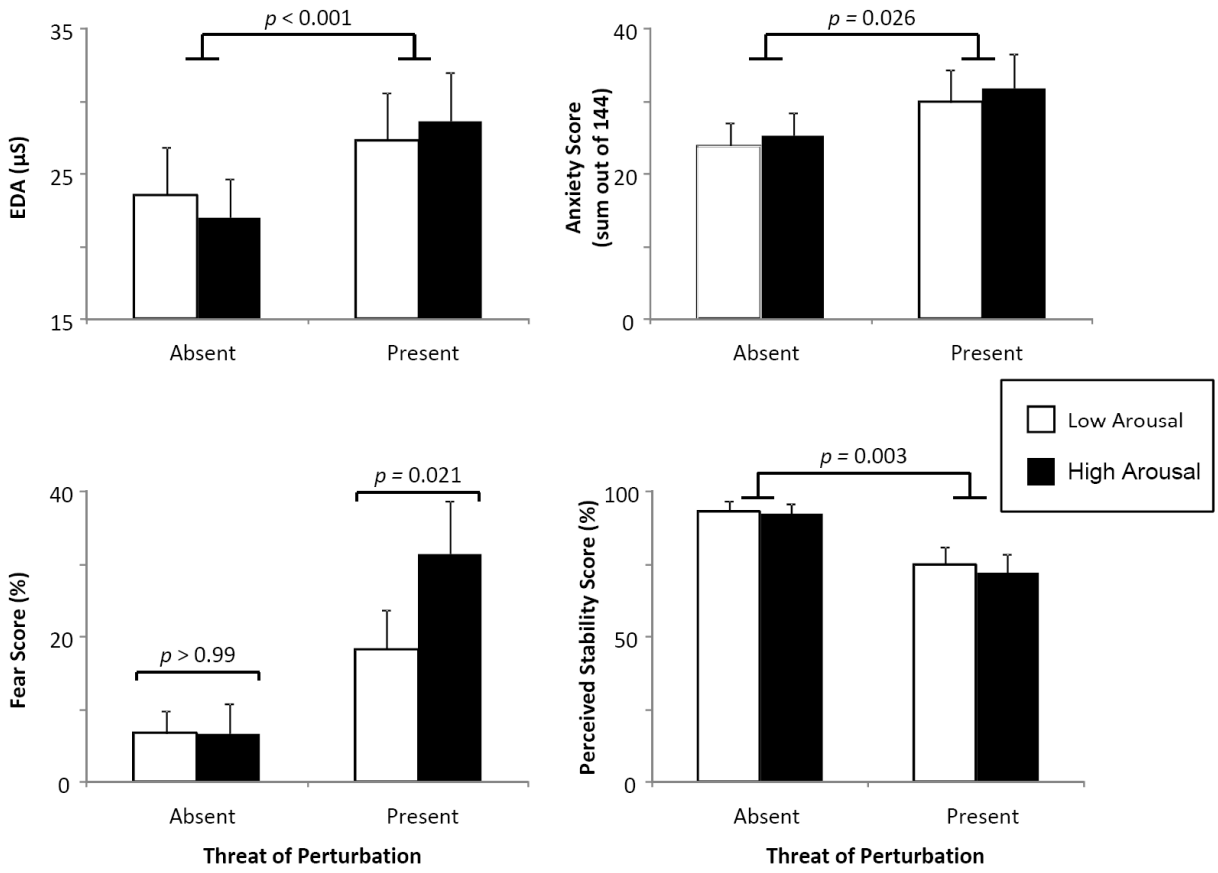

Figure 2- 5: Threat of perturbation effects on EDA, anxiety, fear and perceived stability group means (+SE) from Study 2. The criterion for statistical significance was set to $\alpha = 0.05$ for all comparisons depicted.

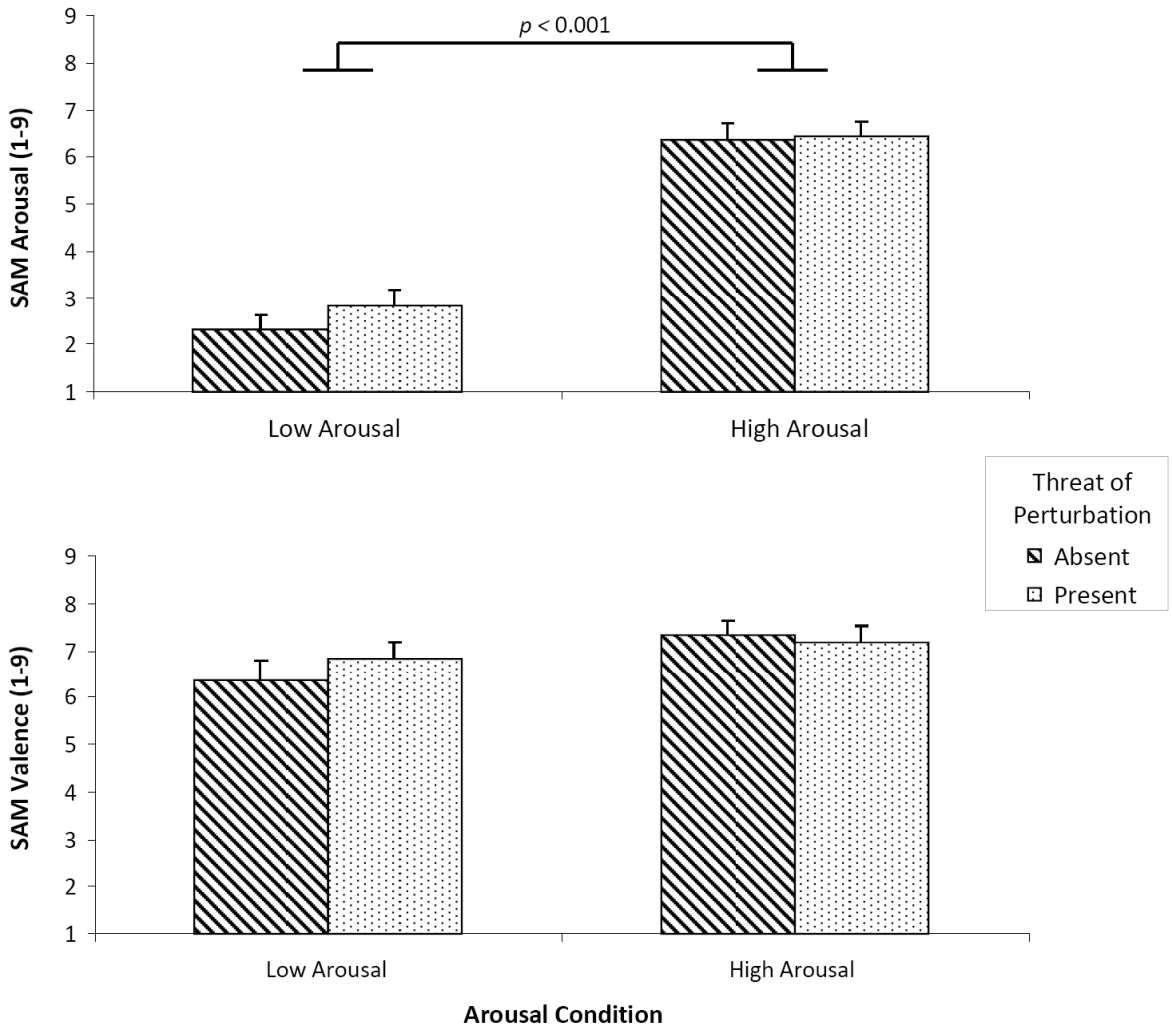

Figure 2- 6: Mean (+SE) ratings of picture arousal (SAM arousal) and picture valence (SAM Valence) by condition.

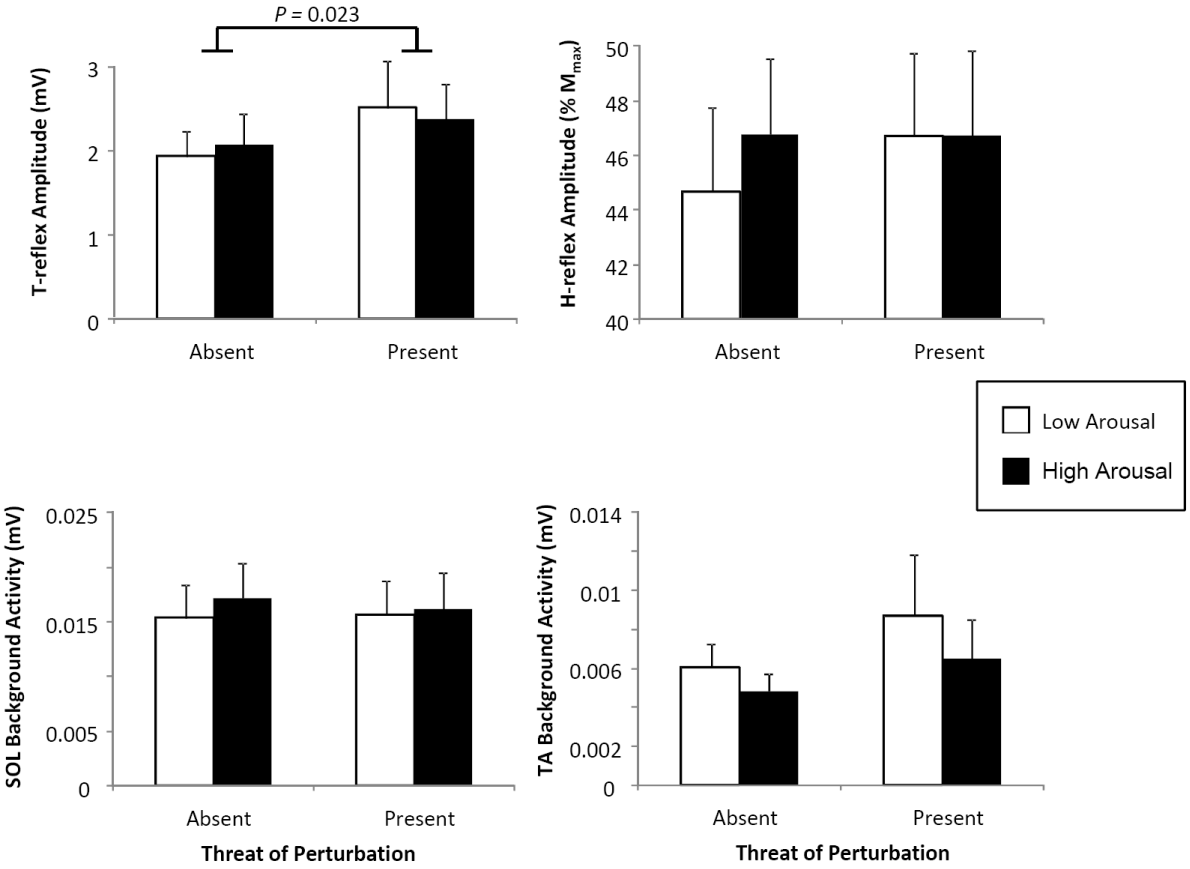

Figure 2- 7: Group mean (+SE) T- and H-reflex amplitudes, as well as mean (+SE) background EMG activity (RMS amplitude) for SOL and TA. The criterion for statistical significance was set to $\alpha = 0.05$ for all comparisons depicted.

Table 2- 1: Mean (SE) soleus and tibialis anterior stretch reflex and balance correcting response amplitudes and latencies in response to toes-up and toes-down postural perturbations.

	Toes-Up				Toes-Down			
	SOL Stretch Reflex		TA Balance Correcting		TA Stretch Reflex		SOL Balance Correcting	
Arousal	Amplitude (μV • s)	Latency (ms)	Amplitude (μV • s)	Latency (ms)	Amplitude (μV • s)	Latency (ms)	Amplitude (μV • s)	Latency (ms)
Low	**2.50**	**46.14**	**25.73**	**131.93**	**5.11**	**94.41**	**4.94**	**154.42**
	(0.45)	(0.86)	(2.68)	(3.64)	(0.59)	(2.11)	(1.1)	(6.61)
High	**2.42**	**46.00**	**25.14**	**129.06**	**5.32**	**93.27**	**4.50**	**149.98**
	(0.41)	(0.87)	(2.56)	(3.17)	(0.64)	(2.39)	(0.91)	(5.23)

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Appendix A: UBC Clinical Research Ethics Board Certificate of Approval.

The University of British Columbia
Office of Research Services
Clinical Research Ethics Board – Room 210, 828 West 10th Avenue, Vancouver, BC V5Z 1L8

ETHICS CERTIFICATE OF EXPEDITED APPROVAL: RENEWAL

PRINCIPAL INVESTIGATOR:	DEPARTMENT:	UBC CREB NUMBER:
Mark G Carpenter	UBC/Education/Human Kinetics	H06-70316

INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT:

Institution	Site
UBC	Vancouver (excludes UBC Hospital)

Other locations where the research will be conducted:
N/A

CO-INVESTIGATOR(S):
Bastiaan Bloem
Justin R. Davis
Allan Adkin
Adam Campbell
Romeo Chua
Brian C. Horslen

SPONSORING AGENCIES:
- Natural Sciences and Engineering Research Council of Canada (NSERC) - "Central and Peripheral Mechanisms Controlling Human Balance Control"
- Natural Sciences and Engineering Research Council of Canada (NSERC) - "State-anxiety Effects on Corticol Response to Predictable and Unpredictable Balance Perturbations"

PROJECT TITLE:
Central and Peripheral Mechanisms Controlling Human Balance Control

EXPIRY DATE OF THIS APPROVAL: July 23, 2011

APPROVAL DATE: July 23, 2010

CERTIFICATION:
In respect of clinical trials:
1. The membership of this Research Ethics Board complies with the membership requirements for Research Ethics Boards defined in Division 5 of the Food and Drug Regulations.
2. The Research Ethics Board carries out its functions in a manner consistent with Good Clinical Practices.
3. This Research Ethics Board has reviewed and approved the clinical trial protocol and informed consent form for the trial which is to be conducted by the qualified investigator named above at the specified clinical trial site. This approval and the views of this Research Ethics Board have been documented in writing.

The Chair of the UBC Clinical Research Ethics Board has reviewed the documentation for the above named project. The research study, as presented in the documentation, was found to be acceptable on ethical grounds for research involving human subjects and was approved for renewal by the UBC Clinical Research Ethics Board.

Approval of the Clinical Research Ethics Board by one of:

Dr. Peter Loewen, Chair
Dr. James McCormack, Associate Chair

Appendix B: Emotionally charged pictures used in Study 2, listed by group and sorted by IAPS identification number.

Low Arousal Group 1		Low Arousal Group 2		High Arousal Group 1		High Arousal Group 2	
Name	IAPS ID#	Name	IAPS ID#	Name	IAPS ID#	Name	IAPS ID#
Women	1340	Birds	1419	Erotic Female	4002	Erotic Female	4001
Giraffes	1601	Polar Bears	1441	Erotic Female	4006	Attractive Fem	4007
Butterfly	1603	Rabbit	1610	Attractive Fem	4071	Erotic Female	4008
Butterfly	1604	Antelope	1620	Erotic Female	4141	Erotic Female	4085
Butterfly	1605	Cow	1670	Erotic Female	4142	Bikini	4090
Grouper	1910	Elephants	1812	Erotic Female	4220	Erotic Females	4130
Mother	2170	Fish	1900	Erotic Female	4220	Erotic Female	4180
Boys Reading	2222	Turtles	1942	Erotic Female	4225	Erotic Female	4210
Kids	2274	Kid	2035	Erotic Female	4240	Erotic Female	4232
Child Camera	2302	Father	2165	Attractive Fem	4250	Erotic Female	4235
Girl	2304	Neutral Baby	2260	Erotic Female	4300	Erotic Female	4290
Mother	2310	Family	2299	Erotic Female	4310	Erotic Female	4302
Chef	2331	Binoculars	2314	Attractive Fem	4325	Erotic Female	4311
Family	2360	Children	2341	Erotic Couple	4607	Erotic Couple	4649
Three Men	2370	Family	2358	Erotic Couple	4608	Erotic Couple	4652
Family	2395	Fisherman	2384	Erotic Couple	4647	Erotic Couple	4658
Couple	2501	Kids	2387	Erotic Couple	4651	Erotic Couple	4659
Balloons	2791	Kids	2388	Erotic Couple	4670	Erotic Couple	4660
Romance	4614	Man with Fish	2392	Erotic Couple	4681	Erotic Couple	4664
Flower	5000	Picnic	2560	Erotic Couple	4683	Erotic Couple	4664.1
Flower	5010	City	2594	Erotic Couple	4687	Erotic Couple	4668
Farmland	5720	Family	2598	Erotic Couple	4692	Erotic Couple	4698
Field	5725	Sunflower	5001	Erotic Couple	4693	Female Kiss	4770
Nature	5750	Flowers	5200	Erotic Couple	4694	Erotic Couple	4810
Field	5764	Nature	5201	Erotic Couple	4695	Hiker	5629
Courtyard	5779	Garden	5202	Erotic Couple	4697	Lightning	5950
Flowers	5811	Nature	5250	Erotic Couple	4698	Skier	8030
Clouds	5870	Clouds	5551	Erotic Couple	4800	Sailing	8080
Clouds	5891	Field	5711	Sky Divers	5621	Parachute	8163
Watermelon	7325	Grain	5726	Hockey	8117	Cliff diver	8178
Ice Cream	7340	Nature	5760	Sailboat	8170	Bungee	8179
Ocean	7545	Leaves	5800	Cliff Divers	8180	Skydivers	8185
Rower	8050	Seagulls	5831	Skysurfer	8186	Motorcyclist	8260
Winner	8330	Ferry	7489	Rafters	8400	Rollercoaster	8499
Carnival Ride	8497	Violin	7900	Rollercoaster	8492	Money	8501