Use of bracing and EMG biofeedback to investigate the relationship between soleus and gastrocnemius excitation during cycling

by

Julia Wilkes

B.P.H.E., B.Sc., Queen's University, 2006

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Abstract

Introduction

Our basic understanding of muscle synergies is incomplete. The interaction among the triceps surae muscles (medial and lateral gastrocnemius, soleus) is still under investigation. Studies have shown that these muscles respond differently to cadence manipulations. These excitation differences might relate to the role of gastrocnemius in knee flexion, a role that soleus does not serve.

Purpose

The purpose was to investigate the response of the medial and lateral gastrocnemius muscles when soleus excitation was eliminated using bracing and biofeedback during cycling.

Methods

Participants cycled under braced and unbraced conditions over two sessions. During each session, cycling protocol involved a normalization ride with no brace and no feedback, followed by a second ride without feedback, and a prolonged ride with feedback. The biofeedback consisted of a moving bar graph representing the average soleus excitation for the first half of the pedal cycle and was updated with every pedal stroke. Electromyography of seven muscles was collected and analyzed.

Results

In the unbraced condition, soleus excitation was not modified with visual biofeedback. While wearing the brace, the integrated electromyography (iEMG) of all triceps surae muscles decreased by 30%. With the addition of EMG biofeedback, soleus and lateral gastrocnemius iEMG decreased a further 26% and 21% respectively by the end of the feedback period while medial gastrocnemius excitation did not change. Tibialis anterior excitation was significantly increased while rectus femoris and biceps femoris excitation did not change. Gluteus maximus iEMG decreased with bracing and biofeedback.

Conclusions

While unbraced, soleus excitation was not reduced with biofeedback as it is difficult to modify joint position in this learned motor task. When the known task was modified by applying an ankle-foot orthosis, participants successfully modified soleus excitation under less-familiar task requirements. Participants voluntarily activated the tibialis

anterior muscle, and it is proposed that through reciprocal inhibition pathways, soleus EMG was reduced. Lateral gastrocnemius excitation also decreased. With soleus excitation decreased, medial gastrocnemius excitation was unchanged likely due to its on-going role in knee flexion. This effect was localized to the ankle joint as proximal muscles were unaffected by bracing and by voluntary changes in soleus excitation.

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1 INTRODUCTION

The triceps surae is a muscle group consisting of the soleus (SOL), lateral gastrocnemius (LG), and medial gastrocnemius (MG) muscles. Given their anatomical structures, it was once believed that these muscles worked in unison but imaging technology has shown that this is not always the case (Yanagisawa et al., 2003, Segal and Song, 2005); instead, researchers suspect that these muscles are differentially controlled leading to a load sharing relationship among the triceps surae complex.

Previous work in cycling has shown that while SOL excitation, as measured by surface EMG, was unaffected by cadence manipulations, MG excitation increased as cadence increased (Marsh and Martin, 1995). A series of experiments by Sanderson and colleagues (Sanderson and Kenyon, 2005, Sanderson et al., 2006) have attempted to determine why the muscles of the triceps surae respond differently to cadence manipulations. In extending the protocol of Marsh and Martin (1995), Sanderson et al. (2006) recorded muscle lengths in addition to muscle excitations while cycling at a range of cadences. They suggested that differential responses to cadence manipulations between the SOL and MG muscles might be associated with mechanical properties of the muscles, primarily muscle length (Sanderson et al., 2006).

Because motion at the ankle and knee joints occurs simultaneously during cycling, it is difficult to partition the excitation of the LG and MG muscles to determine the relative contribution of ankle and of knee motion. One way to measure the relative contribution is to eliminate movement at one of the two joints, for example, the ankle joint. By de-recruiting the SOL muscle, we presume that the involvement of the ankle joint motion is removed and that all remaining LG and MG excitation is directed by knee motion. In a previous study designed to investigate the differing relationships with cadence and to separate the muscles' contributions to actions at the knee and ankle joints, participants wore an ankle brace that positioned the ankle joint at 90°. The cadence sensitivity in MG remained while SOL remained insensitive to cadence below 85 rpm (Sanderson and Kenyon, 2005). Because the brace eliminated plantarflexion and dorsiflexion motion, and thus plantarflexor torque could not contribute to the cycling action, one might have expected that the SOL muscle would become silent; however, substantial muscle excitation remained. Since SOL could not contribute to the production

of pedal forces, it was proposed that participants could be trained to reduce SOL excitation during cycling. Visual biofeedback of overly active muscles has been effective in guiding individuals to reduce muscle excitation, particularly in trapezius muscle relaxation exercises (van Dijk and Hermens, 2006). As a preliminary question, the current study determined if cyclists could use electromyography (EMG) biofeedback of the soleus muscle to voluntarily inhibit its excitation. The primary purpose was to determine how the medial and lateral gastrocnemius muscles responded when soleus excitation was eliminated.

1.1 Statement of the Problem

The current study addressed three questions with the primary research question designed to investigate the differential activation of the triceps surae muscles during cycling:

1. Can biofeedback be used in isolation to induce a reduction in soleus excitation (biofeedback only)?
2. Is biofeedback an effective tool to reduce soleus excitation beyond bracing (bracing and biofeedback)?
3. When soleus excitation was significantly reduced, how did the recruitment of the medial and lateral gastrocnemius muscles change?

1.2 Brief Methods

To address these questions, participants were asked to cycle under two conditions: unbraced and braced. The unbraced condition involved normal cycling with no constraints while the braced condition referred to cycling with an ankle-foot orthoses, a plastic L-shaped brace covering the foot and lower leg. Across two days, participants cycled braced and unbraced in conditions with and without biofeedback. The average soleus EMG from the first half of the pedal cycle (0-180° with 0° indicating the top of the pedal stroke) was displayed during a twenty-minute ride while participants attempted to silence soleus excitation. Electromyography was recorded from seven muscles: soleus, lateral gastrocnemius, medial gastrocnemius, tibialis anterior, biceps femoris, rectus femoris, and gluteus maximus.

1.3 Hypothesis

When wearing an ankle-foot orthosis, soleus recruitment will be reduced using feedback of surface EMG in real-time; however, participants will be unable to reduce SOL EMG in the unbraced ankle condition. EMG biofeedback will allow participants to become consciously aware of their soleus excitation; through visual integration of this feedback, braced participants will be able to voluntarily decrease recruitment of SOL. Cannon et al. (2007) found that cyclists can modify mean ankle joint position while cycling without braces; however, while trying to maintain maximal dorsiflexion, the maximal plantarflexion angle did not change although the time in plantarflexion was slightly reduced. Thus, it is expected that during unbraced cycling with feedback, SOL EMG will not change significantly as plantarflexor torque will not be limited.

Medial and lateral gastrocnemius muscle excitation will decrease during SOL EMG biofeedback in the braced condition. Mornieux et al. (2007) found that the ankle moment contributed 21% to the total moment in cycling. Sanderson and Kenyon (2005) showed that while wearing an ankle brace, the excitation of both SOL and MG decreased. The reduction in LG and MG excitation from the elimination of active plantar flexor torque in the braced condition will exceed any increased excitation in these muscles due to increased knee flexor torque requirement. Thus, LG and MG excitation will decrease along with SOL excitation during bracing and biofeedback.

2 REVIEW OF LITERATURE

This literature review focuses on four essential areas of research: understanding how the triceps surae group functions both generally and in cycling, how one uses biofeedback, and how soleus excitation may be influenced by other leg musculature.

2.1 Triceps Surae Anatomy

2.1.1 Anatomy of the lower limb: origin, insertion, and action (Drake, 2005)

The gastrocnemius and soleus muscles are together referred to as the triceps surae muscle group. The gastrocnemius (GAS) has two parts, the medial head (MG) and the lateral head (LG). Since many authors did not differentiate between the MG and LG, the term GAS is used when no specifics are available. The lateral head originates from the upper posterolateral surface of lateral femoral condyle while the medial head originates from the posterior surface of distal femur just superior to the medial condyle. The soleus (SOL) muscle lies deep to the gastrocnemius. It originates from two areas: fibular (the posterior aspect of the head and neck and upper shaft of the fibula) and tibial (the soleal line and adjacent medial border of the tibia); it has a ligamentous arch between the fibular and tibial attachments and, unlike GAS, is considered a single muscle. The SOL, LG, and MG insert on the Achilles tendon which attaches to the posterior surface of the calcaneus.

SOL, LG, and MG are innervated by the tibial nerve with nerves originating from the S1 and S2 spinal segments for the gastrocnemius and soleus respectively. The biarticular LG and MG cross both the knee and ankle joints while the monoarticular SOL crosses only the ankle joint. At the ankle joint, the muscles work as synergists, muscles that actively provide an additive contribution to a particular function during a contraction (Basmajian and DeLuca, 1985). These muscles act to plantarflex or control dorsiflexion of the foot while GAS also flexes or controls extension of the knee. There are other muscles in the posterior compartment of the lower leg that contribute to plantarflexor torque including the plantaris, tibialis posterior, flexor hallucis longus, and peroneus brevis; however, these other muscles are not considered in the current research as the

triceps surae muscles account for 60-80% of plantarflexor torque (Sale et al., 1982) and the specific aim is to investigate the relationship between the SOL, LG, and MG muscles.

The primary antagonist muscle, the tibialis anterior (TA), originates from the upper two-thirds of the lateral surface of the shaft of the tibia and adjacent surface of the interosseous membrane as well as the deep fascia. The muscle fibres converge in the lower third of the leg to form a tendon which descends into the medial side of the foot and attaches to the medial and inferior surfaces of the medial cuneiform and first metatarsal. TA acts to dorsiflex the foot and is innervated by the deep peroneal nerve, a branch of the common fibular nerve.

2.1.2 Evidence for differential activation of the triceps surae

It was once believed that all muscles forming the triceps surae group acted in unison, and while it has been known for some time that this belief is incorrect, direct evidence for the differential activation of triceps surae came only recently from dynamic magnetic resonance imagery (Yanagisawa et al., 2003, Segal and Song, 2005). When a limb is scanned in a magnetic resonance scanner, atoms with an odd number of protons spin along the axis of the scanner. A brief radiofrequency causes the protons to spin off-axis. The time over which a proton moves away from the bore axis is called the T2 time. By measuring the T2 times during different conditions (such as pre- and post-exercise), one can determine the relative activity within and between muscles; a longer T2 time indicates a more active muscle or segment of muscle (Segal and Song, 2005).

Using a heel-raise task, Yanagisawa et al. (2003) found that the T2 times of all plantarflexors increases. While SOL activity increased after plantarflexion, the percentage increase in activity was much higher in MG. Further, researchers have determined that sub-volumes of the muscles of the triceps surae are differentially active during unilateral heel raises. As displayed in Figure 2.1, the spatial distribution of T2 time changes was not homogenous throughout the SOL, MG, and LG; instead, the proximal components were more active than the distal ones (Segal and Song, 2005). These findings suggested a possible compartmentalization to recruitment and showed between and within muscle differences in the triceps surae during dynamic activities.

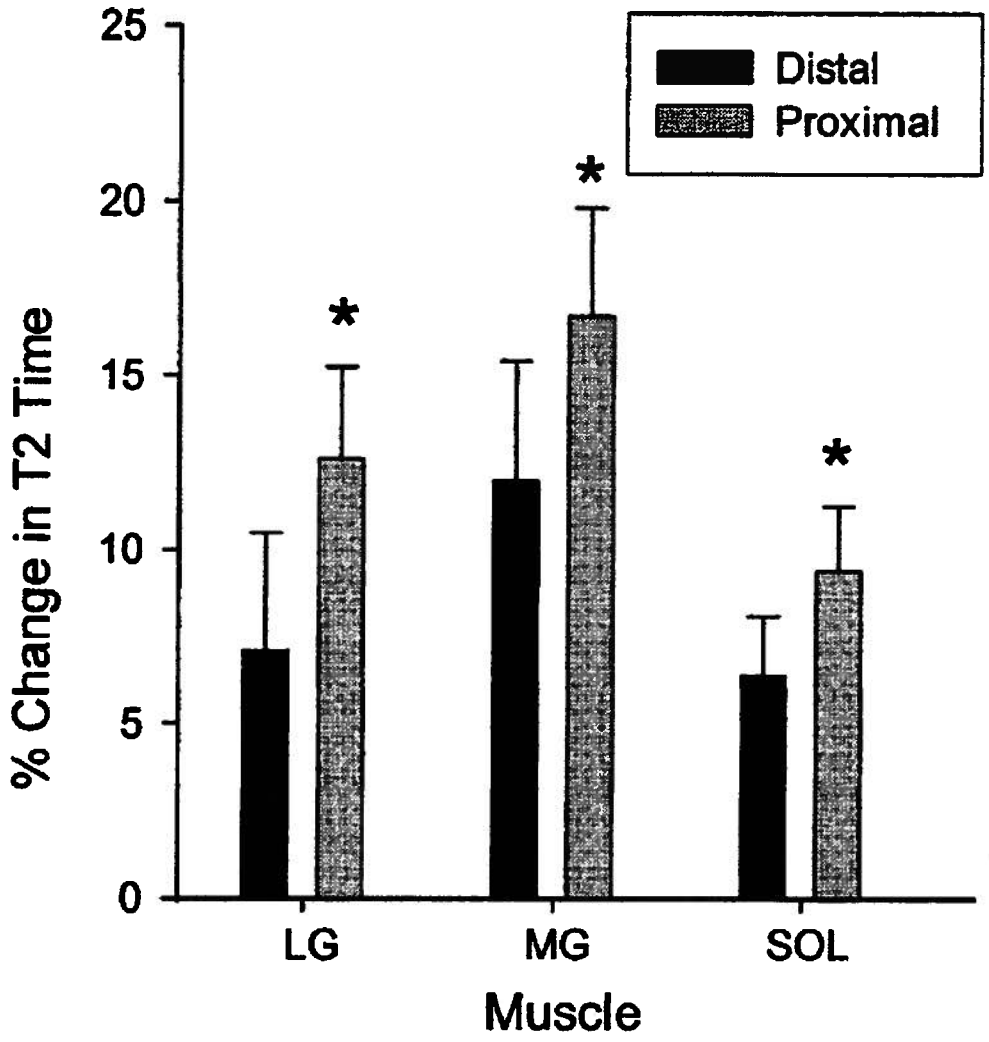

Figure 2.1. Change in T2 times in distal and proximal segments of four lower leg muscles (Adapted from Segal & Song, 2005).

Further differences in activation of the triceps surae have been illuminated through factors such as weight bearing and activation level. Fiebert et al. (2000) collected surface EMG during weight-bearing isometric plantarflexion contractions at 30, 50, 70, and 100% of body mass. They reported that excitation was greater in MG than LG for all conditions but that the excitation difference between muscles decreased as the degree of weight bearing increased (Fiebert et al., 2000). Compared to resting levels, Giordano and Segal (2006) found that neither the LG, MG, nor SOL had increased T2 times from resting to isometric contraction at 25% maximum voluntary contraction (MVC); however, at 65% MVC, T2 times increased in all three muscles, with proximal segments of the MG and LG more active than distal ones.

Clearly, the triceps surae group does not always act as a single muscle; instead, regional differences between and within muscles are apparent with factors such as weight bearing and activation level influencing the extent of excitation of each muscle. Given these differences during isometric conditions, the dynamic load sharing relationship between these muscles is likely to differ between activities, such as walking and cycling, and within an activity, across changes in workload and cadence.

2.2 Triceps Surae in Cycling

2.2.1 Characteristic cycling action

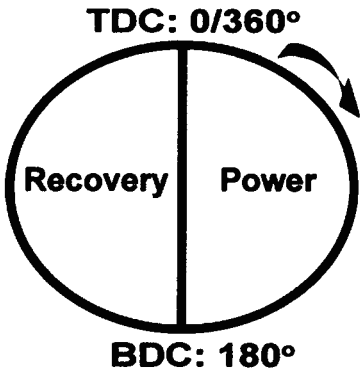

Figure 2.2. Pedal cycle with clockwise pedal movement.

Due to the constrained nature of cycling, the general pattern of ankle and knee joint movement has been shown to be relatively consistent. As shown in Figure 2.2, a pedal cycle has been divided into two phases: the power phase lasting from top dead centre (TDC, 0° crank angle) to bottom dead centre (BDC, 180°) and the recovery phase from BDC back to TDC (180° to 360°, or 0° in the subsequent cycle).

Standard movement patterns have been described by Sanderson et al. (2006). Ankle joint dorsiflexion occurred from TDC to a 45° crank position. Ankle plantarflexion lasted from 45° until just prior to BDC. The maximum plantarflexion angle was 3-7° beyond neutral (90°) and occurred near BDC. The ankle

remained in dorsiflexion throughout the recovery phase. Generally, the knee joint underwent extension throughout the power phase and flexion during the recovery phase. At TDC, the knee joint was at 100° of flexion and extended until reaching a 30° joint angle prior to BDC. Knee flexion occurred until the 330° crank position when knee extension began (Figure 2.3).

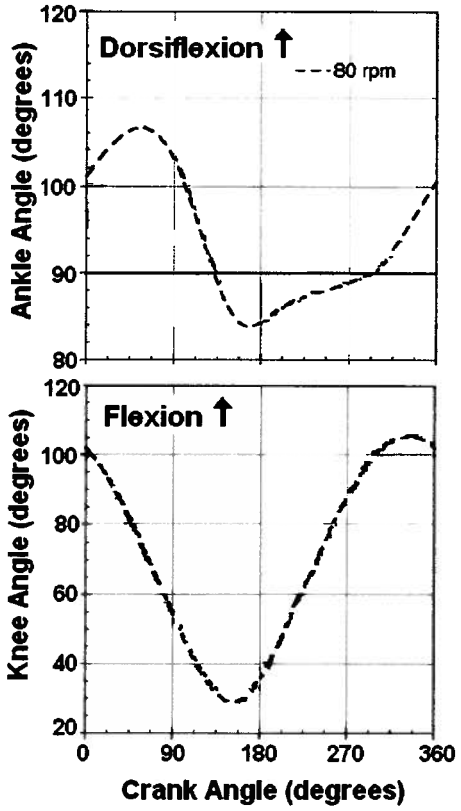

Figure 2.3. Ankle and knee angles throughout a pedal cycle at a cadence of 80rpm (Sanderson et al., 2006).

These general patterns of ankle and knee joint motion are influenced by factors such as cadence. Sanderson et al. (2006) showed that as cadence increases, the ankle joint becomes more plantarflexed, the knee joint becomes more flexed, and the range of motion decreases.

In addition to kinematic analyses, previous researchers have focused on muscle excitation in cycling. A single peak was seen in SOL excitation (at 90° crank angle) while GAS excitation peaked twice and remained active for longer (Amoroso, 1994, Chapman et al., 2006, Sanderson et al., 2006). After peaking at a 90° crank angle, SOL excitation subsided quickly to baseline by 180°. LG had two activation peaks, the larger occurring at 100° crank angle and the smaller shortly after BDC. With increased cadence, the amplitude of the normalized EMG of the first peak increased while the second peak and the SOL peak remained relatively constant (Sanderson et al., 2006). Similarly, researchers found that LG was active from 32±10° to 230±30° while SOL became active at 20-30°, peaked at 90°, and subsided rapidly (Figure 2.4, Chapman et al., 2006). Sanderson et al. (2006) proposed a potential difference in function with SOL involved in generating initial propulsive forces and GAS overlapping that excitation and prolonging excitation later in the cycle to provide continual force at the pedal.

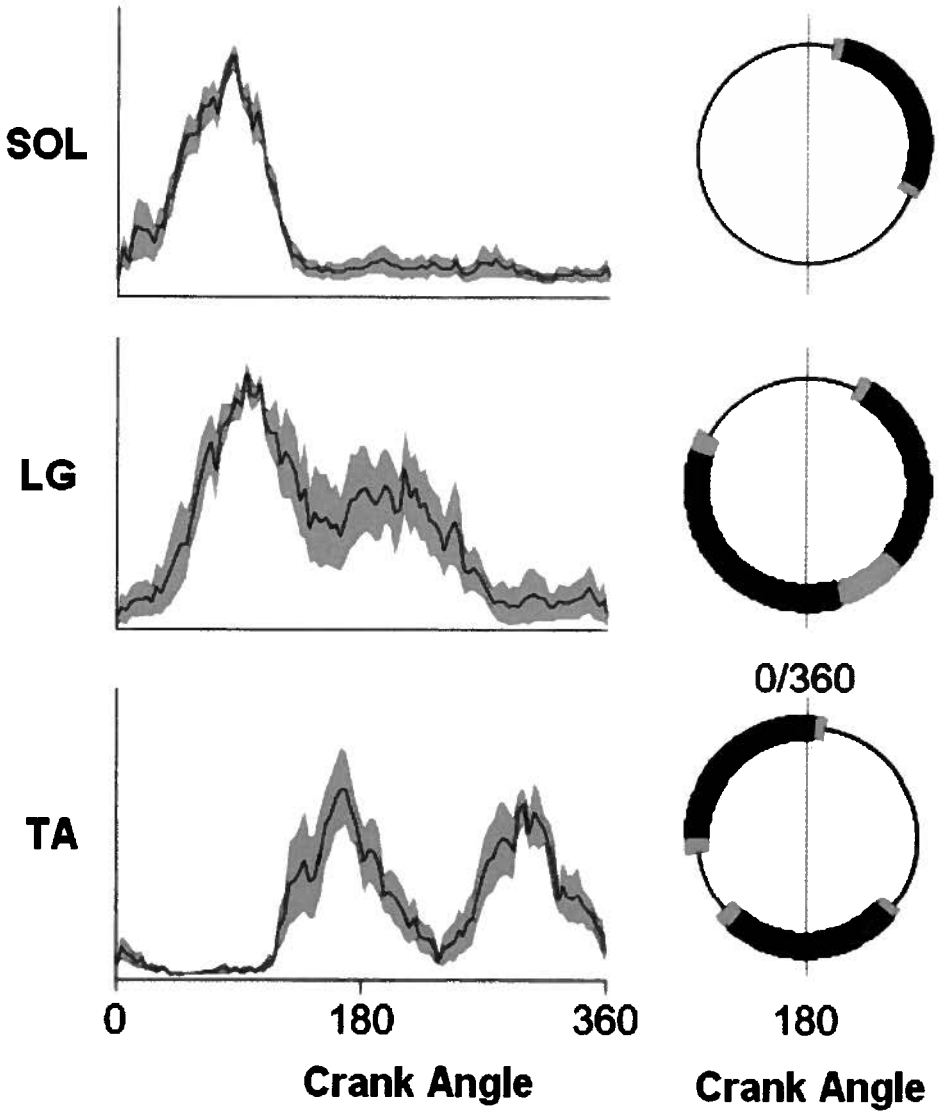

Figure 2.4. Muscle excitation of SOL, LG, and TA for one pedal cycle (TDC to TDC). The grey shading indicates standard deviation across participants (Adapted from Chapman et al., 2006).

2.2.2 Evidence for differential activation of the triceps surae in cycling

Much of the evidence to suggest that the triceps surae muscles function differently during cycling came from studies of cadence manipulations. With few exceptions, researchers found that SOL was insensitive to cadence manipulations while GAS excitation increases as cadence increases (Table 2.1).

Table 2.1. Effect of cadence manipulations during cycling on triceps surae excitation.

Authors	Cadences (rpm)	Power Output (W)	SOL response to cadence increase	GAS response to cadence increase
Ericson et al. (1985)	40, 60, 80, 100	120	No change	Linear increase
Duchateau et al. (1986)	30, 55, 80, 110, 140, 170	10N	No change or slight decrease	Linear increase
Marsh and Martin (1995)	50, 65, 80, 95, 110	200	No change	Increase (MG)
MacIntosh et al. (2000)	50, 60, 80, 100, 120	100, 200, 300, 400	100, 200, 300 W: No change 400W: Increase	100, 200 W: Linear increase 300, 400W: Quadratic increase
Sanderson et al. (2006)	50, 65, 80, 95, 110	200	No change	Increase (MG)

Marsh and Martin (1995) proposed many explanations such as fibre type and muscle length changes to explain these differential responses to cadence manipulations. SOL is predominantly formed by slow-twitch fibres so it may have been less effective at developing force as movement speed increased. It was also proposed that SOL underwent greater muscle length changes from TDC to BDC when SOL was most active; thus, SOL was rendered less effective due to the increased shortening and lengthening velocity changes across cadence manipulations.

Sanderson et al. (2006) replicated the procedure of Marsh and Martin (1995) while collecting both kinematic and EMG data to decipher the relationship of muscle length and muscle excitation across cadence. They provided a potential explanation for the differing cadence sensitivities focused on the force-length-velocity relationship. There were times during the pedal cycle when one of the triceps surae muscles was active while another was silent; for example, directly after BDC, MG excitation peaked while SOL remained relatively quiet. Additionally, Sanderson and colleagues found periods in which SOL was concentrically contracting (shortening) while GAS was eccentrically

contracting (lengthening). They showed evidence of the stretch-shorten cycle in both muscles; the extent of lengthening decreased and extent of shortening increased as cadence increased while the velocity of both shortening and lengthening increased across cadence. The researchers proposed that the muscle excitation was dictated by the muscle length and velocity relationship and that optimal force production occurred within a certain operating length (Sanderson et al., 2006).

Sanderson and Kenyon (2005) suggested that a difference in mechanical properties of SOL and MG led to their different responses to cadence manipulations. To eliminate the effect of the ankle joint, they braced the ankle at 90° of flexion and again replicated the procedure of Marsh and Martin (1995). Movement at the ankle joint was effectively reduced, limited to 3° as dictated by the flex of the rigid plastic brace. As expected, SOL excitation was reduced, on average by 30% from the baseline, unbraced condition. MG excitation decreased by 33%, a surprising finding as excitation was expected to increase to compensate for the reduction in SOL excitation. Since the ankle joint contributed 21% to the total moment during cycling (Mornieux et al., 2007), it was expected that the moments at the knee and hip would increase to compensate; however, it was apparent that the MG did not fill this compensatory role. Surprisingly, SOL was still highly active; by eliminating plantarflexion, its major action, its ability to contribute to pedal forces was removed. The remaining SOL excitation could not generate purposeful movement and may have acted to stabilize the joint and control dorsiflexion.

2.2.3 Manipulation of the ankle joint during cycling

Many investigations have explored cycling efficiency through modifications in pedalling cadence, crank length, and seat height, but only Cannon et al. (2007) focused on manipulation of the position of the talocrural joint (more often referred to as the ankle joint). Joint position was measured by an electrogoniometer attached to the lateral border of the ankle and anchored along the axis of the tibia and metatarsals. Using a one-week training protocol, participants were instructed to pedal with maximal dorsiflexion or maximal plantarflexion throughout the entire pedal cycle at 90 rpm and a power output that elicited 80% of maximal oxygen uptake. Compared to the mean joint angle held in control trials, cyclists maintained a mean joint angle of 7.1° of dorsiflexion and 6.9° of plantarflexion (Figure 2.5).

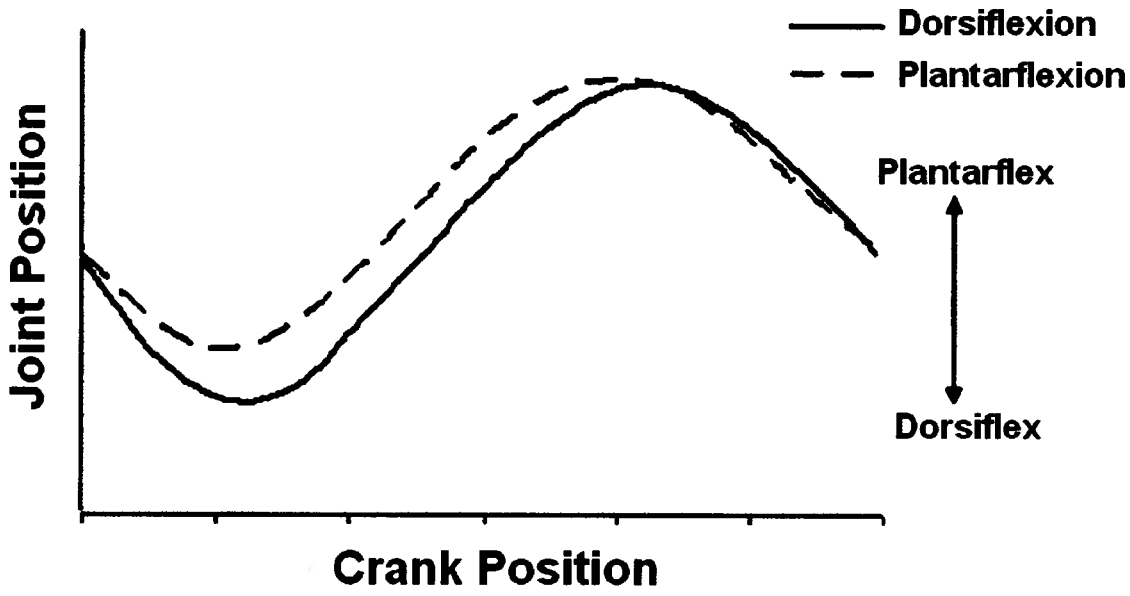

Figure 2.5. Talocrural position during cycling while attempting to hold the joint in dorsiflexion or plantarflexion (Cannon et al., 2007).

Participants were unable to eliminate dorsiflexion or plantarflexion. However, in the plantarflexion condition, the maximal dorsiflexion angle and total time in dorsiflexion were reduced; in the dorsiflexion condition, the overall time in plantarflexion was reduced while the maximal plantarflexion angle remained constant. With respect to EMG, TA showed no statistically significant changes but compared to the control condition, mean TA excitation tended to decrease in plantarflexion and increase in dorsiflexion. GAS EMG increased significantly in the dorsiflexed condition compared to the control condition but no difference was found in the plantarflexed condition. During the dorsiflexion trials, the increased GAS excitation could have been due to increased knee flexion. Additionally, it was not surprising that there was no reduction in GAS EMG since there was no reduction in maximal plantarflexion angle.

Due to an inaccurate definition of TDC, the authors' interpretation was based on a pedal cycle shifted by an unknown number of degrees and led to erroneous conclusions; however, this study provided an example of successful manipulation of cycling technique from control of the ankle joint and exposed potential muscular changes with voluntary control of ankle position. Through instructions to adopt a certain ankle position and verbal feedback from the researcher monitoring joint angle, adaptation to normal cycling technique was shown possible (Cannon et al., 2007).

2.3 Tools to Reduce Soleus Excitation

2.3.1 Bracing and biofeedback

The ankle bracing technique provided evidence for the ability to reduce SOL EMG through external means (Sanderson and Kenyon, 2005). However, since ankle bracing alone did not remove SOL excitation entirely, other tools were pursued. Cannon et al. (2007) showed that an individual could voluntarily manipulate the position of the ankle joint during cycling but had no recording from the SOL muscle. It remained uncertain whether one could voluntarily modify the known skill of cycling to reduce SOL excitation.

During performance of a motor skill, individuals are known to receive task-intrinsic feedback, the sensory-perceptual information that is a natural part of performing the skill. This might include visual, proprioceptive, and auditory feedback. While learning to

modify known skills, attending to and understanding this feedback can be difficult so enhancing sensory feedback can facilitate goal achievement. Augmented feedback involves adding to or enhancing task-intrinsic feedback (Magill, 2001) and can aid skill acquisition. While one receives intrinsic feedback about muscle excitation, determining one's exact level of muscle excitation is difficult without an augmented feedback display. Previously, Sanderson (1986) used visual feedback of pedal force application to modify cycling technique. Using a one-week training protocol, he showed that feedback can effectively aid in alteration of pedalling technique.

Biofeedback has been defined as "the use of instrumentation to make covert physiological processes more overt" (Huang et al., 2006). It allows individuals to gain voluntary control over a psychophysiological process that is considered beyond conscious awareness (Blumenstein et al., 2002). The basis of EMG biofeedback is electromyography, a tool that measures electrical activity preceding muscle contraction. Visual EMG biofeedback displays present the activity of one or more muscles allowing individuals to understand their activation levels or patterns and make adjustments as required.

2.3.2 Theories of biofeedback function

Individuals who suffer from sensorimotor deficits have been shown to adapt their muscle excitation as they become cognizant of the EMG signal through biofeedback. Basmajian (1982) posited two potential neurological mechanisms to explain the enhanced ability to voluntarily control muscle activation in diseased or injured individuals. He stated that either new pathways were developed or auxiliary feedback loops recruited existent but dormant cerebral and spinal pathways. Fitting with the second rationale, Wolf (1983) suggested that in executing motor commands, both visual and auditory feedback activate unused or underused synapses.

Having tested healthy individuals, Palmerud et al. (1995) presented two rationales for trapezius muscle silence during a biofeedback relaxation task. First, there might have been a load shift to other muscles compensating for the reduced excitation of the trapezius. Second, there might have been an initial over-activity in the trapezius and its antagonists leading to overstabilization of the shoulder joint. They used single wire intramuscular EMG on five shoulder muscles and determined that there was no

concomitant increase in synergist muscles when trapezius muscle excitation decreased, suggesting that the latter hypothesis was more valid (Palmerud et al., 1995). It is not certain why individuals activated muscles that were not required to effectively perform the task. When attempting to decrease muscle activity, healthy individuals viewing EMG biofeedback were able to decrease recruitment of active muscles by reducing seemingly unnecessary muscle excitation.

2.3.3 Previous use of EMG biofeedback

EMG biofeedback has proven successful in altering muscle patterns. In clinical gait research, EMG biofeedback was used to correct abnormal movement patterns by overlaying target levels of muscle excitation on patients' muscle excitation traces (Colborne et al., 1994, Petrofsky, 2001, Aiello et al., 2005). These three gait studies provided examples of the successful implementation of EMG biofeedback to alter excitation of select muscles during dynamic tasks. While these tasks have frequently involved an attempt to increase muscle excitation, the aim to voluntarily reduce muscle excitation is not a novel task. A much-investigated area of EMG biofeedback targeted relaxation of postural muscles and provided a useful model. Both qualitative and quantitative studies have shown that providing biofeedback of the trapezius muscle induced a reduction in muscle excitation during static and dynamic tasks in both injured and healthy participants (Nord et al., 2001, van Dijk et al., 2005). These studies have provided evidence for the successful use of EMG biofeedback to reduce excitation in select muscles.

2.3.4 Selection of biofeedback displays

Researchers and clinicians must determine the best way to deliver biofeedback incorporating considerations such as timing, frequency, and presentation type. EMG biofeedback has been presented as concurrent or terminal feedback. Concurrent feedback occurs in real-time while the muscle is active whereas terminal feedback occurs after task completion (van Dijk and Hermens, 2006). In a simple bottle movement task, individuals aiming to reduce trapezius EMG saw performance improvements (compared to a no feedback trial) regardless of timing condition, perhaps due to the simplicity of the task and the focus on a single muscle (van Dijk and Hermens, 2006). In quick, discrete tasks,

the short latency of terminal feedback reduced the difference in performance between concurrent and terminal feedback; however, as the length and complexity of the task increased, terminal feedback might occur too late to allow individuals to effectively integrate feedback (Schmidt et al., 1990) and alter muscle excitation.

Feedback presented too frequently might also be detrimental to optimal performance. With auditory feedback provided at one of three time periods (5, 10, or 20 seconds), the frequency of information which led to the greatest reduction in trapezius excitation was the ten-second interval (Voerman et al., 2004). Both the timing and frequency of the feedback are important considerations in presenting the appropriate amount of feedback.

Previous researchers have implemented many types of EMG biofeedback displays, primarily visual or auditory. In visual EMG biofeedback, the display might have consisted of a simple on/off bar, a single value such as average EMG calculated over a select time interval, or an online EMG trace (Blumenstein et al., 2002). Others have used an auditory tone presented if muscle excitation was above threshold or multiple tones presented with different tones relating to different excitation levels (Lam and Dietz, 2004). The feedback display must be designed balancing the need for completeness with that of simplicity. Many muscle relaxation studies have employed a visual EMG trace (Palmerud et al., 1995, van Dijk and Hermens, 2006) but the best form of presentation is highly dependent on the task. In the case of a cyclical activity in which the muscle of interest has a single active period, an EMG trace allowing for participants to understand the timing of muscle activity bursts might become unnecessary (Madeleine et al., 2006). A waveform could be replaced by an on/off feedback display or bar chart which requires minimal integration and processing.

2.3.5 Multiple types of biofeedback

When two types of biofeedback are presented simultaneously, individuals must decide which feedback display to attend. The use of two types of feedback has proven successful. Many studies have been conducted that required participants to ramp up plantarflexion or dorsiflexion torque to a set level and then, while maintaining EMG activity, co-contract to bring the torque level back to zero. This required the simultaneous presentation of EMG and torque biofeedback, and researchers noted that,

even though they were required to constantly monitor both levels, participants were able to complete the task without difficulty within minutes of training (Nielsen et al., 1994). In a cycling study, Chu (2006) used augmented feedback to train cadence. Participants trained 20 minutes a day for 10 days at a predetermined optimal cadence. Cadence feedback was withheld from the control group while the experimental group was given feedback at set intervals. The amount of feedback decreased across days, and testing occurred without feedback. Both the cadence consistency and variability were effectively reduced in the feedback group. During cycling with feedback, participants were able to maintain the desired cadence throughout the display period (Chu, 2006). During presentation of both EMG and cadence feedback, participants can be instructed to attend sporadically to cadence feedback while focusing on the EMG biofeedback, without too much concern for maintenance of appropriate cadence.

2.4 Interaction Among Motor Neuron Pools

Motor neuron pools are a collection of motor neurons which innervate a single skeletal muscle. They are located in the spinal cord; generally, motor neuron pools for distal muscles are located more laterally in the ventral horns. They have been shown to be influenced primarily by the descending fibres of the lateral corticospinal and reticulospinal pathways and may be influenced by the vestibulospinal pathways as well (Rossignol et al., 2006). A motor neuron pool from one muscle can influence the excitability of other muscles such that input to a motor neuron, and eventually to a muscle fibre, does not come from a single source. Instead, antagonists, synergists, and even muscles from the contralateral limb affect the firing of a motor neuron. Voluntary reductions in SOL muscle excitation may be affected by the ipsilateral TA and GAS and the contralateral SOL and thus their connections must be examined (Figure 2.6).

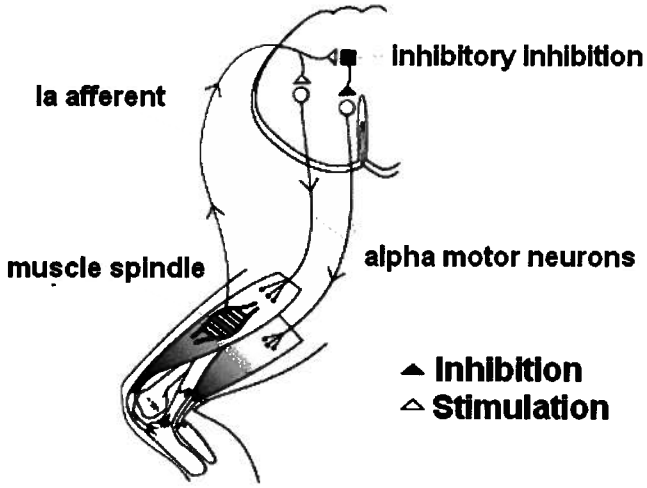

Figure 2.6. Descending control of reciprocal inhibition. Activation of the flexor muscle spindles excites the flexor muscle and inhibits the extensor muscle (Dale, 2004).

2.4.1 Measuring excitability

Because there is no direct measure of the effect of one motor neuron pool on another, motor neuron pool excitability before and after stimulation of group Ia afferents has been used as an indirect measure. Researchers employed the Hoffman reflex (H-reflex) technique, an artificially elicited response that tests the efficiency of transmission of a stimulus passing from the afferent fibres to the efferent fibres, through the motor neuron pool (Enoka, 2002). A change in the excitability of the motor neurons influences the H-reflex; an inhibitory post-synaptic potential (IPSP) elicited by stimulation of an antagonist Ia afferent transiently hyperpolarized the motor neuron resulting in a decrease in the size of the H-reflex (Nielsen, 2004). Stimulation of the Ia afferents from the TA (via the common peroneal nerve) depressed the SOL H-reflex. A depression in the H-reflex indicated decreased motor drive and led to a decreased muscle excitation. Influences from synergists and muscles on the opposite limb can be tested in the same way simply by stimulating a different nerve. H-reflexes within lower leg muscles can be evoked by stimulation of the tibial nerve for SOL reflex, common peroneal nerve for TA, and medial gastrocnemius motor nerve for GAS (Nielsen and Kagamihara, 1993). A high level of SOL H-reflex inhibition from any of these sources would result in low excitability of the SOL muscle.

The H-reflex, while a useful tool, is not an unambiguous measure of motor neuron excitability (Zehr, 2002, Misiaszek, 2003). Through altered presynaptic inhibition, the amplitude of the H-reflex changed without a corresponding change in the postsynaptic membrane, including the motor neuron (Zehr, 2002). Unless the pre- and post-test H-reflexes were collected with the same postural orientation, intention, level of muscle excitation, and while stationary, presynaptic inhibition will likely influence the H-reflex. This presynaptic inhibition can arise from descending supraspinal commands as well as from afferent feedback from peripheral receptors such as Golgi tendon organs, muscle spindles, and cutaneous mechanoreceptors. These factors can only be ruled out by controlling the posture and intention of the participant. The amplitude of the H-reflex was also sensitive to mechanisms that directly affect neurotransmitter release from the Ia afferent terminals (Zehr, 2002, Misiaszek, 2003). The H-reflex technique is confounded by influences other than group Ia afferents and must be interpreted with caution.

2.4.2 Sources of inhibition on the motor neuron pool

Group Ia afferents leave a muscle and branch to the motor neuron associated with the agonist muscle and to an interneuron associated with the antagonist muscle. The synapse with an inhibitory interneuron allows for IPSPs to the motor neuron of the antagonist. It lowers the excitability of the antagonist motor neuron and is known as the reciprocal-inhibition reflex (Enoka, 2002). Activation of muscle spindles in TA elicits an excitation of the agonist motor neurons but inhibits those of the antagonistic SOL. Since the net muscle excitation about a joint results from the difference in excitation of the agonist-antagonist pair, reciprocal inhibition increases the likelihood that a stimulus to activate a muscle will elicit a meaningful response in that muscle. Muscles of the lower leg are also influenced by reciprocal connections with muscles above the knee (Misiaszek, 2003, Wilmink and Nichols, 2003). The quadriceps (rectus femoris and vasti muscles) has a purely inhibitory influence on GAS while the influence on SOL is either excitatory or inhibitory.

Another source of inhibition that has been demonstrated is recurrent inhibition resulting from the neural connection of synergists. It occurred through two interneurons, rather than a single one in reciprocal inhibition. Windhorst (2007) explained that group Ia afferents from a synergist muscle synapse on Renshaw cells which synapse on Ia inhibitory interneurons. The main source of the long-lasting inhibition on SOL motor neurons came from activity from the Renshaw cells of the MG (Rossi et al., 1994). Thus, isolated activation of the MG had an inhibitory effect on SOL muscle excitation.

Lastly, crossed inhibition involved inhibition from the muscles on the opposite leg. Crossed inhibition has been found to be mediated through the same neural pathway as reciprocal and recurrent inhibition (Cheng et al., 1998). Group Ia muscle afferents have been reported to contralaterally modulate reflex responses and motor neuronal activities in humans (Cheng et al., 1998). In a cyclical action, activation of the right SOL muscle inhibits the left SOL. Potential sources of inhibition on SOL excitation included reciprocal inhibition from the ipsilateral TA, recurrent inhibition from the ipsilateral GAS, and crossed inhibition from the contralateral SOL.

2.4.3 Influence of single muscle excitation on soleus excitability

During voluntary movement, reciprocal inhibition on the activated motor neuron was reduced with respect to passive movement thus increasing the H-reflex magnitude of the active muscle (Crone et al., 1985, Iles, 1986). In simple plantarflexion and dorsiflexion movements, voluntary activation of TA led to increased reciprocal inhibition on the SOL muscle compared to rest; further, inactivation of SOL led to decreased reciprocal inhibition on TA (Tanaka, 1974, Pyndt et al., 2003, Nielsen, 2004). With voluntary activation, there was a general increase in the excitability of the TA motor neuron pool resulting in sub-threshold depolarization of a number of motor neurons within the pool. Thus, with the same afferent dischange, more motor neurons were able to generate action potentials (Latash, 1998). Increasing activation of TA led to an increased excitability of TA and a decreased SOL excitability.

Isometric leg flexion by tonic activation of GAS without activation of SOL induced an inhibition of SOL H-reflex even at very low levels of GAS EMG (Gritti and Schieppati, 1989). Tonic voluntary contraction of the SOL at less than 20% MVC abolished the inhibitory effect on the H-reflex of both isolated stimulation in TA and GAS as well as in combined stimulations (Schieppati et al., 1990). Thus, excitation of SOL increased the SOL H-reflex while excitation of TA and GAS led to a decrease in SOL H-reflex.

2.4.4 Excitation of multiple muscles

Co-contraction of agonist/antagonist pairs

In a more complex situation with excitation of both TA and SOL (co-contraction), the SOL H-reflex was decreased with respect to isolated plantarflexion thus co-contraction decreased excitability (Nielsen et al., 1994). During activation of both muscles, the reciprocal inhibition was greater than with isolated activation of the agonist but smaller than isolated activation of the antagonist.

Interaction between reciprocal and recurrent inhibition

Since concurrent activation of TA and GAS occurs during cycling, it is important to determine how reciprocal and recurrent inhibitions interact. Throughout the pedal cycle, the main period of co-contraction occurred from approximately 160-190 degrees

on crank rotation (Chapman et al., 2006). Schieppati et al. (1990) investigated the interaction inhibition from TA and GAS on SOL H-reflex through isolated and combined stimulation of the nerves and found that combined stimulation significantly reduced the H-reflex in SOL beyond the inhibition in either isolated case. This suggested convergence of Ia fibres from synergistic and antagonistic muscles onto common inhibitory interneurons. Reduction of the SOL H-reflex appeared to have a ceiling level; with stimulus intensity at 1.3 times motor threshold in both the TA and GAS, the combined stimulus did not further inhibit the H-reflex suggesting a saturation effect (Schieppati et al., 1990). The concurrent excitation of TA and GAS (up to a certain threshold level) inhibited SOL excitability to a greater extent than isolated activation of either muscle.

Influence of TA and SOL on GAS excitability

Nielsen and Kagamihara (1993) tested the SOL, TA, and GAS H-reflexes during plantarflexion, dorsiflexion, and co-contraction. Maintaining a constant level of EMG activity in the agonist muscle, the SOL and TA H-reflexes were found to be smaller during co-contraction than during an isolated agonist contraction. Contrarily, the medial GAS H-reflex was the same size during co-contraction as during isolated plantarflexion. Thus, it appears that TA excitation differentially affects SOL and GAS excitability. While TA clearly provides reciprocal inhibition to the SOL muscle, reciprocal inhibition to GAS was small if present. This was confirmed by PSTH which showed a depression of the peak excitation in SOL motor units but not in medial GAS motor units during activation of TA (Nielsen and Kagamihara, 1993).

There appears to be directionality of recurrent inhibition between SOL and GAS. Synergism, observed through inhibitory reflexes among SOL, medial GAS, and lateral GAS, was apparent only at low to moderate forces in cats. An electrically evoked cross-extension reflex showed a unidirectional inhibitory reflex from both LG and MG to SOL and showed an increased inhibition with increased force in GAS (Nichols, 1989). By inhibiting SOL H-reflex, LG and MG might reduce redundant excitation in the plantarflexors. It appeared that recurrent inhibition from GAS to SOL was not bi-directional and, like TA, SOL excitation did not induce inhibition of the GAS H-reflex.

2.4.5 Influence of the non-test limb

Passive cyclic movement of one leg induced SOL H-reflex inhibition in the static test limb (McIlroy et al., 1992, Collins et al., 1993). This inhibition was attributed to reciprocal inhibition from the contralateral TA (crossed reciprocal inhibition) and was weaker than ipsilateral reciprocal inhibition in the moving limb (Cheng et al., 1998). It was found that when both legs are moving, inhibition of the SOL H-reflex was not increased over single, ipsilateral leg movement. This redundant system had no summation of ipsilateral and contralateral inhibition but rather a degree of overlapping inhibition (McIlroy et al., 1992). If both TA muscles worked simultaneously, the crossed reciprocal inhibition would be entirely redundant; however, in cycling, the legs are 180° out of phase so the contralateral TA is most active when the ipsilateral SOL is active. Since tonic activity in the agonist muscle decreased reciprocal inhibition on itself (Crone et al., 1985, Iles, 1986), the heightened H-reflex from the active SOL would likely erase the depression in H-reflex caused by the crossed reciprocal inhibition. Thus, it appeared that cycling was substantially directed by a single limb suggesting that the vast majority of contralateral controls were redundant and could be ignored when determining SOL excitability (Boylls et al., 1984).

2.4.6 Soleus excitability in cycling

Brooke et al. (1992) suggested that there are three movement features with a clear influence in determining H-reflex magnitude: limb joint angles (percentage in the cycle), SOL contraction (with voluntary contraction decreasing inhibition of SOL), and TA contraction (with voluntary contraction facilitating inhibition of SOL). At each point in the pedal cycle, the level of TA and SOL activation interacted resulting in isolated contraction of one of the two muscles or co-contraction. The highest reciprocal inhibition on SOL occurred during recovery (specifically 225 to 270°) when TA was most active; within the power phase, reciprocal inhibition was higher in the second half (90° - 180°) (Pyndt et al., 2003). In another study, SOL H-reflex was found to differ between four set crank positions (55, 140, 250, 330 degrees) with H-reflexes most inhibited at a 330° crank angle (Brooke et al., 1992, McIlroy et al., 1992, Collins et al., 1993). Brooke et al. (1992) and McIlroy et al. (1992) found that SOL H-reflex magnitudes were largest during

the power producing phase and were reduced to near zero during the recovery phase. These findings were congruent with the idea that the highest degree of reciprocal inhibition on SOL occurred when TA, the antagonist muscle, was active (Pyndt et al., 2003, Nielsen, 2004). The three factors proposed by Brooke et al. (1992) interacted to control SOL activation and neural drive with the amount of inhibition changing throughout the pedal cycle.

2.4.7 Other influential factors

Workload

Pyndt et al. (2003) considered multiple workloads finding that as a percentage of background EMG, as load increased, the reciprocal inhibition of SOL by TA decreased. As the SOL was required to increase activation to meet the demands of an increased workload, there was a gradual decline in reciprocal inhibition from TA. This was confirmed by Nielsen and Kagamihara (1993) in a plantarflexion, dorsiflexion, and co-contraction task in which the level of contraction was varied. With increasing levels of plantarflexion, SOL H-reflex size increased while TA H-reflex size decreased; the opposite occurred with increasing dorsiflexion levels (Nielsen and Kagamihara, 1993). This confounding factor is easily eliminated by maintaining a constant workload throughout the testing protocol.

Cadence manipulations

While many researchers have investigated the effects of cadence changes on SOL H-reflexes, few have maintained a constant power output making it difficult to separate out the effects of cadence from those of increased workload. McIlroy et al. (1992) reported a negative linear relationship between cadence and SOL H-reflex magnitude during passive cycling but saw the magnitude plateau between 30-60rpm indicating that all cadences beyond that point resulted in the same SOL H-reflex magnitude. Other passive movement studies indicate that as the velocity of passive cyclic rotation of the leg increased, the H-reflex in the stationary contralateral limb became progressively inhibited (Cheng et al., 1998). The opposite relationship was found as Pyndt et al. (2003) studied the effect of cadence on reciprocal inhibition during an active cycling period. As cadence increased, reciprocal inhibition as a percentage of background EMG decreased. While the relationship differed from passive cycling, active cycling studies showed that the SOL

H-reflex magnitude was influenced by cadence, with inhibition decreasing as cadence increased.

Ankle bracing

McIlroy et al. (1992) considered the possibility that the use of an ankle foot-orthosis to position the ankle joint at 90° might lead to a differential discharge of cutaneous receptors which could influence H-reflex modulation. The authors contended that any contribution specific to the AFO would be limited based on the similarities in H-reflex between active pedalling with and without the brace.

2.5 Summary

The preceding literature review highlighted previous research which showed that in certain conditions, parts of the triceps surae group responded differently to perturbations both in cycling and other activities (Segal and Song, 2005, Sanderson et al., 2006). Using a braced cycling paradigm, attempts to identify the relative contribution of the LG and MG to each of ankle plantarflexion and knee flexion were unsuccessful (Sanderson and Kenyon, 2005). The current study aimed to employ bracing and EMG biofeedback to successfully eliminate plantarflexion from the cycling motion and isolate knee flexion as the only action of the gastrocnemius muscle. The potential neurophysiology behind the ability to accomplish this task was described, with reductions in soleus excitation related to inhibitory influences from a number of leg muscles primarily tibialis anterior and gastrocnemius.

3 METHODS

3.1 Participants

A study group of 13 individuals, recruited primarily from the university graduate student population, gave their informed consent to participate. All participants were 19 years of age or older. Potential participants were excluded if they had any lower-limb injuries or neurological conditions or if they felt that they may be unable to maintain the required workload. Cyclists were asked to refrain from exercising on test day to eliminate the effects of fatigue.

3.2 Instrumentation

Preformed ankle-foot orthoses (AFOs) were fitted to the participant. The AFOs were L-shaped braces made from plastic polypropylene covering sections of the front and back of the lower leg and underneath the foot. Padding was added in areas where the brace did not contact the foot comfortably. The AFOs were slipped into appropriately sized cycling shoes which were usually one size larger than the participants' normal shoes.

Sagittal-view kinematics of the left lower limb were collected at 60Hz using a Panasonic video camera (WDV 5100, Okayama-City Okayama, Japan). Reflective markers were placed on the left side over the greater trochanter, lateral midline of the knee, lateral malleolus, and on the cycling shoes at the base of the calcaneus and the head of the fifth metatarsal. Additionally, a reflective marker was positioned over the lateral malleolus on the left AFO for braced trials. Two points in the pedal cycle were recorded using a 1024-step optical encoder and foil switches to record top dead centre (TDC) and a magnetic reed switch at bottom dead centre (BDC).

Surface EMG data were collected at 600Hz from seven muscles. The skin over the muscles was shaved, abraded, and cleaned to prepare for attachment of pre-amplified surface electrodes (Therapeutics Unlimited, Model 544, Iowa City, IA, USA, gain =35). Muscle excitation was recorded from the left soleus (SOL), medial gastrocnemius (MG), lateral gastrocnemius (LG), tibialis anterior (TA), biceps femoris (BF), rectus femoris

(RF), and gluteus maximus (GM). Electrodes were placed in accordance with Basmanjian and De Luca (1985) and were positioned to avoid contact with the brace.

With electrodes and markers in place, bicycle seat height was adjusted to 100% trochanteric length (measured from the greater trochanter to the floor while standing barefoot). Participants rode on a medium size standard frame mounted on a Schwinn Velodyne electronically braked cycle ergometer which controlled power output. A Cateye cadence monitor attached to the handlebars helped participants maintain the target cadence.

A heart-rate strap was worn by participants to monitor how heart rate changed during the cycling period. Heart rate, an indirect measure of fatigue, was collected to ensure that fatigue did not influence muscle excitation during prolonged cycling with biofeedback. Due to cardiac drift, heart rate increases over a prolonged cycling bout. Lepers et al. (2000) reported a 7.3% and a 12.7% increase in heart rate over the first 50 and 100 minutes of a 120 minute cycling bout at a power output corresponding to 65% of maximal aerobic power. If we assume that our protocol occurred at 65% of maximal power and that the cardiac drift occurred evenly throughout the entire hour, we would expect a 2.9% increase in heart rate over our twenty-minute cycling period. It was expected that heart rate would increase with time, but any large increase in this measure could indicate neuromuscular fatigue.

EMG biofeedback from the left SOL was provided during designated feedback trials. As shown in Figure 3.1, participants watched a computer screen with an actively updating real-time bar graph representing average rectified SOL EMG over the power phase, from TDC to BDC (screen developed in LabView Version 5.0, National Instruments, Austin, TX). The participants were given the following instructions:

You will continue cycling at 150W and 80 rpm. After you have reached the desired cadence, I will turn on the biofeedback and start the prolonged trial. Each time that the left pedal reaches the top of the cycle, the bar graph will update. This bar indicates your average soleus muscle activity from the first half of the pedal stroke. Your aim is to silence the muscle – a completely unfilled bar indicates no muscle excitation. At various times throughout the cycling period, I will collect data. You will not be informed when I am collecting so please try to maintain minimal muscle activity at all times during the testing period. Try to attend to the biofeedback constantly but be sure to check your cadence regularly and keep it steady at 80 rpm. Try to find a comfortable position for your arms on the handlebars and please do not change positions during the trials.

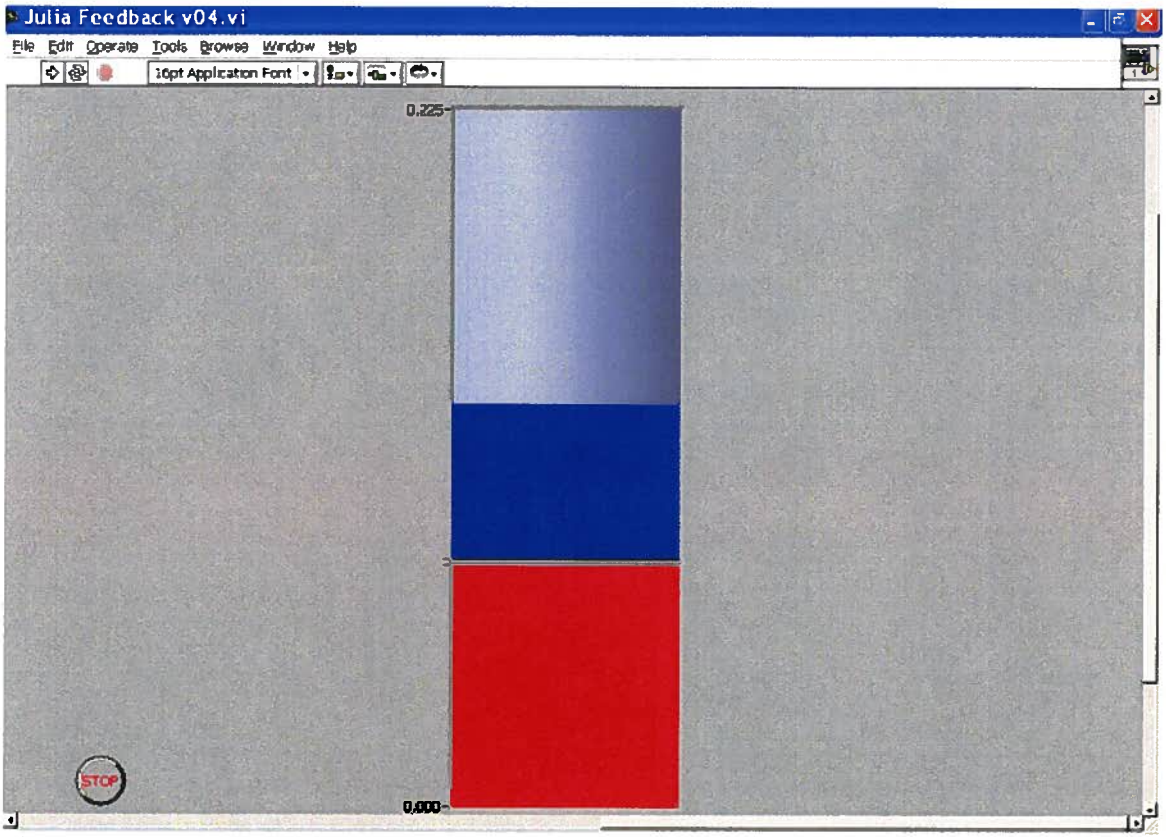

Figure 3.1. Biofeedback display screen.

The maximum height of the biofeedback bar was set to the average rectified SOL EMG across 20 pedal cycles during the baseline, no feedback condition. This collection occurred during the second half of the four minute trial and normalized the display output between participants.

To ensure that participants always had high-resolution feedback allowing them to visualize minor changes in SOL EMG, the bar was coloured in two parts. As displayed in Figure 3.2, when the EMG was greater than 100%, the bar was fully coloured with the top part in blue and the bottom in red. The line separating the two segments of the bar was set to 20% of the full height of the bar, or 20% of the baseline SOL EMG. As the EMG fell, the height of the coloured bar decreased. When EMG was greater than 20%, the red portion remained saturated. The height of the bar was recorded online to generate a profile of how the feedback display changed over time.

On a second computer, data from four lower leg muscles (SOL, LG, MG, and TA) were captured at a sampling frequency of 1000Hz across the entire feedback period. In trials without biofeedback, participants were asked to direct their attention forward towards to computer screen.

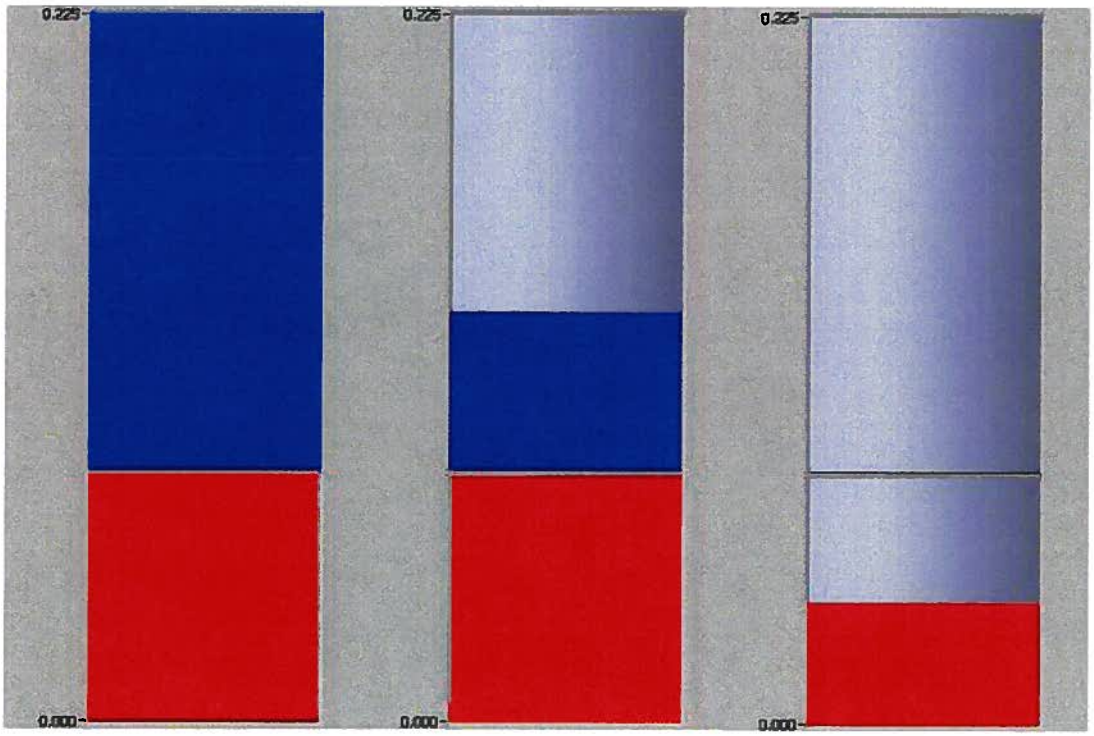

Figure 3.2. Examples of SOL EMG biofeedback. The bars (from left to right) display 100+% excitation, 50% excitation, and 10% excitation.

3.3 Procedures

Data were collected at the University of British Columbia's Biomechanics Laboratory. The protocol was performed over two days. On day one, participants were randomly assigned to a starting condition (braced or unbraced). Of the nine complete data sets, five participants completed the unbraced condition on day one.

The testing procedure is illustrated in Figure 3.3. In both conditions, participants warmed up for 10 minutes at a comfortable, self-selected power output and cadence. Next, they completed a four minute no brace-no feedback (NB-NF) trial to allow for normalization across days, with data collection occurring during the final 10s of cycling. For all trials, participants were required to maintain a fixed power output (150W) at a fixed cadence (80 rpm). In the braced condition, participants dismounted from the cycle ergometer and applied the braces bilaterally. After mounting the bike, they completed a four minute braced-no feedback (B-NF) trial. To ensure that cycling and rest time were kept constant between conditions, during the unbraced condition, cyclists rested for the same length of time as it took to dismount, slip on the braces, and climb back onto the bicycle; then, participants completed a second four minute no brace-no feedback (NB-NF2) trial. Next, in both conditions, participants were presented with EMG biofeedback for 20 minutes. Data were collected for 10s at the end of every minute. One week later, participants returned to the lab to complete the other condition. During each session, total riding time was 38 minutes while total testing time was about two hours.

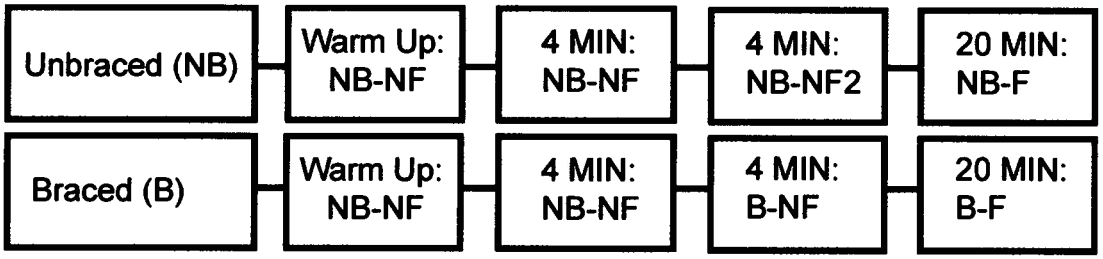

Figure 3.3. Testing Procedure (B = braced, NB = unbraced, F = feedback, NF = no feedback). During four minute trials, data were recorded during the last 10s; during the 20 minute trials, data were recorded for 10s every minute.

3.4 Data Analysis

For each trial, EMG data from ten pedal cycles were analysed. EMG data were rectified and integrated over each pedal cycle and a mean was calculated. For each trial, the mean integrated EMG (iEMG) was normalized to the mean iEMG from the ten pedal cycles in the first NB-NF condition of each testing session. For repeatability calculations, root mean square (RMS) EMG with a 50 ms window for each of the seven muscles was calculated for the NB-NF and NB-NF2 conditions. A power spectrum of each of the seven muscles for each of the data collection periods was computed and the median power frequency (MPF) was determined. Additionally, peak EMG was calculated as the highest 50ms window of rectified, filtered, normalized EMG for each trial. Then, the time-to-peak EMG was calculated for each pedal cycle and averaged to create trial means; the time-to-peak data were analyzed as a relative measure, in percent of pedal cycle.

3.5 Statistical Analyses

A power analysis estimating necessary sample size was calculated using G*Power 3.0.8 (Erdfelder et al., 1996) with conservative estimates of power, effect size, and correlation between repeated measures. Data from the braced measures in Sanderson and Kenyon (2005) were used in these estimates.

For each of the bracing conditions, a 7x4 (muscle x time points) repeated measures analysis of variance (ANOVA) tests was conducted. In the unbraced condition, the four time points were the two no-brace, no-feedback conditions (NB-NF and NB-NF2) and the first and last minute of feedback (NB-F1 and NB-F20). Similar collection periods were chosen in the braced condition with the NB-NF2 trial replaced by B-NF; thus, analysis occurred on NB-NF, B-NF, B-F1, and B-F20. When Mauchly's test of sphericity was significant, p-values were corrected by using the Greenhouse-Geisser adjusted values. The alpha level was set *a priori* to 0.05.

When the interaction term of the 7x4 ANOVA test was significant, planned post hoc analyses were conducted. First, pairwise comparisons between the three triceps surae muscles were calculated using a Bonferroni correction for multiple comparisons. Next, a 1x4 ANOVA was conducted for each muscle. When the 1x4 ANOVA test was

significant, all possible pairwise comparisons between conditions were conducted resulting in six comparisons (Table 3.1). The Bonferroni correction factor was again applied to all comparisons.

Table 3.1. Pairwise comparisons computed when 1x4 ANOVA was significant

	NB-NF	B-NF	B-F1	BF-20
B-NF	1			
B-F1	2	4		
BF-20	3	5	6	

To test for fatigue, the median power frequency of each muscle was computed on the demeaned EMG data and the four time points of interest were compared with a series of 1x4 ANOVA tests. Similar analyses were conducted on time-to-peak EMG data to determine if a shift in the temporal structure of the responses occurred. When the ANOVA tests were significant, post hoc contrast analyses were conducted comparing each time point to the baseline, NB-NF, condition as well as to the previous condition (ie. B-NF to NB-NF, B-F1 to B-NF, and B-F20 to B-F1).

To test the repeatability of EMG data during cycling, paired samples t-tests were used to compare the NB-NF and NB-NF2 trials for each muscle. RMS EMG was used to allow for direct comparison with Dorel et al. (2007) who recently reported that there were no differences in RMS EMG of ten lower limb muscles across testing periods separated by a one hour non-fatiguing protocol.

4 RESULTS

4.1 Description of Participants

Thirteen participants, eight males and five females, volunteered to participate in this investigation. Three female participants were unable to maintain the required workload at the testing cadence, so data collection was stopped and their data were considered incomplete. Due to equipment failure, one male data set from the braced condition was lost leaving nine complete sets of data. The nine participants included in the analysis had a mean (SD) age of 25.67 (± 1.32) years, mean height of 1.78 (± 0.05) m, and mean mass of 74.28 (± 9.81) kg.

4.2 Cadence

Since prior studies have shown that SOL and GAS respond differently to cadence manipulations, it was important to confirm that participants cycled at the standardized 80 rpm across the entire testing period. Table 4.1 shows the average cadence maintained during the ten cycles analyzed from the data collection period during select trials. The four trials used in all analyses include the two no-feedback trials as well as the first and last minute of the feedback period. There was no significant difference in cadence within the unbraced ($p = 0.63$, $F_{3,24} = 0.59$, MS = 0.972, $\eta_p^2 = 0.07$) or braced ($p = 0.91$, $F_{3,24} = 0.18$, MS = 0.47, $\eta_p^2 = 0.02$) conditions as calculated using two separate 1x4 repeated measures ANOVAs. Individual cadence data are provided in Appendix B.

Table 4.1. Mean cadence and standard deviation across participants in the four analyzed collection periods, with and without the brace.

No Brace	Average Cadence (rpm)	Standard Deviation	Brace	Average Cadence (rpm)	Standard Deviation
NB-NF	81.34	1.21	NB-NF	80.73	0.77
NB-NF2	80.94	1.24	B-NF	80.29	1.52
NB-F1	80.55	1.86	B-F1	80.65	1.63
NB-F20	81.07	1.21	B-F20	80.30	2.22
Mean	80.98	1.38	Mean	80.49	1.56

4.3 Integrated Electromyography

Changes in iEMG of each of the seven muscles in response to different conditions were the major variables of interest in the current study. The following results present only the *p*-values in the discussion of significance. When means are presented followed by the symbol ± and another number, the latter number refers to the standard deviation. Full results of the statistical tests are presented in Appendix C: for ANOVA and post hoc contrasts - F-value, degrees of freedom, mean sum of squares, effect size, *p*-value; for pairwise comparisons - mean difference, standard error, lower and upper bounds of 95% confidence interval, *p*-value.

4.3.1 Unbraced

ANOVA

In the unbraced condition, there was no significant muscle by condition interaction effect ($p = 0.07$) nor was there a significant main effect of condition ($p = 0.17$). There was a significant main effect of muscle ($p = 0.04$) indicating that the iEMG differed between muscles. The main effect of muscle was not a primary concern without an interaction effect showing differences across conditions, so no further analyses were conducted.

The largest decrease in SOL iEMG was 18±27% at NB-F20 compared to the NB-NF condition. Although it was likely due to participants' inability to use biofeedback without the brace, another potential reason for the non-significant ANOVA test could have been the large variability within certain muscles; for example, within the TA iEMG, there were large increases of up to 197±267% (NB-NF vs NB-F1) but the variability masked this increase in the statistical tests. GM iEMG decreased by 16±14% from the NB-NF to the NB-F20 condition. Figures 4.1 and 4.2 display the SOL and TA iEMG and the SOL, LG, and MG iEMG across conditions while all mean (SD) data from each muscle are shown in Table 4.2.

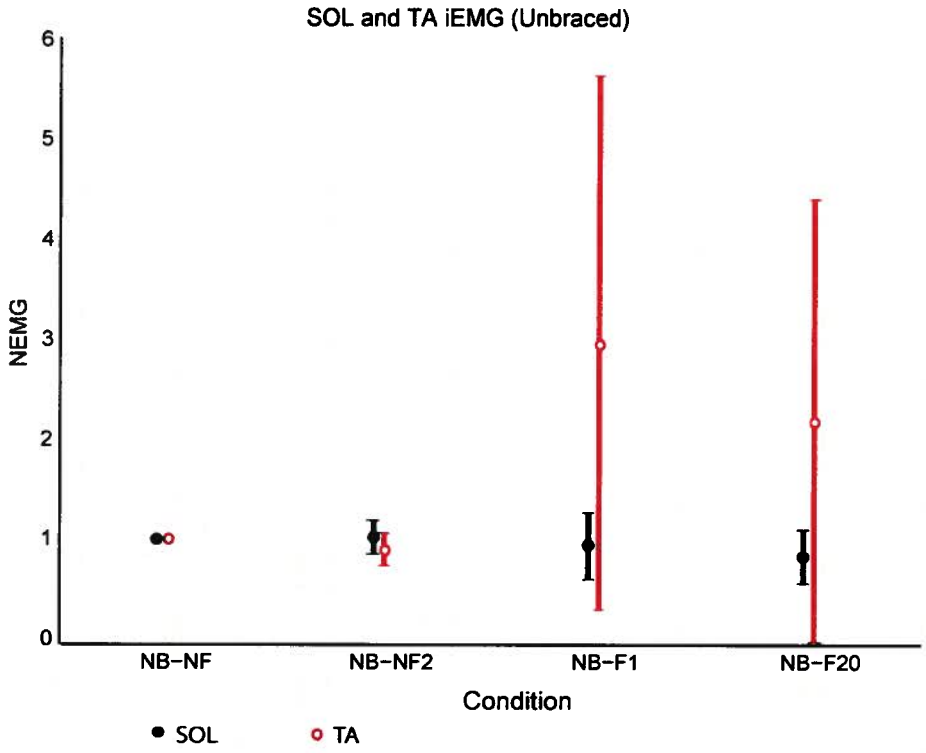

Figure 4.1. Mean participant (SD) SOL and TA iEMG from four conditions averaged across ten pedal cycles during the unbraced condition.

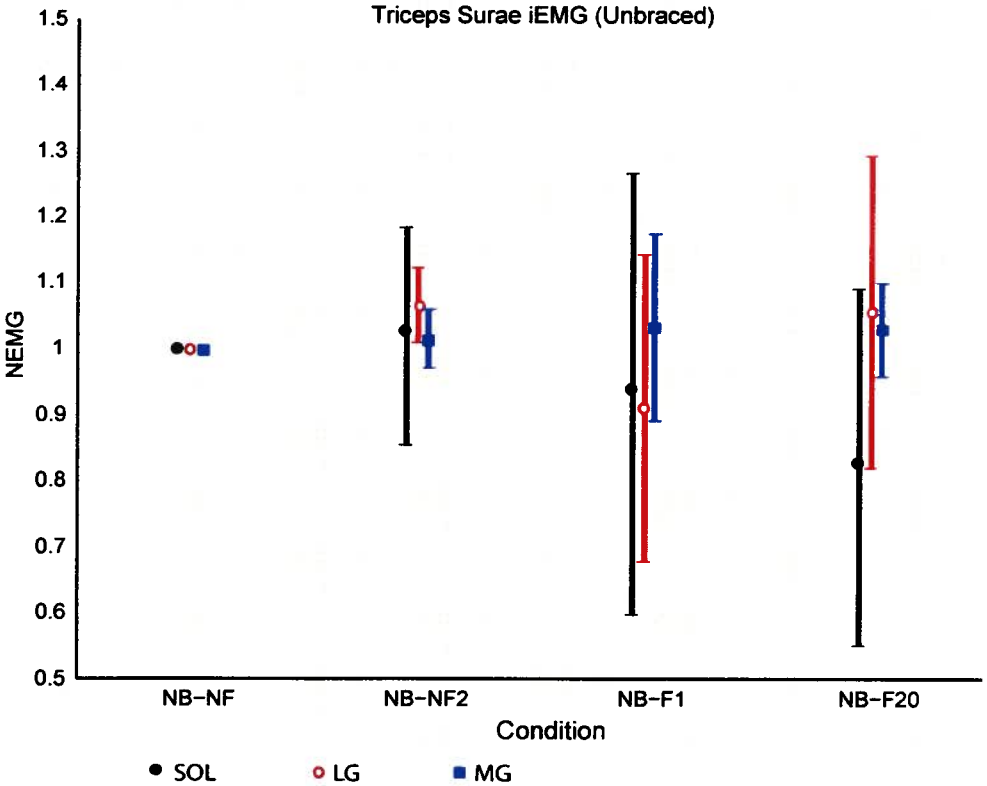

Figure 4.2. Mean participant (SD) SOL, LG, and MG iEMG from four conditions averaged across ten pedal cycles during the unbraced condition.

Table 4.2. Normalized iEMG (to the NB-NF condition) for each muscle averaged across participants for three different trials in the unbraced condition.

	NB-NF2		NB-F1		NB-F20	
	Mean	SD	Mean	SD	Mean	SD
SOL	1.02	0.17	0.93	0.33	0.82	0.27
LG	1.06	0.06	0.91	0.23	1.06	0.24
MG	1.02	0.04	1.03	0.14	1.03	0.07
TA	0.90	0.17	2.97	2.67	2.19	2.21
BF	1.04	0.12	1.02	0.34	1.18	0.41
RF	0.92	0.09	1.09	0.26	1.04	0.39
GM	1.05	0.17	0.92	0.10	0.84	0.14

4.3.2 Braced

ANOVA

In the braced condition, the 7x4 (muscle x time points) repeated measures ANOVA test showed a significant muscle by condition interaction ($p = 0.01$) as well as a significant main effect of muscle ($p < 0.001$). There was no main effect of condition ($p = 0.29$).

Having revealed a significant interaction effect, seven 1x4 ANOVA tests were conducted to investigate trends within a single muscle. All three triceps surae muscles showed significant decreases in iEMG (SOL, LG, MG: $p < 0.001$) while TA iEMG increased significantly ($p = 0.03$). Neither the iEMG of BF ($p = 0.11$) nor RF ($p = 0.64$) changed with feedback during the braced condition. There was a significant decrease in GM iEMG in the braced condition ($p < 0.01$). Figures 4.3 and 4.4 display the iEMG of the SOL and TA and the SOL, LG, and MG respectively during the braced conditions while all mean (SD) data are shown in Table 4.3.

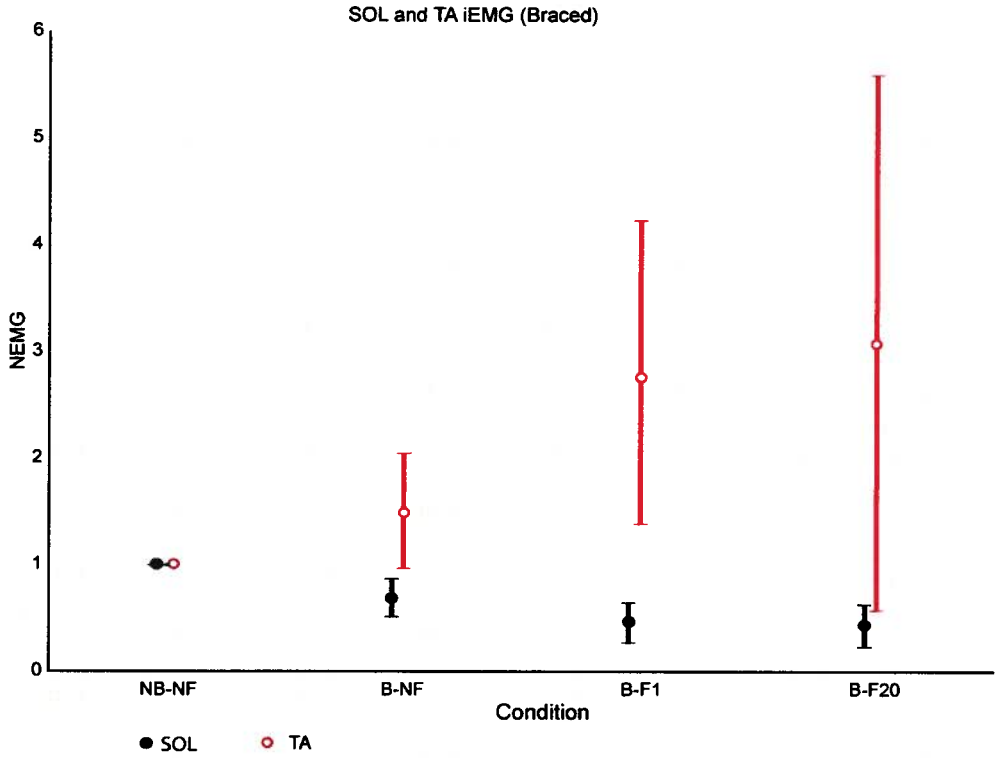

Figure 4.3. Mean participant (SD) SOL and TA iEMG from four trials averaged across ten pedal cycles during the braced condition.

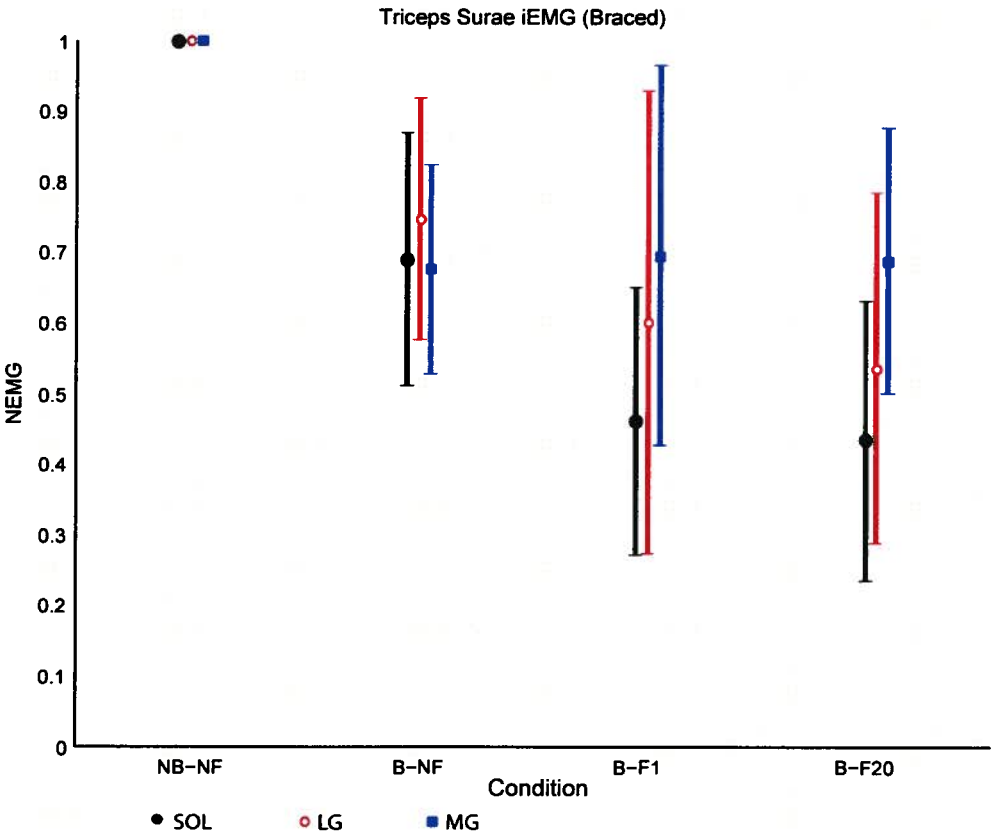

Figure 4.4. Mean participant (SD) SOL, LG, and MG iEMG from four trials averaged across ten pedal cycles during the braced condition.

Table 4.3. Normalized iEMG (to the NB-NF condition) for each muscle averaged across participants for three different trials in the braced condition.

	B-NF		B-F1		B-F20	
	Mean	SD	Mean	SD	Mean	SD
SOL	0.69	0.18	0.46	0.19	0.43	0.20
LG	0.75	0.17	0.60	0.33	0.54	0.25
MG	0.68	0.15	0.70	0.27	0.69	0.19
TA	1.51	0.54	2.81	1.42	3.09	2.51
BF	1.10	0.18	1.34	0.49	1.43	0.70
RF	1.04	0.31	1.16	0.41	1.00	0.45
GM	1.05	0.22	0.89	0.17	0.80	0.15

Pairwise Comparisons

In order to draw conclusions about the effect of bracing and biofeedback between the triceps surae muscles, pairwise comparisons were necessary. The Bonferroni correction factor was applied manually so the p-value was significant at 0.017. The tests showed that there were no significant differences between SOL and LG ($p = 0.08$) or LG and MG ($p = 0.27$); however, there was a significant difference between iEMG of the SOL and MG muscles ($p = 0.017$).

For each of the five muscles that revealed a significant ANOVA test, six pairwise comparisons were conducted to determine in which conditions iEMG differed. Compared to the NB-NF condition, there was a significant reduction in iEMG in all three of the other conditions (B-NF, B-F1, and B-F20) in SOL ($p < 0.01, p < 0.001, p < 0.001$ respectively) and LG ($p = 0.01, p = 0.04, p < 0.01$). There were also significant iEMG reductions in MG in the B-NF and B-F20 conditions ($p < 0.01, p < 0.01$) compared to the NB-NF condition but not when compared to the B-F1 condition ($p = 0.06$). The addition of the brace without feedback (B-NF) led to a 31±18% decrease in SOL iEMG, a 25±17% decrease in LG iEMG, and a 32±15% decrease in MG iEMG. When feedback was first presented, there was a significant decrease in SOL iEMG (B-NF vs B-F1, $p = 0.03$) but there was no significant decrease in iEMG of LG ($p = 0.41$) or MG ($p = 1.00$). However, there was a significant decrease in both SOL iEMG ($p = 0.03$) and LG iEMG ($p = 0.02$) from the B-NF to the B-F20 condition while there was still no change with MG iEMG ($p = 1.00$). With bracing and biofeedback SOL iEMG was 54±19% lower than the initial condition, and 23% lower than with bracing alone. The B-F1 and B-F20

conditions were compared to test for learning across the feedback period. There was no reduction in iEMG across the feedback period in SOL, LG, or MG ($p = 1.00$ for all).

TA iEMG did not increase significantly from the NB-NF condition in the B-NF condition (51±54%, $p = 0.13$) or B-F20 condition (209±251%, $p = 0.223$). However, TA iEMG increased significantly in the B-F1 condition compared to the NB-NF condition with increases of 181±142% in the B-F1 condition ($p = 0.03$). Opposite the decrease seen in SOL iEMG, there was a significant increase in TA iEMG with feedback (B-NF vs B-F1, $p = 0.04$). There was no change in TA iEMG across the feedback period (B-F1 vs B-F20, $p = 1.00$). The large variability in the B-F20 condition likely contributed to the non-significant increases from the NB-NF and B-NF conditions.

The significant reduction in GM iEMG was evident between the NB-NF condition and the B-F20 condition ($p = 0.02$) but not when contrasted with the B-NF ($p = 1.00$) or B-F1 ($p = 0.46$) conditions. There was a significant reduction in GM iEMG with feedback ($p < 0.01$) but no significant reduction across the feedback period ($p = 0.17$).

4.4 Time-to-peak EMG

The time-to-peak EMG was determined in the two gastrocnemius muscles to investigate one aspect of the temporal structure of the pedal cycle and how it might change across conditions. In the unbraced condition, the ANOVA test determined that there were no significant differences in time-to-peak EMG in the LG or MG muscles ($p = 0.961$ and $p = 0.154$ respectively). However, in the braced condition, the time-to-peak EMG changed in both LG and MG ($p < 0.01$ and $p = 0.05$). Post hoc contrast analysis revealed differences in time-to-peak LG EMG from the NB-NF trial to the B-F1 ($p = 0.02$) and B-F20 ($p = 0.02$) as well as from B-NF to B-F1 ($p = 0.03$). The time-to-peak MG EMG was lengthened in the B-NF and B-F20 trials compared to the NB-NF trial ($p = 0.01$, $p < 0.01$) and in the B-F20 compared to the B-F1 ($p = 0.02$). From the NB-NF to the B-NF and further to the B-F20 trial, the time-to-peak EMG increased from 37.7±10.2% to 42.9±8.9% to 47.6±10.0% in LG and from 31.6±3.3% to 33.2±2.9% to 36.5±5.6% in MG. Figure 4.5 displays the mean time-to-peak EMG for the LG and MG muscles across four trials in the braced condition. Full results of the statistical tests are presented in Appendix C.

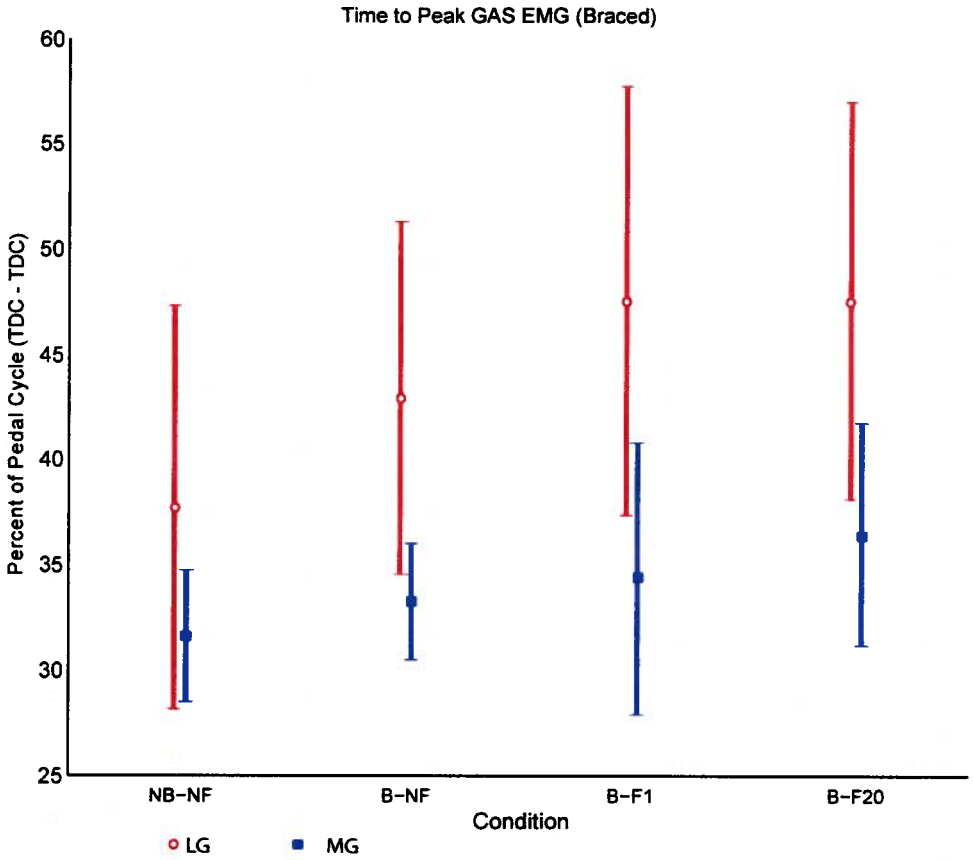

Figure 4.5. Mean time-to-peak EMG in the LG and MG muscles averaged across ten pedal cycles in the braced condition.

4.5 Repeatability

The two NB-NF trials conducted minutes apart allowed testing of the repeatability of muscle excitation during cycling. Paired samples t-tests showed no significant differences in SOL ($p = 0.92$), LG ($p = 0.78$), MG ($p = 0.35$), TA ($p = 0.21$), BF ($p = 0.33$), or GM ($p = 0.51$). A significant difference in RF RMS EMG was found between the NB-NF and NB-NF2 trials ($p = 0.04$); mean RMS EMG decreased from the first trial to the second trial. Full statistical results (t-statistic, degrees of freedom, p-value) are presented in Appendix C.

4.6 Median Power Frequency

Median power frequency (MPF) of EMG is often used as a measure of muscular fatigue. In the unbraced condition, across the four analyzed time points, there were significant differences in the median power frequency of two of the seven tested muscles. Significant ANOVA tests showing differences in SOL ($p = 0.02$) and LG ($p = 0.05$) were followed up with contrast analyses which showed significantly decreased MPF between the NB-NF2 condition and the NB-F1 condition (SOL: $p = 0.01$; LG: $p = 0.01$). The SOL and LG MPF returned to the levels of NB-NF2 by the NB-F20 condition, so this initial reduction did not appear to be representative of muscular fatigue resulting from prolonged exercise. In the braced condition, there was again significantly different MPF in SOL EMG ($p = 0.03$) and LG EMG ($p = 0.01$). Decreases in SOL MPF were seen between the B-NF trial and the B-F20 trial ($p = 0.03$) and in LG MPF between the NB-NF trial and B-F20 trial ($p = 0.01$). Additionally, the GM MPF ($p = 0.02$) increased from the NB-NF to the B-F20 condition ($p = 0.02$). Figures 4.6 and 4.7 display the MPF of lower leg muscles and upper leg muscles respectively for the braced condition. Complete results (F-value, degrees of freedom, mean sum of squares, effect size, p-value) of the statistical tests are presented in Appendix C.

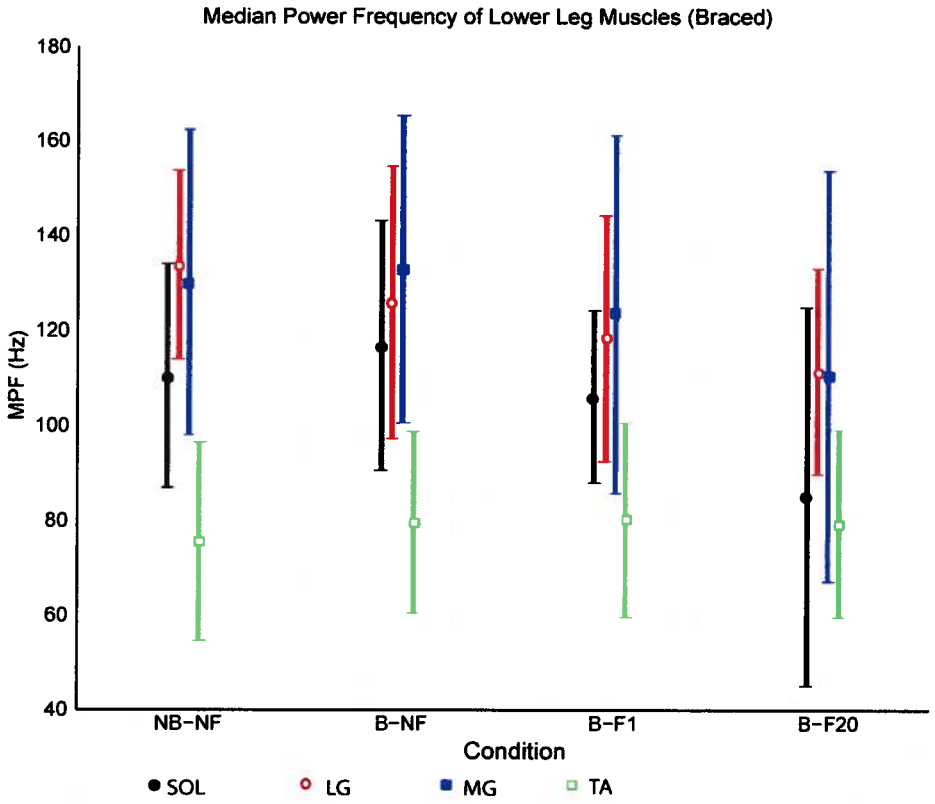

Figure 4.6. Mean participant (SD) median power frequency of the SOL, LG, MG and TA MPF from four trials averaged across ten pedal cycles during the braced condition.

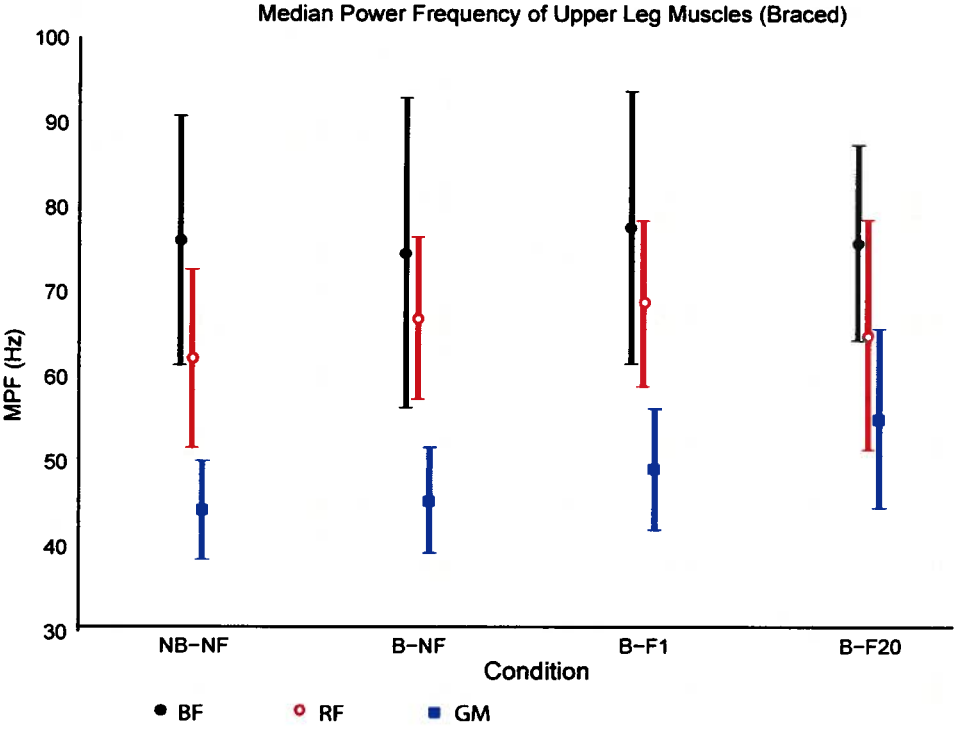

Figure 4.7. Mean participant (SD) BF, RF, and GM MPF from four trials averaged across ten pedal cycles during the braced condition.

4.7 Soleus EMG Biofeedback

Raw SOL EMG was recorded continuously throughout the testing period. This EMG was rectified and averaged and used to provide the visual feedback to the rider. Figure 4.8 shows the rectified, averaged, filtered SOL EMG for each power phase (from TDC to BDC) of one participant. Average SOL EMG decreased from the NB-NF condition to the B-NF condition and then further decreased in the B-F1 condition. After this initial decrease with feedback, average SOL EMG did not improve over time. As reported above, there was no significant difference in SOL iEMG across the feedback period (B-F1 and B-F20) indicating that there was no learning across this single testing session.

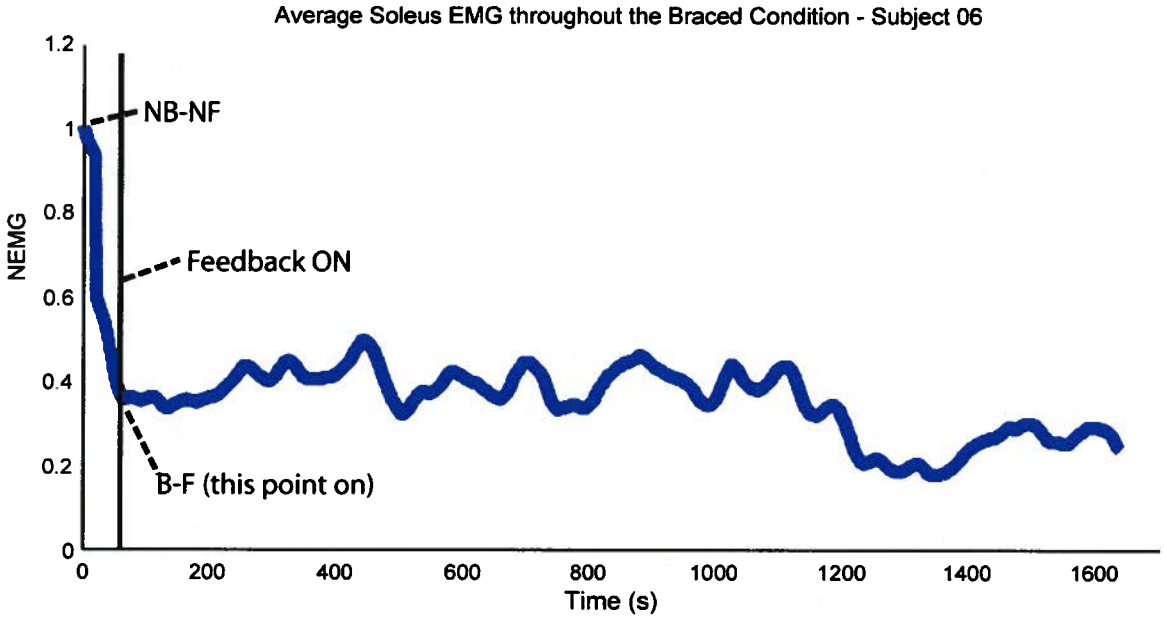

Figure 4.8. Average SOL EMG across the entire testing session in the braced condition from an individual participant (Participant 06). Data were filtered with a 2nd order low pass Butterworth filter with a 10Hz cutoff frequency.

5 DISCUSSION

5.1 Overview

The most important finding of the present study was that using only the bracing method, each of the triceps surae muscles showed a similar reduction of iEMG, approximately 30% compared to baseline; however, with the combined application of bracing and biofeedback, SOL iEMG was immediately reduced by a further 23% while MG and LG excitation did not change. However, by the end of the feedback period, LG iEMG was significantly reduced by 21% whereas the MG iEMG did not change. This finding suggested that the MG iEMG reached an excitation plateau, unable to further reduce its excitation potentially due to its role as a knee flexor. LG iEMG decreased along with SOL iEMG and may have a greater role in ankle plantarflexion.

The present study provided evidence to suggest that SOL and GAS excitations do not always change in the same direction with the same function, leading to the conclusion that they function differentially. The biarticular nature of the gastrocnemius (both medial and lateral heads) leads to its function as both an ankle plantarflexor and a knee flexor but the relative contribution of its excitation to one function or the other remained unclear. Soleus, meanwhile, has no involvement at the knee but shares the function of ankle plantarflexion. While motor neurons are anatomically distinct and direct the activation of a single muscle, due to their role as synergists, it is possible that SOL and GAS could have functionally overlapping motor neuron pools. Conversely, the motor neuron pools could be both anatomically and functionally distinct but activated simultaneously. To isolate the role of GAS at the knee, a combination of ankle bracing and SOL EMG biofeedback was used to passively and actively eliminate plantarflexion; by recording from SOL, an indirect measure of the reduction in the role of GAS as a plantarflexor was determined.

Because bracing and biofeedback were tools employed to help reduce SOL EMG, comparisons between the braced and unbraced condition were not of concern. The unbraced trials were designed to show whether a mechanical tool was necessary to induce changes in SOL EMG. We found that there was no significant effect of condition on muscle excitation during the unbraced trials and thus participants were not able to use the

biofeedback alone effectively; this suggested that there was something inherent to the cycling motion that necessitated the use of the ankle plantarflexors. While one might argue that the feedback image was inadequate to induce the desired modification, its successful implementation during the braced condition made this proposition less probable. The remainder of the discussion focuses on the braced condition in which the desired modification of SOL EMG was induced.

5.2 Support for the Hypotheses

The first hypothesis contained two parts, both of which were accepted. EMG biofeedback was a useful tool in the reduction of SOL EMG beyond that seen in bracing. Additionally, biofeedback was not useful in modifying SOL EMG without the application of a mechanical brace.

The second hypothesis was rejected for the medial gastrocnemius and accepted for the lateral gastrocnemius. MG excitation did not decrease during SOL EMG biofeedback while LG EMG decreased by the end of the biofeedback protocol in the braced condition.

5.3 Mechanism for Reducing Soleus EMG

Sanderson and Kenyon (2006) showed that bracing the ankle with a pre-formed ankle-foot orthosis reduced SOL excitation by an average of 30% during cycling. The current study confirmed these results and sought to further this reduction using SOL EMG biofeedback. Biofeedback was successful in inducing a voluntary reduction in SOL iEMG beyond that seen in bracing. When shown visual feedback, in both the braced and unbraced conditions, participants attempted different strategies to minimize the displayed EMG. While some participants reported using a pull-up strategy in which they focused on pulling up with the quadriceps muscles, one strategy that participants consistently reported using in order to reduce SOL excitation was actively contracting the antagonist TA. This was termed the TA excitation strategy, and the neural implications of this strategy must be understood. Given the large increases in TA excitation of nearly 210%, the likely mechanism through which the decrease in SOL excitation occurred was through heightened reciprocal inhibition from the TA. During voluntary movement, inhibition on the agonist muscle decreased to facilitate its action while inhibition of antagonist muscles increased to promote their silence and to ensure that no unwanted

stretch reflex activity was evoked (Nielsen, 2004, Windhorst, 2007). In the current investigation, by voluntarily ramping up TA excitation, projections to SOL through interneurons in the spinal cord may have created an inhibition in the SOL muscle as has been shown previously by Tanaka (1974), Pyndt et al. (2003), and Nielsen (2004).

We suggested that a prolonged TA excitation strategy was not sustainable as TA is predominantly a fast-twitch muscle and presumably would fatigue throughout the protocol with constant excitation. Under normal conditions during a pedal cycle, TA had two activation bursts, with both peaks occurring in the recovery phase of cycling (180-360°), while SOL had a single excitation burst with a peak at 90° (Chapman et al., 2006). Thus, to counter the SOL excitation burst, the TA excitation strategy would require the muscle to be active during the power phase, in addition to its normal function in the recovery phase, and this might result in fatigue. The TA muscle excitation pattern did change with bracing and feedback; in addition to the two bursts corresponding to dorsiflexion, there was a burst during the early power phase. Because median power frequency of TA EMG did not change over the testing interval, it was concluded that TA did not experience muscular fatigue. It was proposed that participants activated and deactivated TA throughout the 20-minute protocol to avoid reaching muscular fatigue. Thus, from minute to minute, the TA EMG excitation trace may have been quite variable. Across the prolonged feedback period, TA iEMG increased and decreased with no consistent time interval over which the changes occurred. The number of cycles over which TA was highly active varied between participants who were free to implement their own on/off timing; at any one time point, some participants may have been implementing the TA strategy while others may have been "resting" their TA. This accounts for the wide variability seen in the TA iEMG data as the timing and length of activation occurred differently across all participants. This wide variability may also help explain why the TA excitation strategy was not successfully employed in the unbraced condition; between the braced and unbraced conditions, participants had similar TA iEMG increases in the feedback condition compared to the no feedback baseline but the larger variability in the unbraced condition led to non-significant statistical tests. Without the brace neutralizing the position of the ankle joint, it was more difficult to voluntarily eliminate plantarflexion and continually activate the TA muscle.

The likely mechanism whereby SOL excitation was significantly decreased in the braced-biofeedback protocol was through reciprocal inhibition from TA. It is important to note that TA also has reciprocal connections with GAS. However, it has been shown that TA excitation affects SOL and GAS differently with a greater reciprocal inhibition between TA and SOL than TA and GAS (Nielsen and Kagamihara, 1993).

5.4 Effects of Reduced Soleus Excitation on Medial Gastrocnemius Excitation

The previously discussed experiment by Sanderson and Kenyon (2005) was designed to determine the relative contribution of the MG muscle to plantarflexion compared to knee flexion; however, they did not differentiate between the two roles because the changes in MG and SOL excitation were nearly identical, with the bracing protocol leading to an average 30% EMG decrease in both muscles. The current augmentation of the bracing protocol with a SOL EMG biofeedback display led to a further 23% reduction in SOL iEMG with no concurrent reduction in MG iEMG. The appearance of this excitation plateau suggested that MG excitation cannot be further reduced because of its role as a knee flexor. Since MG iEMG was unchanged with biofeedback, one might speculate that the contribution of the MG at the ankle joint was removed by bracing and the remaining excitation was related only to knee joint motion. However, since SOL excitation was not completely eradicated, it was not possible to tease apart this relationship. This result provided evidence for the possibility that the remaining excitation corresponds to knee flexion, but a different methodology must be employed in future studies to verify these results.

Further evidence for the increased contribution of the knee joint motion came from the shift in time-to-peak EMG. In the braced condition, there was a temporal shift in GAS recruitment with peak EMG occurring later in the pedal cycle. The time-to-peak EMG was delayed from 37.7% of the pedal cycle in the NB-NF condition to 47.6% in the B-F20 condition in LG and from 31.6% to 36.5% in MG. During the pedal cycle, the biarticular GAS often has two bursts of activity; the earlier one occurs during the power phase and corresponds to ankle plantarflexion while the latter one occurs during the recovery phase and corresponds to knee flexion. This temporal shift in time-to-peak EMG indicated that the GAS iEMG now reflected only its role in knee flexion. With bracing and biofeedback, the time-to-peak EMG occurs later in the pedal cycle and when

combined with the finding that MG iEMG does not decrease, it suggested that knee flexion because the only role of MG as plantarflexion and SOL EMG are reduced.

The motor neuron pools for the LG and MG are directed by neural drives for both knee flexion and ankle plantarflexion, while that of SOL is directed only by plantarflexion. In the following discussion about motor neuron pools, it is important to note that we are not suggesting that individual motor neurons influence multiple muscles; motor neuron pools are anatomically distinct. Instead, we suggest that there is a functional overlap of the motor neuron pools. Based on their different responses to biofeedback, there must have been some isolation between the motor neuron pools of SOL and MG. Four possible motor neuron pool organizations are displayed in Figure 5.1.

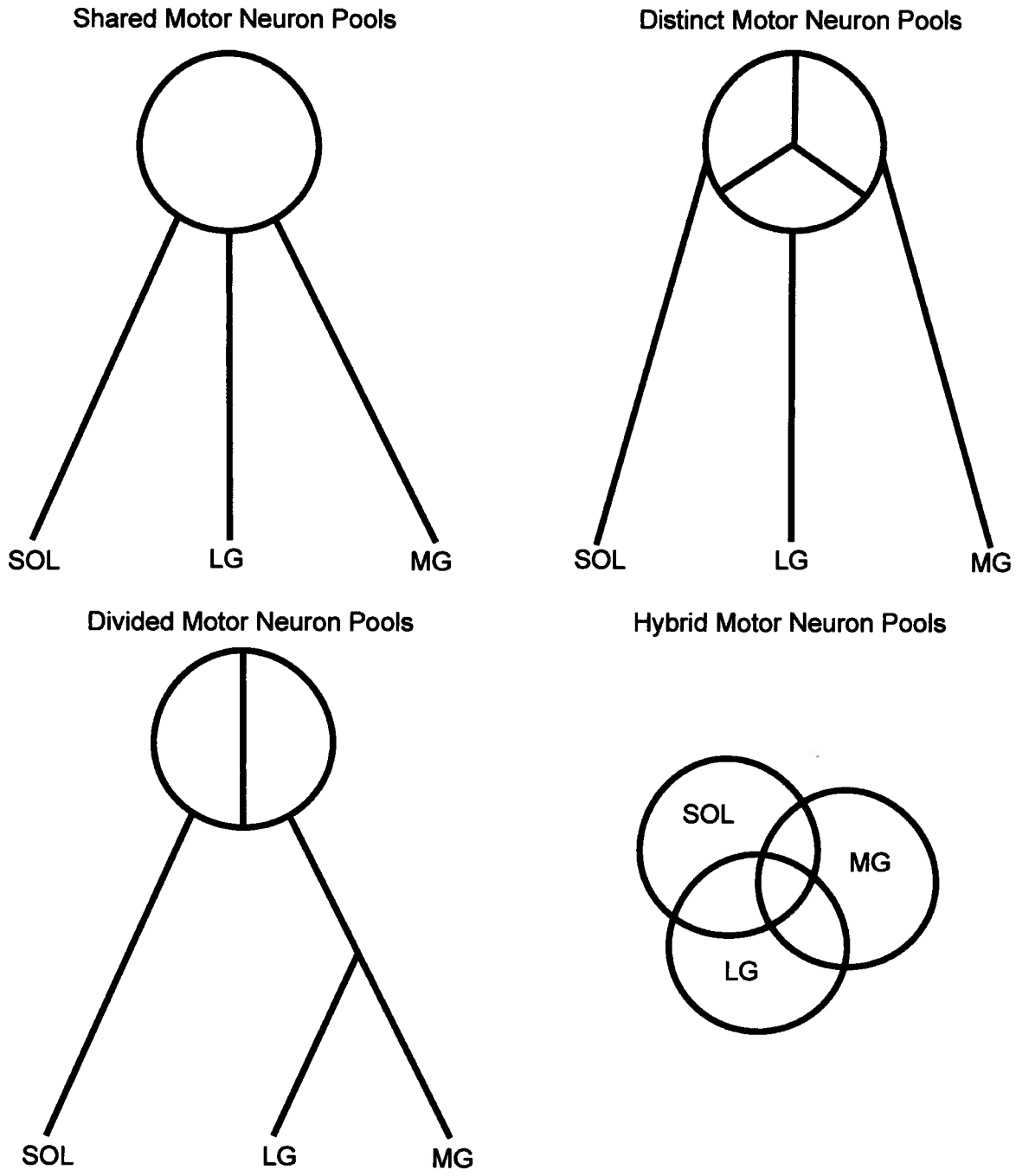

Figure 5.1. Four potential organizations of the triceps surae motor neuron pool including the two extremes, shared or distinct. Two examples of other possible organizations include divided, in this case shared motor neurons between the LG and MG and distinct from SOL, or hybrid, some degree of shared and distinct motor neurons between all three muscles.

The current study suggested that a single, shared motor neuron pool for the triceps surae muscles cannot exist as comparisons show that the SOL and MG iEMG are significantly different. We suggest that the motor drive to MG differed too greatly from that of SOL during biofeedback for a fully common input to exist. Distinct motor neuron pools activated simultaneously could result in these differential responses to biofeedback; however, a hybrid of the two ideas, with some motor neurons contributing to all muscles and others to one part of the triceps surae is also possible. Given the common response to bracing, there are likely some common connections among the three muscles, but it remains to be seen whether this occurs at the motor neuron level. The relationship between MG and LG is not easily deciphered with the present results. Statistically, both muscles show a decrease in iEMG with bracing and no initial change in iEMG with biofeedback; however, by minute twenty of the braced-biofeedback protocol, LG iEMG was significantly decreased beyond bracing alone while MG iEMG did not change from the B-NF condition. In Figure 5.1, the two muscles are diagramed together (divided) or separately (distinct); in both of these examples, the motor neuron pools are distinct from SOL. These different responses to biofeedback indicated that there were some distinct motor neurons within the LG and MG motor neuron pools but pairwise comparisons show no statistical difference between the two muscles when averaged across all conditions and thus limit the ability to comment conclusively. From the current results, we suggest that there are varying levels of common motor neurons between SOL, LG, and MG, and a hybrid system most accurately depicts this possibility.

Since LG decreased significantly by the end of the biofeedback protocol, it is likely that SOL and LG have a greater common input than SOL and MG. McLean and Goudy (2004) studied the effects of sustained contraction on surface EMG of SOL, LG, and MG. The correlation among RMS EMG was highest between SOL and LG ($R^2 = 0.662$) which had a coactivation synergism as opposed to SOL and MG ($R^2 = 0.114$) and LG and MG ($R^2 = 0.155$) which had a trade-off synergism. Knee joint angle was held constant so this synergism only related to the action at the ankle joint. It appeared that SOL and LG were more closely related than both the SOL/MG and LG/MG pairs, and the authors suggested that this might relate to their common innervations. In 26 of 37 cadaver limbs, the SOL and LG nerves branched from a common trunk (Parratte et al., 2002). Further, Sirin and Patla (1987) previously conducted a similar submaximal sustained plantarflexion

experiment at two knee angles (0 and 120°). The predominant synergy was found between SOL and LG not SOL/MG or LG/MG, with coactivation becoming stronger over time. Evaluation of the pairwise comparisons shows that LG iEMG was not significantly different than SOL or MG iEMG but the latter two muscles differed; we suggest that the response in the LG may indicate its intermediate role. The idea of a hybrid system and an intermediate role of LG fits well with the partitioning hypothesis and ideas of neuromuscular compartmentalization. English et al. (1993) and Windhorst et al. (1989) suggested that the gastrocnemius muscles were compartmentalized with four neuromuscular compartments in LG and eight compartments in MG. This compartmentalization might have led to the findings of a different degree of common input to the triceps surae muscles. We must also note that recordings from the SOL muscle were taken from the lateral part of the muscle; had we recorded more medially, we may expect to see a greater common input with MG. LG might share more common input with each of SOL and MG than SOL and MG do with each other. In LG, the concurrent decrease with SOL EMG during biofeedback provided evidence of a possible functional difference between LG and MG, with LG playing a greater relative role in plantarflexion while MG EMG remains heightened due to its larger role in knee flexion. While it is established that all three muscles do not share identical motor neuron pools, the degree of overlap, if any, between each of the three pools remains uncertain and should be investigated in future research.

5.5 Ability to Use Biofeedback

The mechanical brace was necessary to reduce SOL excitation using EMG biofeedback. There are two potential ways in which bracing may have influenced the ability to modify SOL EMG with feedback. The first related to the level of experience with the task while the second related to the mechanical limitations imposed by the brace.

All of the participants had prior cycling experience and were comfortable in performing a consistent cycling action while unbraced. Cycling, like most motor skills, involves the coordination of multiple muscles working about multiple joints. Since synergist muscles execute the same function, the motor task could have been performed in numerous ways with different combinations of muscles leading to the generation of the same joint torques. However, even with the possibility of different muscle excitations

both between individuals and between pedal cycles, healthy individuals employ similar muscle activation patterns while performing the same well-learned tasks (Prilutsky and Zatsiorsky, 2002). Adults who have not cycled for years remember how to ride a bicycle suggesting that cycling is a highly learned task, one that has been trained through hours of practice. It was suspected that under normal conditions, ankle plantarflexion is incorporated into the cycling motor pattern; current results indicated that the muscle excitation pattern for cycling was too well-established to be voluntarily modified by the biofeedback within a single testing session. While the cycling action can be accomplished in other ways, the use of the triceps surae muscles to transfer forces to the pedal and to help generate the necessary extensor moment was ingrained in the movement pattern.

The application of the brace modified the requirements of the cycling task creating a new, but similar task. In studying the SOL H-reflex, bracing is often employed to ensure that the geometry of the muscle does not change. Misiaszek (2003) noted that the shortcoming of this method was that the movement became unnatural creating a novel and artificial motor task. We suggest that participants were able to immediately modify this similar task and effectively use biofeedback because the muscle activation pattern is not as well-defined. The current study employed the same perturbation in two similar tasks; however, the difference in experience with each task results in a different response to the perturbation, with participants only able to use biofeedback in the less familiar braced condition.

The second, and perhaps the primary reason, for the difference in the ability to use biofeedback related to the kinematic requirements of the task. Modification of the cycling task can change the movement pattern required during its performance. For example, lowering the seat height reduced or eliminated ankle plantarflexion and led to changes in muscle requirements of the task (Sanderson and Cawsey, personal communication). Participants were still able to perform the cycling task; however, its muscle activation patterns were altered. In the unbraced condition, the ankle joint moved freely whereas in the braced condition, it was constrained to the neutral position. When participants were unable to eliminate plantarflexion, SOL EMG was not reduced. Cannon et al. (2007) showed that when participants were instructed to maintain the ankle in maximal dorsiflexion throughout the pedal cycle, they did not decrease the maximal

plantarflexion angle from normal cycling. In agreement with the aforementioned study, it took the elimination of plantarflexion through bracing to significantly reduce SOL EMG. It is suggested that participants were unable to reduce SOL EMG and use biofeedback in the unbraced condition because they could not restrict ankle joint motion without the aid of an external brace.

By mechanically bracing the ankle joint, the primary action of the SOL was removed. Providing biofeedback allowed for an individual to visualize the online SOL excitation and modify pedalling technique to reduce SOL EMG. However, within the braced condition, participants were unable to eliminate SOL EMG. It is difficult to predict the effect of previously learned skills on a similar task but it has been noted that when the environmental context of two performance situations were similar but the movement characteristics required were different, negative transfer effects can occur. This was particularly evident when it involved a change in spatial locations of a movement or timing structure of a movement (Magill, 2001). It was proposed that the movement and timing structure of the known skill of cycling may have interfered with one's ability to silence SOL with biofeedback. The residual excitation likely arose from the similarity between the two tasks creating some skill transfer from the unbraced condition. The SOL was actively involved in performing the unconstrained cycling motion and became active in the similar task. While biofeedback allows for participants to modify the task that they are learning, the well-learned task of cycling interferes with their ability to entirely eradicate SOL excitation. It is important to note that in the braced condition, after the initial improvement with feedback, participants did not continue to improve over time. It is suspected that the twenty-minute biofeedback period was too short to allow for learning to occur. Two groups of individuals with incomplete spinal cord injury and who suffered from Trendelenburg gait were given biofeedback to modify their gait. In this abnormal walking pattern, gluteus medius excitation is too low; audio biofeedback was provided to individuals when the muscle excitation was below a certain threshold. One group received feedback for 30 minutes per day while the other group received it throughout the entire day. Over the two-month period, the limited biofeedback group reduced their hip drop by 50% while the constant feedback group returned to almost normal gait (Petrofsky, 2001). Over time, one might induce long term changes to supraspinal and sensory afferent inputs with biofeedback leading to

modification of the output from the motor cortex, potentially in both the braced and unbraced conditions.

While the braced motor task was likely influenced by unbraced cycling, it was modifiable by biofeedback. The way in which feedback modifies the motor pattern can only be speculated. Perez et al. (2005) suggested that during motor learning, changes to reflex circuitry required changes in presynaptic inhibition of synapses between sensory afferents and motor neurons. He proposed that visual input travels from the visual cortex to alter the motor cortex, which sends certain commands from descending drive to spinal interneurons. In the current study, those descending commands direct a reduction in SOL EMG. In their novel visuo-motor skill training task, Perez et al. (2005) found that feedback improved performance of the task and led to a significant immediate reduction of the SOL H-reflex recruitment curve. They contended that there were changes in descending drive to the interneurons conveying the inhibition and suggested that to optimize the motor pattern during skill acquisition, visual input and proprioceptive information were centrally integrated. The visuomotor task required increased attention resulting in an increased motor cortical excitability, an adaptation in the motor cortex. The proposed pathway explains the successful modification in SOL EMG, and we suggest that the descending drive to SOL was difficult to silence due to residual excitation from the known motor pattern.

5.6 Localized Effect

Previous experiments by Sanderson and colleagues (Sanderson and Kenyon, 2005, Sanderson et al., 2006) limited EMG collection to the triceps surae and TA muscles. The addition of muscle excitation recordings from more proximal muscles showed that the effect of bracing and biofeedback may have been limited to the muscles controlling the ankle joint including both monoarticular and biarticular muscles. Excitation of BF, RF, and GM which act at the knee and hip joints were not increased to compensate for the decreased contribution of the ankle muscles to the total extensor moment. Instead, BF and RF excitation did not change while GM excitation decreased. Thus, the recorded upper leg muscles were not responsible for compensating for the reduced ankle extensor moment. The compensatory increase in excitation may have come from other proximal muscles from which we did not record. Comments from

participants suggested that the quadriceps muscles worked harder in the braced condition compared to the unbraced condition. So while the present results suggested that the modifications induced by bracing and biofeedback were localized, with the effect narrowly focused on the muscles surrounding the ankle, it was likely that the vasti muscle excitation increased as the compensatory mechanism for decruitment of the triceps surae muscles. It was a novel finding that changes in the triceps surae group were independent of BF, RF, and GM.

5.7 Methodological Considerations

A number of factors have to be controlled or considered in the interpretation of the results including cadence, repeatability, fatigue, cutaneous input, and individual differences. Because Sanderson et al. (2006) among others (Ericson et al., 1985, Duchateau et al., 1986, Marsh and Martin, 1995, MacIntosh et al., 2000) have shown that cadence changes differentially affect SOL and MG, cadence must be held constant in order to compare between trials. Statistical analysis revealed that cadence was unchanged across the testing period. It was possible that monitoring cadence interfered with participants' abilities to modify SOL EMG. Given two different types of feedback, cadence and EMG, participants had to determine how frequently to focus on one type or the other. They were asked by the researcher to focus on the EMG biofeedback but to glance occasionally at the cadence feedback. If the researcher noticed the participant focusing too much on the cadence monitor, she verbally encouraged a switch to the biofeedback; however, there was no measure of how frequently this occurred. Future research employing two types of biofeedback should have a cadence monitor appear on the feedback display at regular intervals to ensure near constant attention is directed to the EMG biofeedback. Given the current measures, it was possible that the participants were focused on neither the biofeedback nor the cadence feedback. Qualitatively, many participants mentioned that in the first few minutes, they attended continuously to the EMG biofeedback as they tested strategies to minimize SOL EMG; however, they suggested that once they chose a strategy, they tend to pay less attention to the biofeedback as they could identify the lowest level of SOL excitation by 'feel'. While the participants were asked to minimize SOL EMG and we expected them to continually attempt to further reduce SOL EMG throughout the protocol, it seemed as though many

participants employed a self-defined optimal strategy chosen within the first few minutes. They continually used this strategy throughout the protocol which led to an excitation plateau and no further improvement with time. Devising a method to ensure that participants constantly work to reduce SOL excitation with EMG feedback would be useful for future applications of biofeedback.

By completing two NB-NF trials, data to test the repeatability of EMG patterns in cycling were available. Among the seven muscles, only the RF is significantly different between the two trials. The present data were compared to Dorel et al. (2007) who looked at the natural physiological variability of EMG patterns while cycling at 150W; current data were analyzed as RMS EMG over a 50ms window to match their analysis. Much like the previous study, these data showed that although there were intra-individual differences, the group means were not significantly different (with the exception of RF). Thus, the decision to normalize to a NB-NF baseline was deemed appropriate although the RF data may not have a representative normalization and confidence in these measures is reduced.

Due to the length of the cycling period, muscular fatigue could have played a role in altering muscle excitation. During sustained isometric contractions, a decrease in MPF has been accepted as a sign of muscle fatigue. The decrease in MPF was proposed to be caused by a decline in mean muscle fibre conduction velocity and discharge synchronization of the motor units (Ament et al., 1996). In the braced condition, MPF decreased in the SOL, LG, and GM with significant differences between either the NB-NF or B-NF condition and the B-F20 condition. Muscular fatigue in SOL and LG was surprising given the aim to voluntarily reduce SOL excitation. GM is a powerful hip extensor and because bracing of the ankle increased the need for hip extension (Sanderson and Kenyon, 2005), it might follow that muscular fatigue in this muscle occurred; however, since the GM MPF increased, fatigue was not related to the changing MPF. A second, indirect measure of neuromuscular fatigue was heart rate. From the third to the eighteenth minute, heart changes from 147.4±19.8bpm to 152.7±20.5bpm in unbraced cycling and 145.0±18.4bpm to 153.6±18.7bpm in braced cycling, variations of 3.5% and 5.9% respectively. Heart rate increased by 7.3% and 12.7% after 50 minutes and 100 minutes during a two hour cycling protocol (Lepers et al., 2000) which we can

extract to a 2.9% change over twenty minutes. Heart rate increased continually as testing progressed, and this increase was within the normal range expected due to cardiac drift.

Cutaneous afferents have been shown to influence the motor neuron excitability in SOL (Iles, 1996) so it was important to ensure that this was not the only influence. Cutaneous input from stimulation of branches of the common peroneal nerve on the dorsum of the foot led to a significant reduction in presynaptic inhibition of soleus Ia afferents (Iles, 1996); however, it is suspected that on the foot, the cutaneous input from the brace would be much like that of the shoe. McIlroy (1992) argued that the addition of an ankle-foot orthosis would not modulate the SOL H-reflex greatly; if there had been no change in the SOL, LG, or MG excitation during biofeedback, one might suggest that the changes to the cutaneous inputs from the brace itself could have played a major role in the reduction of muscle excitation. However, the biofeedback protocol induced a further decrease in SOL and LG iEMG with no changes in cutaneous input from the bracing alone protocol. Thus, decreased SOL excitation is not due to altered cutaneous input alone but the extent of the influence of cutaneous afferents in the braced protocol remains uncertain.

In the unbraced condition, there was a non-significant increase in TA iEMG, with the non-significant statistical test likely due to the variability in the data. Participants attempted to maintain a heightened TA excitation as they did with the brace, but statistical analysis showed that they were unable to do so. While some participants were capable of lowering SOL excitation during unbraced cycling, individual variation created a non-significant statistical result. One potential reason for the individual variation is the fitness level of participants. This study employed an absolute workload of 150W rather than a relative workload established based on individual criterion such as VO_2max; thus, some participants were working harder relative to their maximum capabilities. At higher workloads, the reciprocal inhibition on SOL by TA decreased; to meet the demands of an increased workload, SOL excitation increased and there was a gradual decline in reciprocal inhibition from TA (Pyndt, 2003). While there were no recorded measures of fitness in the present work, it was possible that more fit individuals were better able to reduce SOL excitation. Qualitatively, some participants commented that the workload felt more difficult than they might normally perform in self-designed workouts; conversely, other participants admitted that they were working well below the workload

of their normal cycling sessions. While all participants were able to complete the physical cycling task, those who were more physically fit were likely better able to concentrate on and use the feedback rather than simply focussing on completing the cycling task. Thus, the variation displayed in the muscle excitation data might be related to the range in fitness levels among participants.

6 CONCLUSION

The present study tested the immediate effects of reduction of SOL EMG on MG and LG excitation during cycling. It confirmed the results of Sanderson and Kenyon (2005) that during braced cycling, all three muscles respond similarly to the perturbation. It builds on this previous study by showing that SOL excitation was further reduced with biofeedback. The application of SOL EMG biofeedback led to a selective reduction in SOL and LG excitation while MG excitation was not significantly changed. These findings provided evidence to suggest that there are different motor neuron pools for the triceps surae muscles and that there may be some shared motor neurons creating overlapping pools. Even with biofeedback, individuals were unable to voluntarily eliminate SOL excitation which might be due to the use of the learned cycling skill. The effects of bracing and biofeedback were localized to the muscles acting at the ankle joint and thus do not lead to changes in superior muscles acting above the ankle at the knee or hip joints. Biofeedback was an effective tool to induce changes in SOL excitation in the short term and could help to induce a complete elimination of SOL muscle excitation in unbraced and braced cycling during a long term learning study. This project furthered our current knowledge of the interaction of muscles within the triceps surae complex and provided evidence that biofeedback can be employed for the modification of SOL excitation.

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8 APPENDICES

Appendix A: UBC Research Ethics Board Certificates of Approval

The University of British Columbia
Office of Research Services
Behavioural Research Ethics Board
Suite 102, 6190 Agronomy Road,
Vancouver, B.C. V6T 1Z3

CERTIFICATE OF APPROVAL - MINIMAL RISK

PRINCIPAL INVESTIGATOR:	INSTITUTION / DEPARTMENT:	UBC BREB NUMBER:
David J. Sanderson	UBC/Education/Human Kinetics	H07-02893

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):
Julia Marjorie Wilkes

SPONSORING AGENCIES:
N/A

PROJECT TITLE:
Use of EMG biofeedback to voluntarily reduce soleus excitation during cycling while the ankle is passively braced

CERTIFICATE EXPIRY DATE: February 20, 2009

DOCUMENTS INCLUDED IN THIS APPROVAL:		DATE APPROVED: February 20, 2008
Document Name	**Version**	**Date**
Consent Forms:		
Main Study Consent	2	February 13, 2008
Advertisements:		
Advertisement	2	February 13, 2008

The application for ethical review and the document(s) listed above have been reviewed and the procedures were found to be acceptable on ethical grounds for research involving human subjects.

Approval is issued on behalf of the Behavioural Research Ethics Board and signed electronically by one of the following:

Dr. M. Judith Lynam, Chair
Dr. Ken Craig, Chair
Dr. Jim Rupert, Associate Chair
Dr. Laurie Ford, Associate Chair
Dr. Daniel Salhani, Associate Chair
Dr. Anita Ho, Associate Chair

Appendix B: Individual Cadence Data

Average Cadence during 10 Pedal Cycles in Each Data Collection Period

Unbraced

Condition	Participant Number								
	5	6	7	8	14	15	16	17	18
NB-NF	80.94	80.94	83.57	81.91	80.43	82.27	81.62	79.26	81.15
NB-NF2	81.60	80.69	79.79	82.51	81.10	81.08	82.78	79.16	79.79
NB-F1	79.45	82.68	78.45	81.89	80.90	82.59	79.53	77.61	81.88
NB-F2	79.89	78.21	77.39	79.86	79.52	82.70	80.32	81.36	81.45
NB-F3	77.67	77.99	76.34	80.38	80.92	82.97	81.14	80.94	81.39
NB-F4	79.40	79.66	77.04	80.48	82.08	82.17	79.40	79.17	81.50
NB-F5	80.66	80.72	76.76	82.61	79.23	82.85	82.17	79.05	80.63
NB-F6	80.00	79.21	77.52	81.86	80.18	83.96	78.75	81.92	81.21
NB-F7	79.53	80.90	75.62	80.94	80.10	83.47	78.52	79.49	80.43
NB-F8	79.65	80.41	76.26	82.97	81.91	84.25	78.55	80.65	81.54
NB-F9	79.82	80.94	75.83	81.82	80.90	81.47	78.69	80.43	81.93
NB-F10	79.33	78.83	76.46	81.10	80.41	85.13	81.16	81.99	81.71
NB-F11	82.55	79.86	76.21	81.78	80.70	82.48	81.73	80.27	80.66
NB-F12	81.71	79.56	79.35	81.25	79.95	83.26	80.81	79.74	77.19
NB-F13	80.45	79.28	78.59	82.80	78.21	82.76	80.79	78.18	81.30
NB-F14	80.86	80.47	79.89	81.58	80.83	82.53	81.03	77.22	81.50
NB-F15	78.38	78.79	76.55	84.65	80.47	82.66	80.11	79.77	80.30
NB-F16	82.53	79.47	75.84	81.04	79.09	81.42	80.29	82.14	81.19
NB-F17	81.20	79.47	78.02	81.50	80.61	83.65	82.38	79.33	82.53
NB-F18	80.07	81.37	79.47	81.30	80.22	82.57	78.96	82.56	76.78
NB-F19	79.74	79.45	80.81	81.34	81.60	83.36	79.47	80.30	81.82
NB-F20	83.14	79.45	81.03	81.47	79.65	81.97	80.56	80.33	82.08

Braced

Condition	Participant Number								
	5	6	7	8	14	15	16	17	18
NB-NF	80.72	80.16	82.40	80.88	79.56	80.50	80.45	80.88	80.97
B-NF	81.30	78.21	82.74	81.10	79.88	78.69	81.03	78.66	81.01
B-F1	81.67	80.77	79.84	77.64	79.98	82.38	81.01	79.56	83.03
B-F2	80.92	83.45	80.09	79.77	79.74	78.35	80.65	79.84	80.77
B-F3	81.50	75.84	81.71	82.18	79.44	80.34	80.91	80.85	80.90
B-F4	80.94	80.81	80.71	82.25	79.89	79.95	80.00	83.20	82.31
B-F5	80.68	77.89	80.24	80.36	84.57	80.04	80.64	81.56	81.43
B-F6	80.58	78.86	82.01	82.30	81.03	77.11	80.47	78.26	82.06
B-F7	80.41	79.24	80.36	80.92	79.04	78.45	81.95	82.27	80.95
B-F8	80.95	78.88	78.08	82.10	80.16	80.05	82.12	82.49	81.69
B-F9	81.56	79.45	78.78	82.03	80.87	80.72	80.61	79.74	81.58
B-F10	80.72	80.20	80.11	82.17	81.16	82.66	82.96	81.61	81.25
B-F11	80.22	77.10	79.02	80.88	82.38	80.15	80.74	78.89	80.95
B-F12	80.29	80.90	74.17	81.61	80.02	78.93	81.26	81.99	81.86
B-F13	82.55	80.97	78.41	83.24	80.40	82.34	81.01	80.90	81.71
B-F14	81.38	78.64	79.91	82.80	77.92	80.92	80.75	83.10	81.41
B-F15	80.96	81.21	80.77	82.12	80.14	79.83	80.45	82.61	80.56
B-F16	83.05	83.90	78.85	82.25	80.13	79.79	81.56	78.19	83.14
B-F17	80.79	82.72	81.52	82.51	79.32	81.36	81.91	79.25	81.84
B-F18	80.36	81.19	79.86	82.48	79.59	79.83	80.99	83.24	82.10
B-F19	82.93	80.87	79.35	82.82	78.90	82.46	81.43	82.68	80.77
B-F20	82.01	78.15	76.78	83.05	77.79	82.34	81.36	80.56	80.68

Appendix C: Statistical Tests

Integrated EMG

7x4 Repeated Measures ANOVA

- Degrees of freedom, without Greenhouse-Geisser correction factor are:
  Muscle (6, 48) Condition (3, 24) Muscle x Condition (18, 144)

Unbraced

	F	df	MS	p	η_p^2
Muscle	5.62	1.190, 9.516	15.502	**0.036**	0.413
Condition	2.082	1.477, 11.818	2.192	0.173	0.207
Muscle x Condition	3.148	2.108, 16.862	11.538	0.067	0.282

Braced

	F	df	MS	p	η_p^2
Muscle	17.910	1.505, 12.038	35.858	**<0.001**	0.692
Condition	1.358	1.547, 12.373	0.938	0.285	0.145
Muscle x Condition	6.128	1.825, 14.604	17.060	**0.013**	0.434

Post-hoc Analysis

Note: Post hoc analyses were conducted only in the braced condition

Pairwise Comparisons between Muscles

- Bonferroni correction: $p \leq 0.017$

	MD	SE	Lower	Upper	p
SOL vs LG	-0.075	0.037	-0.159	0.009	0.075
SOL vs MG	-0.119	0.040	-0.211	-0.028	**0.017**
LG vs MG	-0.045	0.037	-0.131	0.042	0.267

1x4 Repeated Measures ANOVA

	F	df	MS	*p*	η_p^2
SOL	35.185	3, 24	0.621	**<0.001**	0.815
LG	15.485	3, 24	0.382	**<0.001**	0.659
MG	12.324	3, 24	0.221	**<0.001**	0.606
TA	5.165	1.445, 11.557	18.912	**0.033**	0.392
BF	2.810	1.456, 11.650	0.757	0.111	0.260
RF	0.569	2.570, 20.562	0.048	0.641	0.066
GM	10.446	1.542, 12.339	0.229	**0.003**	0.566

Pairwise Comparisons between Conditions

MD = Mean difference SE = Standard error
Lower = Lower bound of 95% confidence interval
Upper = Upper bound of 95% confidence interval

NB-NF vs B-NF

	MD	SE	Lower	Upper	*p*
SOL	0.310	0.060	0.103	0.518	**0.005**
LG	0.254	0.057	0.056	0.452	**0.012**
MG	0.324	0.049	0.153	0.495	**0.001**
TA	-0.508	0.180	-1.133	0.117	0.133
GM	-0.050	0.074	-0.308	0.207	1.000

NB-NF vs B-F1

	MD	SE	Lower	Upper	*p*
SOL	0.540	0.063	0.320	0.759	**<0.001**
LG	0.399	0.109	0.019	0.780	**0.039**
MG	0.304	0.090	-0.009	0.617	0.058
TA	-1.807	0.473	-3.453	-0.161	**0.031**
GM	0.114	0.056	-0.081	0.308	0.463

NB-NF vs B-F20

	MD	SE	Lower	Upper	*p*
SOL	0.567	0.066	0.337	0.797	**<0.001**
LG	0.465	0.083	0.176	0.753	**0.003**
MG	0.311	0.063	0.093	0.529	**0.007**
TA	-2.088	0.837	-4.999	0.824	0.223
GM	0.204	0.05	0.031	0.377	**0.021**

B-NF vs B-F1

	MD	SE	Lower	Upper	*p*
SOL	0.229	0.060	0.021	0.438	**0.030**
LG	0.145	0.069	-0.095	0.385	0.413
MG	-0.020	0.061	-0.234	0.193	1.000
TA	-1.299	0.362	-2.560	-0.038	**0.043**
GM	0.164	0.025	0.075	0.252	**0.001**

B-NF vs B-F20

	MD	SE	Lower	Upper	*p*
SOL	0.527	0.067	0.022	0.491	**0.031**
LG	0.210	0.053	0.026	0.394	**0.024**
MG	-0.013	0.046	-0.174	0.148	1.000
TA	-1.579	0.856	-4.556	1.397	0.613
GM	0.254	0.046	0.093	0.415	**0.003**

B-F1 vs B-F20

	MD	SE	Lower	Upper	*p*
SOL	0.270	0.059	-0.177	0.232	1.000
LG	0.065	0.057	-0.133	0.264	1.000
MG	0.007	0.06	-0.201	0.215	1.000
TA	-0.281	0.729	-2.815	2.254	1.000
GM	0.091	0.034	-0.027	0.208	0.167

Median Power Frequency

1x4 Repeated Measures Analysis of Variance

If df is not 3,24, the Greenhouse Geisser correction factor has been used.
Bold = $p < 0.05$

Unbraced

	F	df	MS	p	η_p^2
SOL	4.120	3,24	463.964	**0.017**	0.340
LG	3.123	3,24	384.467	**0.045**	0.281
MG	1.225	3,24	240.135	0.322	0.133
TA	0.857	1.531, 12.251	623.375	0.420	0.097
BF	3.428	1.859, 14.873	496.542	0.062	0.300
RF	1.606	3, 24	42.632	0.223	0.167
GM	1.622	3, 24	65.204	0.211	0.169

Braced

	F	df	MS	p	η_p^2
SOL	5.401	1.387, 11.100	3705.915	**0.032**	0.403
LG	4.856	2.344, 18.751	840.035	**0.009**	0.378
MG	1.485	1.391, 11.129	1971.10	0.260	0.157
TA	1.164	3, 24	43.992	0.344	0.127
BF	0.192	3, 24	14.980	0.901	0.023
RF	0.871	3, 24	73.289	0.470	0.098
GM	4.043	3, 24	216.560	**0.018**	0.336

Post Hoc Contrasts

Unbraced
SOL

	F	df	MS	p	η_p^2
NB-NF vs NB-NF2	4.993	1, 8	190.90	0.056	0.384
NB-NF2 vs NB-F1	12.116	1, 8	2284.776	**0.008**	0.602
NB-F1 vs NB-F20	8.686	1, 8	1834.123	0.521	0.019

LG

	F	df	MS	p	η_p^2
NB-NF vs NB-NF2	0.477	1, 8	33.216	0.509	0.056
NB-NF2 vs NB-F1	10.082	1, 8	1161.674	**0.013**	0.558
NB-F1 vs NB-F20	4.550	1, 8	1763.720	0.065	0.363

Braced – only the significant results are shown

SOL

	F	df	MS	p	η_p^2
B-NF vs B-F20	7.336	1, 8	9150.389	**0.027**	0.478

LG

	F	df	MS	p	η_p^2
NB-NF vs B-F20	12.639	1, 8	4494.540	**0.007**	0.612

GM

	F	df	MS	p	η_p^2
NB-NF vs B-F20	9.596	1, 8	1067.024	**0.015**	0.545

Repeatability

NB-NF vs NB-NF2 with Paired Samples T-test

	t	df	p	Cohen's d
SOL	-0.108	8	0.916	0.809
LG	0.287	8	0.781	1.257
MG	-0.997	8	0.348	0.427
TA	1.366	8	0.209	1.682
BF	-1.032	8	0.332	3.921
RF	2.541	8	**0.035**	18.586
GM	-0.686	8	0.512	0.779

Time-to-peak EMG

1x4 Repeated Measures Analysis of Variance

If df is not 3,24, the Greenhouse Geisser correction factor has been used.

Bold = $p < 0.05$

Unbraced

	F	df	MS	p	η_p^2
LG	0.097	3, 24	3.395	0.961	0.012
MG	1.913	1.169, 9.355	25.533	0.154	0.193

Braced

	F	df	MS	p	η_p^2
LG	6.076	3, 24	198.258	**0.003**	0.432
MG	4.931	1.218, 9.745	93.205	**0.046**	0.381

Post Hoc Contrasts

Braced
LG

	F	df	MS	p	η_p^2
NB-NF vs B-NF	3.638	1, 8	242.581	0.093	0.313
NB-NF vs B-F1	9.163	1, 8	869.090	**0.016**	0.534
NB-NF vs B-F20	9.223	1, 8	876.969	**0.016**	0.536
B-NF vs B-F1	7.154	1, 8	193.358	**0.028**	0.472
B-F1 vs B-F20	0.000	1, 8	0.018	0.988	0.000

MG

	F	df	MS	p	η_p^2
NB-NF vs B-NF	10.875	1, 8	25.003	**0.011**	0.576
NB-NF vs B-F1	2.834	1, 8	70.616	0.131	0.262
NB-NF vs B-F20	15.891	1, 8	214.710	**0.004**	0.665
B-NF vs B-F1	0.395	1, 8	11.580	0.547	0.047
B-F1 vs B-F20	9.427	1, 8	39.058	**0.015**	0.541

Appendix D: Individual Participant Data

Integrated EMG

SOL

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	1.00	0.98	0.59	0.58
6	1.00	1.06	0.93	0.93
7	1.00	1.39	1.43	1.06
8	1.00	1.07	0.44	0.47
14	1.00	0.91	0.99	0.89
15	1.00	0.97	1.34	0.77
16	1.00	0.77	0.65	0.43
17	1.00	0.99	1.14	1.13
18	1.00	1.02	0.86	1.11

Braced

	NB-NF	B-NF	B-F1	B-F20
5	1.00	0.77	0.30	0.33
6	1.00	0.59	0.62	0.47
7	1.00	0.86	0.70	0.43
8	1.00	0.78	0.26	0.19
14	1.00	0.88	0.53	0.86
15	1.00	0.51	0.33	0.26
16	1.00	0.48	0.36	0.49
17	1.00	0.89	0.74	0.56
18	1.00	0.46	0.30	0.31

LG

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	1.00	1.15	0.77	1.18
6	1.00	1.13	0.85	1.08
7	1.00	1.05	0.80	0.74
8	1.00	1.05	0.56	0.86
14	1.00	1.13	1.37	1.27
15	1.00	0.99	1.12	0.89
16	1.00	1.03	0.82	1.49
17	1.00	1.03	0.94	0.88
18	1.00	1.02	0.96	1.11

Braced

	NB-NF	B-NF	B-F1	B-F20
5	1.00	0.69	0.53	0.54
6	1.00	0.69	0.48	0.67
7	1.00	0.84	0.79	0.54
8	1.00	0.70	0.24	0.30
14	1.00	1.07	1.33	1.11
15	1.00	0.68	0.67	0.50
16	1.00	0.73	0.35	0.48
17	1.00	0.87	0.70	0.43
18	1.00	0.44	0.33	0.25

MG

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	1.00	1.04	0.97	1.04
6	1.00	1.09	1.17	1.03
7	1.00	0.97	1.13	1.07
8	1.00	1.01	0.77	0.90
14	1.00	0.97	0.95	1.08
15	1.00	1.02	1.24	0.94
16	1.00	1.05	1.03	1.11
17	1.00	0.97	0.97	1.00
18	1.00	1.01	1.05	1.07

Braced

	NB-NF	B-NF	B-F1	B-F20
5	1.00	0.68	0.53	0.63
6	1.00	0.61	0.48	0.73
7	1.00	1.00	1.00	0.85
8	1.00	0.61	0.57	0.84
14	1.00	0.75	1.21	0.95
15	1.00	0.76	0.87	0.75
16	1.00	0.53	0.44	0.41
17	1.00	0.62	0.67	0.61
18	1.00	0.52	0.49	0.42

TA

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	1.00	0.78	1.85	0.93
6	1.00	1.10	3.89	0.88
7	1.00	0.77	4.49	5.98
8	1.00	1.15	2.05	6.05
14	1.00	0.86	2.24	1.76
15	1.00	0.65	9.23	1.50
16	1.00	1.04	0.60	0.26
17	1.00	0.84	1.05	0.94
18	1.00	0.91	1.29	1.41

Braced

	NB-NF	B-NF	B-F1	B-F20
5	1.00	1.33	1.29	1.46
6	1.00	2.52	5.07	3.07
7	1.00	1.21	3.64	9.06
8	1.00	1.39	3.75	2.54
14	1.00	1.87	3.73	3.59
15	1.00	1.78	3.21	2.47
16	1.00	0.74	0.63	0.17
17	1.00	0.96	2.00	3.79
18	1.00	1.79	1.94	1.63

BF

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	1.00	1.17	0.63	1.64
6	1.00	1.21	1.59	1.91
7	1.00	0.94	0.97	1.25
8	1.00	0.85	0.72	0.86
14	1.00	0.94	0.76	0.75
15	1.00	1.05	1.21	0.93
16	1.00	1.09	1.50	1.48
17	1.00	1.00	0.82	0.94
18	1.00	1.16	0.98	0.84

Braced

	NB-NF	B-NF	B-F1	B-F20
5	1.00	0.77	1.05	1.89
6	1.00	1.31	2.30	2.77
7	1.00	1.15	1.43	2.02
8	1.00	1.10	0.63	0.79
14	1.00	1.23	1.67	1.41
15	1.00	1.00	1.43	1.13
16	1.00	1.14	1.18	0.77
17	1.00	0.93	0.87	0.64
18	1.00	1.31	1.51	1.49

RF

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	1.00	0.94	1.56	0.80
6	1.00	0.84	0.89	0.85
7	1.00	1.07	1.27	1.18
8	1.00	0.86	0.81	1.01
14	1.00	0.95	0.91	0.88
15	1.00	0.78	1.17	0.79
16	1.00	0.93	1.23	2.02
17	1.00	0.96	1.19	0.92
18	1.00	0.95	0.77	0.91

Braced

	NB-NF	B-NF	B-F1	B-F20
5	1.00	1.22	1.46	1.97
6	1.00	1.16	1.02	0.53
7	1.00	1.31	1.34	1.27
8	1.00	0.74	1.93	0.97
14	1.00	0.70	0.52	0.77
15	1.00	1.13	1.15	1.08
16	1.00	0.68	1.08	0.71
17	1.00	0.88	0.75	0.56
18	1.00	1.56	1.15	1.16

GM

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	1.00	0.91	0.91	0.97
6	1.00	1.01	0.78	0.78
7	1.00	0.86	1.06	0.68
8	1.00	1.10	0.94	0.68
14	1.00	0.95	0.82	0.84
15	1.00	1.16	0.91	0.69
16	1.00	0.93	0.81	0.94
17	1.00	1.10	1.04	0.93
18	1.00	1.41	1.02	1.03

Braced

	NB-NF	B-NF	B-F1	B-F20
5	1.00	0.98	0.79	0.80
6	1.00	1.25	1.03	0.93
7	1.00	1.21	1.10	1.04
8	1.00	1.16	0.90	0.92
14	1.00	1.21	1.00	0.72
15	1.00	1.01	0.84	0.74
16	1.00	0.54	0.53	0.54
17	1.00	0.94	0.85	0.71
18	1.00	1.16	0.96	0.78

Time-to-peak EMG

SOL

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	20.18	19.03	15.80	17.51
6	14.17	11.11	18.65	15.55
7	18.05	19.49	16.48	14.97
8	20.77	20.21	17.22	27.58
14	27.15	25.87	22.49	23.90
15	18.33	16.92	15.63	18.85
16	26.15	24.60	25.52	24.93
17	22.62	24.23	25.32	23.99
18	19.35	19.44	18.51	21.12

Braced

	NB-NF	B-NF	B-F1	B-F20
5	20.36	22.67	23.75	24.01
6	19.75	21.68	32.23	29.91
7	18.30	19.49	21.23	36.95
8	18.18	25.37	26.19	36.30
14	26.76	27.04	26.09	25.85
15	17.61	20.13	23.49	19.50
16	27.21	24.21	27.63	47.10
17	20.09	25.43	24.51	24.63
18	21.01	28.22	25.68	24.91

LG

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	54.43	47.26	53.12	42.93
6	24.55	24.83	29.77	44.37
7	40.94	37.61	21.56	24.76
8	50.21	48.07	51.21	39.73
14	46.11	45.85	43.31	41.74
15	53.25	57.91	51.91	51.24
16	39.18	40.42	47.78	50.36
17	26.01	30.80	29.17	24.93
18	20.88	22.27	21.79	23.68

Braced

	NB-NF	B-NF	B-F1	B-F20
5	35.62	56.72	58.55	59.45
6	27.38	37.45	53.10	35.27
7	29.47	41.59	43.32	55.33
8	47.95	48.28	57.51	55.02
14	48.94	51.51	57.73	50.96
15	54.23	49.83	53.52	54.33
16	34.49	35.30	39.80	48.69
17	35.06	32.25	31.69	33.88
18	26.37	33.31	32.75	35.43

MG

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	27.10	26.28	28.41	34.14
6	31.01	31.60	30.73	32.18
7	30.31	31.18	29.54	27.18
8	24.42	26.05	26.97	34.28
14	35.62	35.92	35.99	38.09
15	34.19	34.31	31.10	33.05
16	32.89	31.96	31.44	35.67
17	35.17	35.43	30.07	30.59
18	29.64	29.60	27.32	29.36

Braced

	NB-NF	B-NF	B-F1	B-F20
5	28.07	29.43	31.82	34.60
6	31.61	31.48	29.06	32.04
7	29.15	30.18	30.00	35.08
8	27.34	32.17	31.08	32.48
14	37.14	37.24	51.96	50.50
15	32.73	35.44	34.48	35.33
16	35.46	37.68	33.92	38.62
17	30.23	32.27	32.51	33.61
18	32.41	33.24	34.53	35.85

TA

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	85.43	87.03	79.38	69.54
6	86.82	81.45	84.61	81.76
7	63.62	72.86	65.50	78.33
8	80.41	85.75	85.38	93.30
14	86.00	86.76	67.96	87.22
15	58.81	41.13	65.59	55.44
16	86.24	83.56	76.85	34.10
17	36.08	40.13	53.99	35.68
18	85.91	81.03	83.90	85.57

Braced

	NB-NF	B-NF	B-F1	B-F20
5	85.85	82.42	65.07	38.47
6	87.65	68.91	32.88	68.67
7	74.16	69.10	57.82	63.44
8	85.54	85.71	49.72	39.70
14	88.56	81.67	63.42	65.57
15	63.92	63.36	50.25	27.94
16	83.60	76.75	74.40	75.67
17	46.23	4.28	36.51	44.52
18	84.28	59.01	54.37	50.20

BF

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	32.53	30.09	31.35	29.67
6	37.91	36.55	37.23	37.68
7	33.81	32.28	30.68	29.10
8	21.78	16.99	42.85	33.51
14	36.72	40.70	59.12	45.66
15	18.99	24.13	27.63	26.81
16	9.52	37.02	29.25	35.38
17	36.36	35.43	29.80	29.55
18	32.66	27.02	33.37	36.21

Braced

	NB-NF	B-NF	B-F1	B-F20
5	30.91	32.08	37.77	33.09
6	36.09	33.92	36.09	40.50
7	39.61	41.77	37.37	37.66
8	25.23	19.01	14.39	27.69
14	48.45	36.26	58.89	33.02
15	28.57	29.30	26.94	28.08
16	28.61	20.16	27.38	24.56
17	28.24	26.60	28.67	29.10
18	8.15	13.18	17.88	15.36

RF

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	9.64	11.77	69.18	75.84
6	21.96	22.91	56.90	62.07
7	16.40	30.58	58.53	74.67
8	23.03	14.41	21.62	34.73
14	26.70	17.87	26.08	47.95
15	14.21	33.58	72.97	77.62
16	73.94	80.14	79.18	80.94
17	8.76	8.31	24.34	8.02
18	9.12	30.00	18.35	27.85

Braced

	NB-NF	B-NF	B-F1	B-F20
5	15.99	15.66	69.73	81.37
6	47.63	30.55	69.35	41.64
7	47.79	25.28	80.36	46.09
8	59.96	15.67	55.33	52.89
14	13.59	16.88	15.87	15.61
15	35.35	22.85	68.97	69.43
16	80.36	83.50	80.87	77.48
17	10.14	6.62	7.69	20.05
18	32.88	10.23	51.62	17.11

GM

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	17.83	14.72	15.62	11.54
6	13.29	13.45	8.85	21.49
7	16.64	13.80	15.80	13.45
8	18.31	17.70	19.00	16.58
14	19.55	19.71	32.97	27.91
15	14.44	17.46	18.92	16.93
16	18.18	16.12	15.34	17.44
17	18.83	19.56	18.41	19.14
18	15.78	21.97	18.58	11.85

Braced

	NB-NF	B-NF	B-F1	B-F20
5	16.60	19.30	15.25	11.35
6	12.30	13.92	10.38	10.81
7	19.61	15.65	14.34	18.37
8	18.47	18.78	18.94	18.68
14	40.18	16.01	18.18	17.61
15	17.29	17.50	18.18	14.94
16	17.46	48.09	48.57	60.88
17	18.33	16.38	16.96	13.97
18	9.10	16.58	12.19	12.24

Median Power Frequency

SOL

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	101.51	103.56	105.03	113.53
6	99.32	102.39	82.76	113.53
7	105.47	109.13	95.65	98.44
8	122.46	137.40	105.47	92.43
14	145.17	137.26	100.63	124.66
15	105.76	113.96	91.99	125.98
16	93.60	98.44	100.49	112.06
17	100.63	109.57	90.67	108.25
18	78.37	82.03	77.64	89.94

Braced

	NB-NF	B-NF	B-F1	B-F20
5	143.85	133.15	129.64	122.17
6	102.25	107.23	98.88	88.77
7	108.25	121.44	114.11	61.82
8	131.98	151.03	116.02	53.61
14	141.36	145.31	113.96	138.13
15	113.67	136.96	128.76	128.17
16	87.16	85.11	76.17	13.77
17	97.71	103.42	100.05	84.23
18	76.17	77.05	86.72	83.06

LG

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	138.13	133.01	129.20	157.62
6	157.03	159.81	130.22	179.00
7	158.50	145.75	120.56	134.03
8	139.89	147.95	132.13	108.11
14	126.71	118.36	111.18	116.31
15	121.00	115.43	113.67	135.79
16	125.98	136.82	122.31	138.43
17	119.09	108.98	110.89	116.02
18	106.79	109.72	103.42	114.26

Braced

	NB-NF	B-NF	B-F1	B-F20
5	136.52	130.37	136.96	111.91
6	143.85	115.28	105.18	111.77
7	147.07	178.27	177.10	146.63
8	177.10	162.30	127.00	119.97
14	125.83	111.77	94.78	108.40
15	131.69	128.91	117.33	111.47
16	118.36	86.57	97.27	96.24
17	107.08	102.10	101.07	72.66
18	131.54	134.03	123.05	138.87

MG

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	123.63	128.47	134.33	159.67
6	115.58	131.10	103.86	124.37
7	135.21	129.79	102.39	115.87
8	188.96	185.30	151.46	137.70
14	164.21	155.57	145.02	163.62
15	107.81	90.53	84.23	104.74
16	91.41	89.21	108.69	109.86
17	107.81	110.74	107.81	109.13
18	161.87	138.43	161.87	174.61

Braced

	NB-NF	B-NF	B-F1	B-F20
5	140.19	158.35	158.94	161.13
6	129.93	112.65	103.86	116.60
7	141.50	165.97	150.59	149.56
8	176.51	166.55	174.02	36.18
14	157.32	142.97	149.56	151.90
15	71.48	106.05	91.11	93.46
16	102.69	85.69	77.64	79.25
17	112.21	106.05	76.47	76.03
18	156.15	169.34	144.73	145.90

TA

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	123.63	115.87	128.17	126.12
6	69.87	64.75	62.99	60.50
7	56.25	51.27	72.51	85.99
8	88.48	86.28	112.21	126.42
14	107.08	104.15	115.28	98.15
15	78.81	87.74	133.59	28.86
16	114.84	112.35	95.51	60.65
17	53.47	50.54	50.83	63.43
18	112.06	109.86	121.44	127.00

Braced

	NB-NF	B-NF	B-F1	B-F20
5	51.86	85.99	73.10	71.63
6	60.50	70.90	58.74	52.59
7	53.91	48.05	53.91	58.74
8	103.56	100.78	107.81	104.15
14	100.93	106.79	108.25	104.59
15	86.28	81.01	84.52	83.35
16	80.27	80.86	89.65	84.81
17	56.84	58.15	62.26	65.19
18	93.60	92.14	93.02	100.34

BF

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	76.90	80.71	69.87	89.50
6	90.82	96.39	100.20	102.10
7	79.69	86.43	77.93	94.63
8	82.18	85.25	47.31	57.42
14	81.01	75.29	52.30	71.63
15	66.06	71.34	68.56	82.03
16	81.30	80.71	62.40	61.23
17	87.89	92.58	66.94	90.38
18	74.71	69.29	74.12	57.57

Braced

	NB-NF	B-NF	B-F1	B-F20
5	81.01	61.08	69.58	78.81
6	104.59	105.18	99.61	95.95
7	56.84	65.77	68.99	72.80
8	89.21	80.57	70.75	59.18
14	61.52	62.55	64.75	78.81
15	66.65	60.79	70.02	69.43
16	73.83	67.24	73.24	66.36
17	81.01	106.49	111.62	90.09
18	78.22	69.14	78.52	78.96

RF

Unbraced

	NB-NF	NB-NF2	NB-F1	NB-F20
5	59.18	63.14	64.75	71.19
6	71.05	58.45	66.50	66.36
7	57.42	67.53	65.48	75.73
8	61.52	62.99	76.32	67.82
14	63.14	73.68	60.21	72.07
15	60.06	59.47	58.74	63.43
16	48.05	60.35	54.05	57.86
17	55.23	61.67	54.05	56.10
18	63.14	61.08	57.42	54.93

Braced

	NB-NF	B-NF	B-F1	B-F20
5	60.79	65.48	81.89	78.52
6	67.82	69.14	69.14	50.98
7	76.76	80.13	76.61	83.79
8	67.68	64.31	79.25	82.18
14	61.38	53.76	49.95	56.40
15	37.79	62.55	61.08	68.70
16	65.92	64.60	68.99	58.15
17	62.99	85.11	68.26	47.31
18	63.87	64.16	69.58	65.04

GM

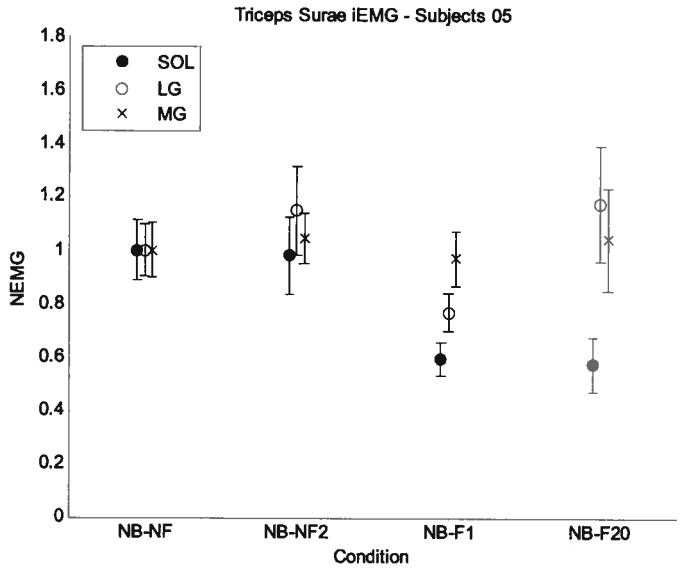
Unbraced

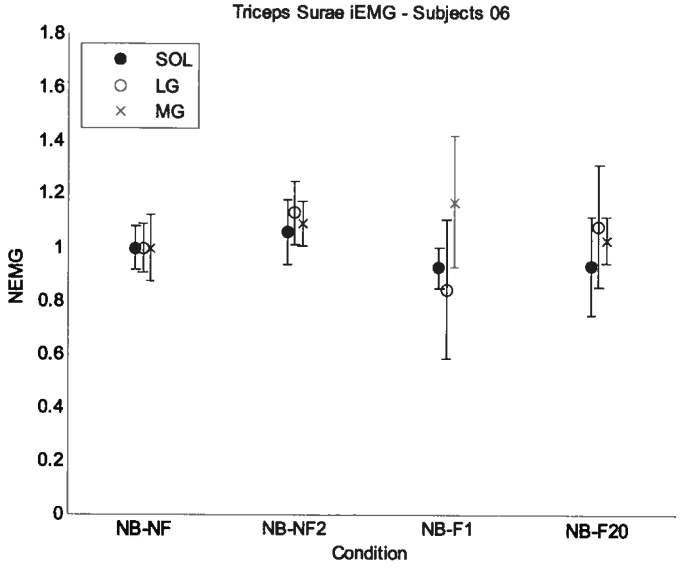
	NB-NF	NB-NF2	NB-F1	NB-F20
5	30.47	36.48	32.23	32.37
6	37.06	43.21	39.84	42.77
7	53.47	48.78	30.76	68.41
8	44.82	46.00	48.93	45.70
14	50.83	53.03	58.59	60.06
15	49.51	44.09	45.41	50.98
16	42.04	35.30	41.31	42.92
17	38.82	38.38	41.90	40.58
18	62.55	44.24	42.48	52.00

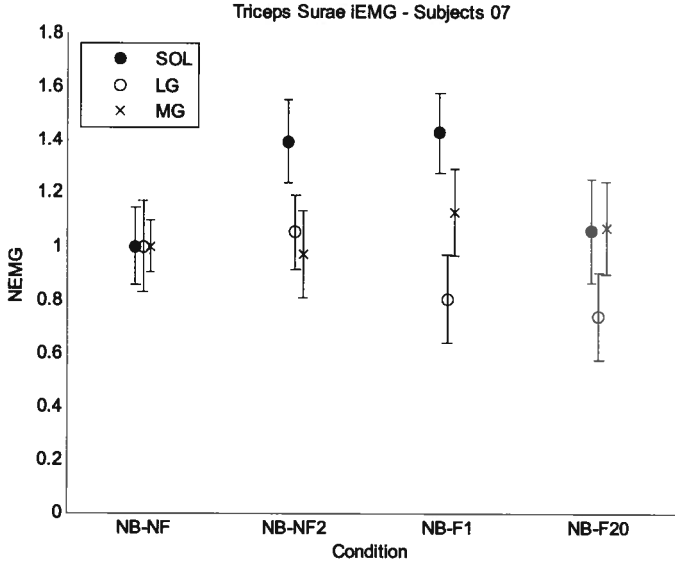
Braced

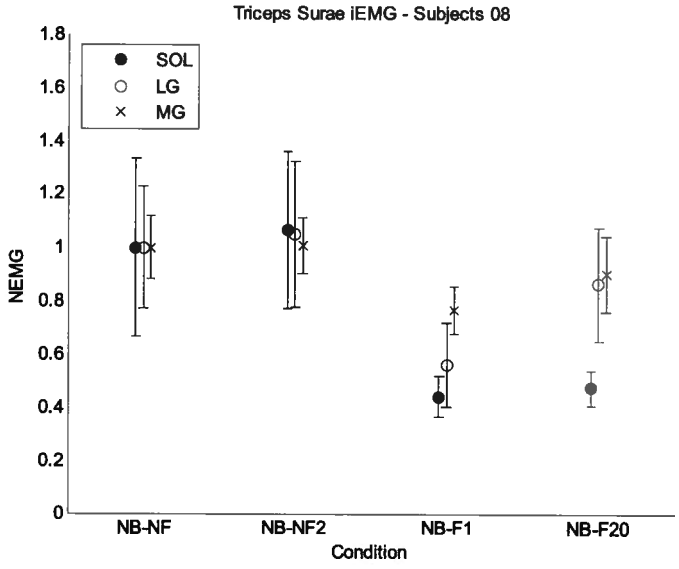
	NB-NF	B-NF	B-F1	B-F20
5	36.48	39.11	50.68	44.53
6	53.32	47.90	56.25	59.47
7	50.83	44.68	55.96	60.06
8	42.92	44.68	51.12	42.48
14	51.12	39.99	39.55	61.23
15	44.68	47.61	46.00	46.00
16	38.97	59.91	59.91	60.06
17	41.60	42.77	39.99	75.44
18	43.80	47.17	47.31	52.44

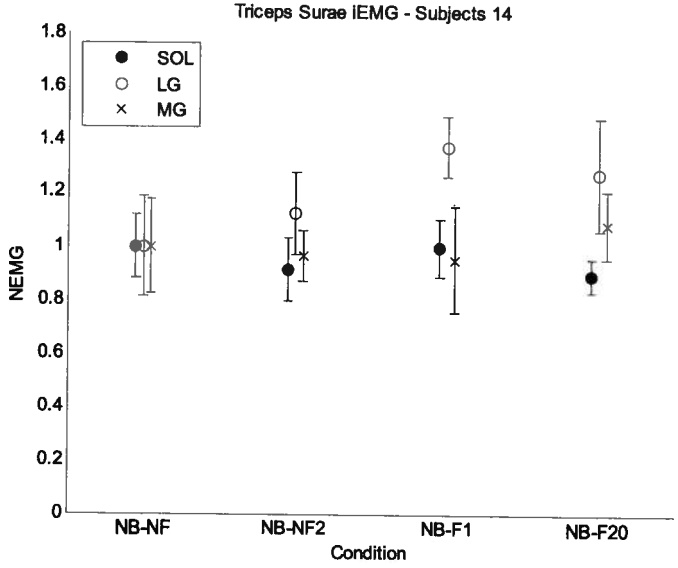
UNBRACED CONDITION







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MG
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NB-F1
NB-F20
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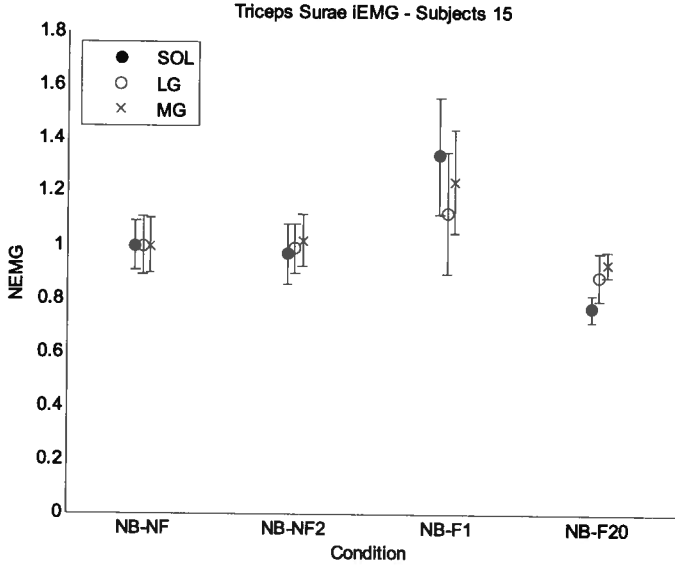
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MG
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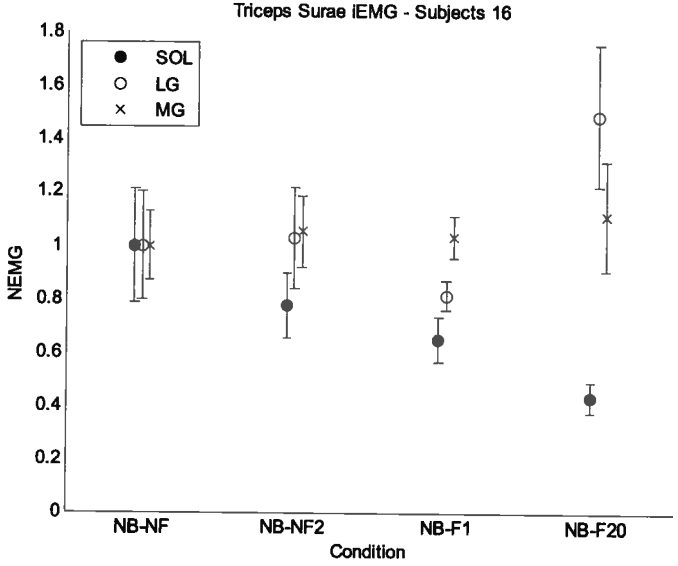
Condition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LG
MG
NEMG
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0.2
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NB-NF
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NB-F1
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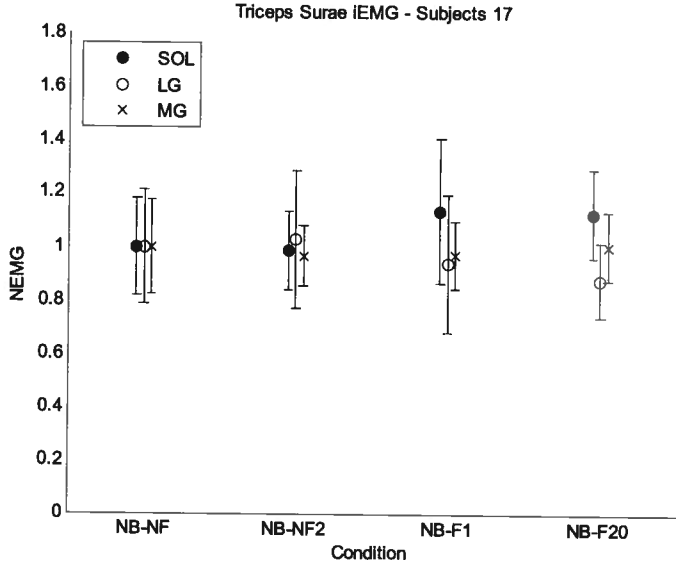
Condition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MG
NEMG
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1.6
1.4
1.2
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0.8
0.6
0.4
0.2
0
NB-NF
NB-NF2
NB-F1
NB-F20
Condition

Triceps Surae iEMG - Subjects 18
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LG
MG
NEMG
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1.6
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0.4
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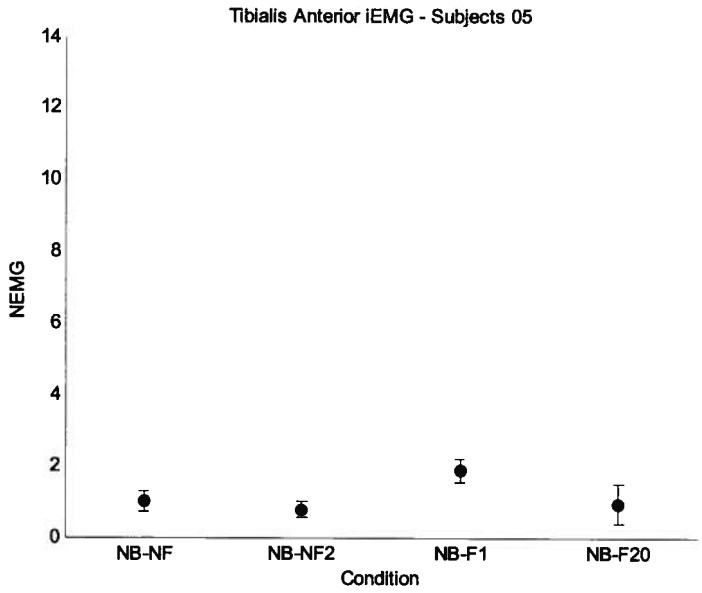
NB-NF2
NB-F1
NB-F20
Condition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6
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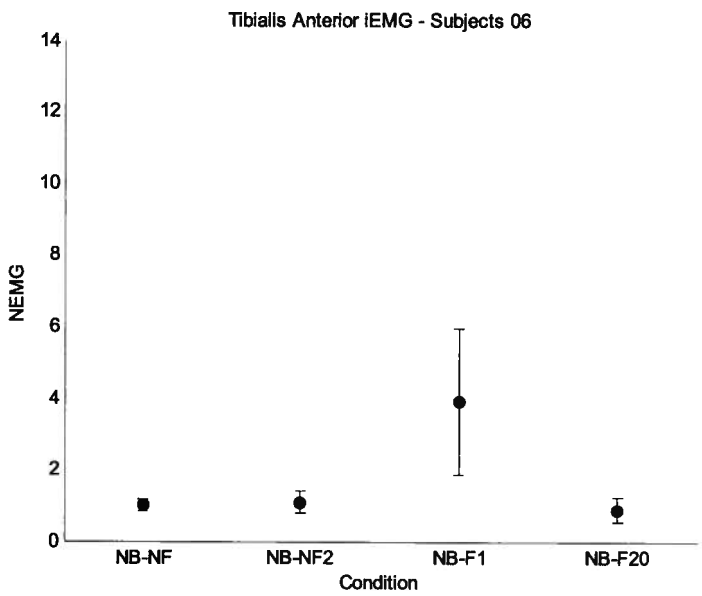
NB-NF
NB-NF2
NB-F1
NB-F20
Condition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NB-F1
NB-F20
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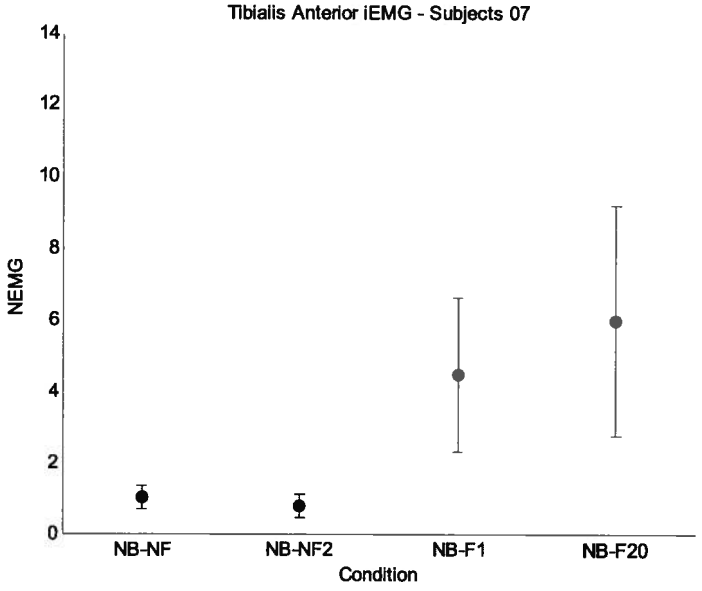
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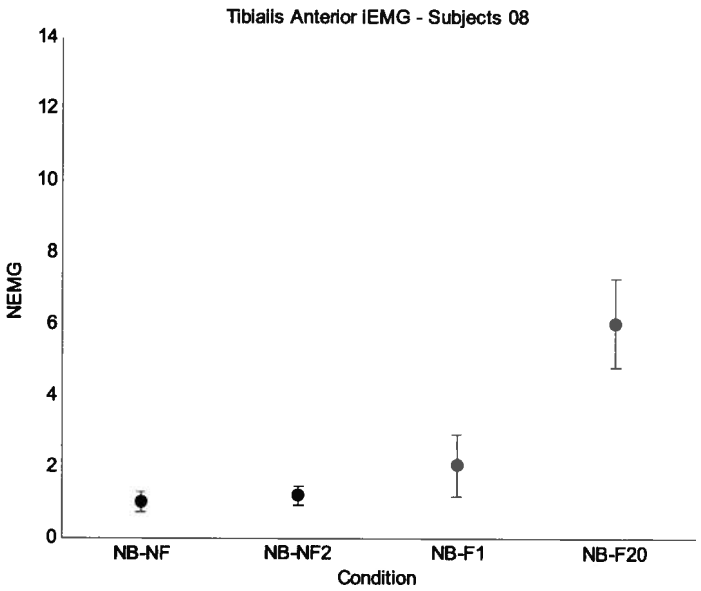
NB-NF2
NB-F1
NB-F20
Condition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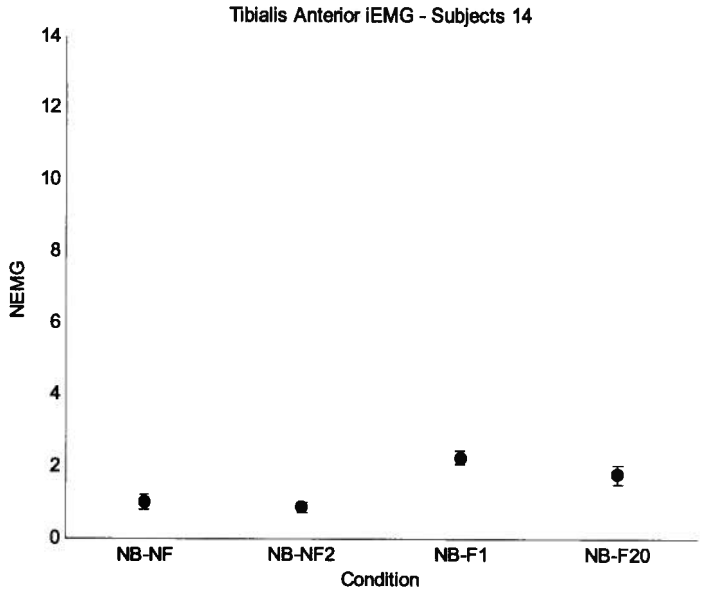
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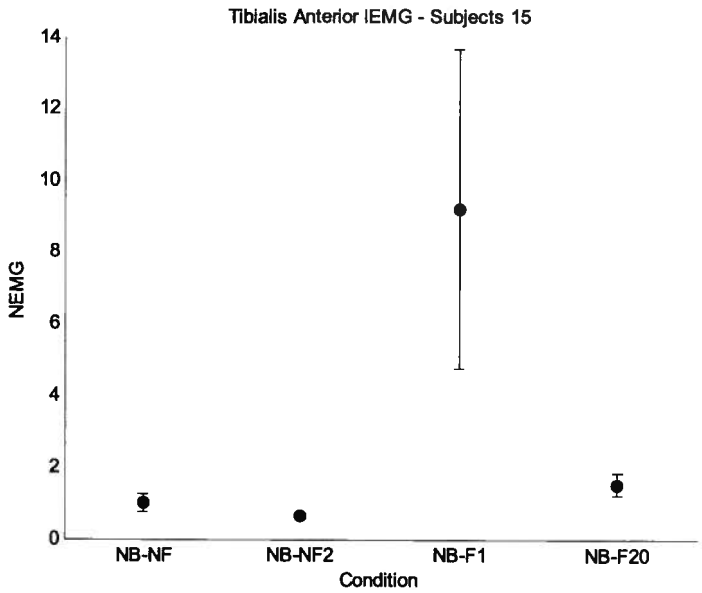
Condition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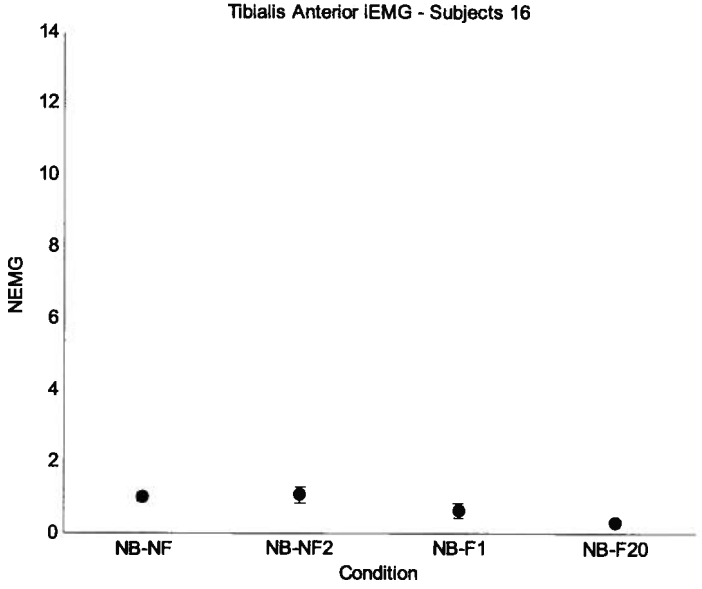
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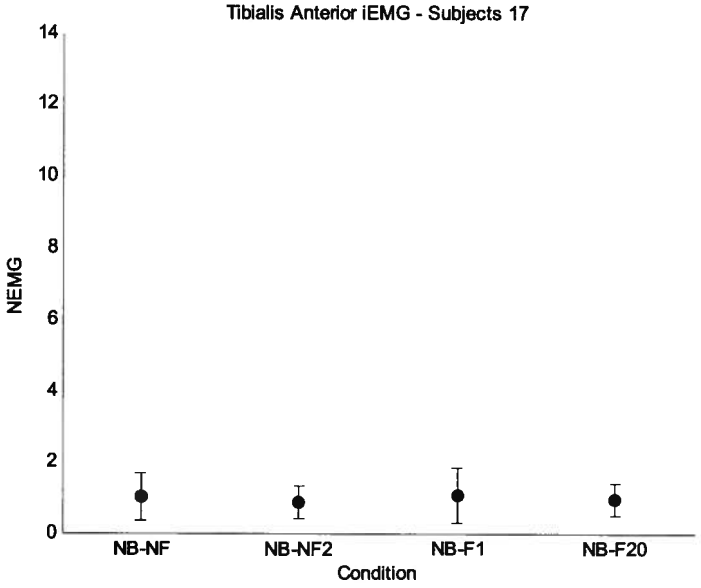
NB-F1
NB-F20
Condition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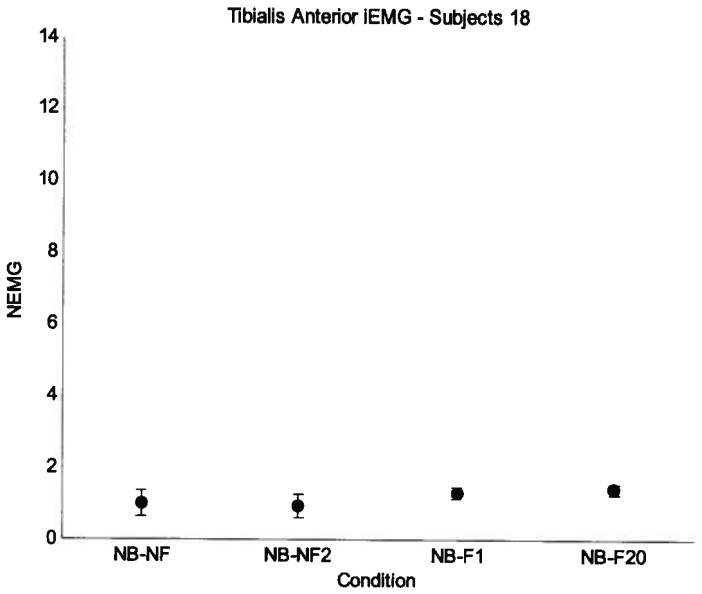
NB-F1
NB-F20
Condition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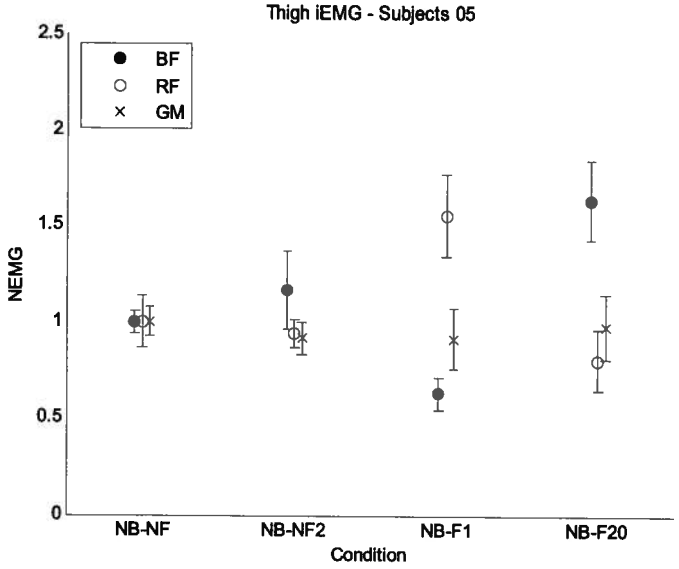
NB-NF2
NB-F1
NB-F20
Condition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RF
GM
NEMG
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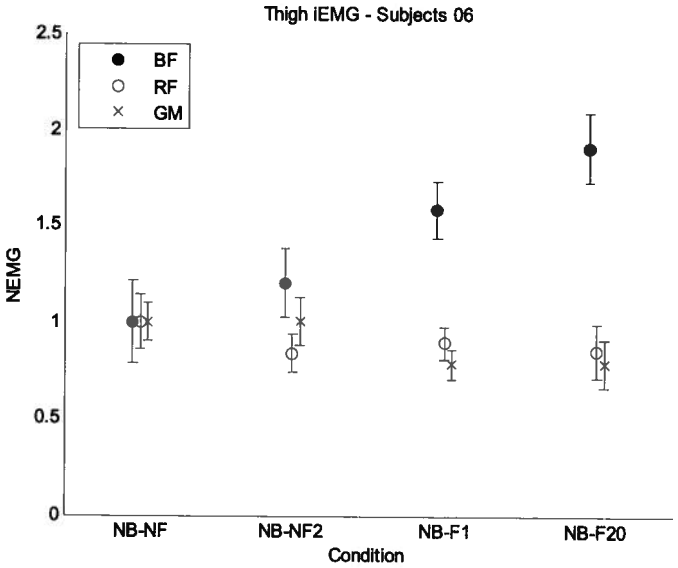
NB-NF
NB-NF2
NB-F1
NB-F20
Condition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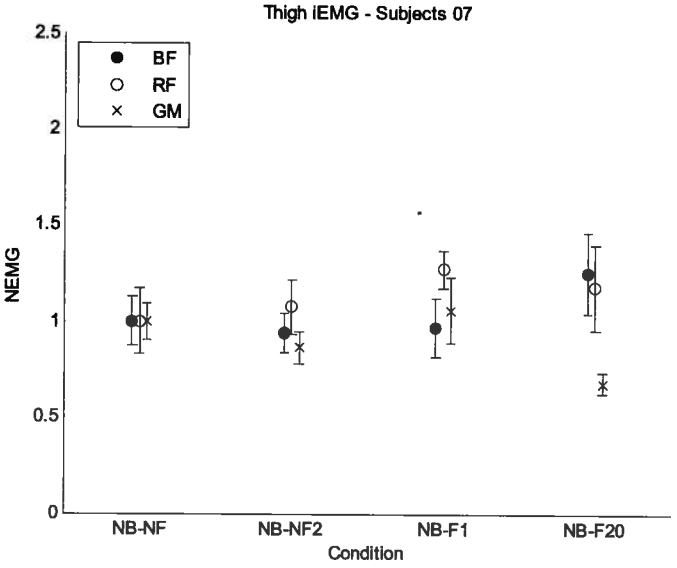
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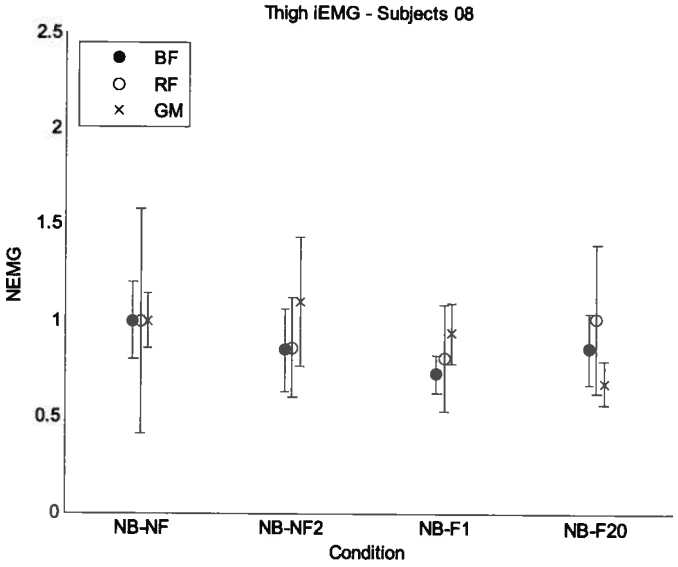
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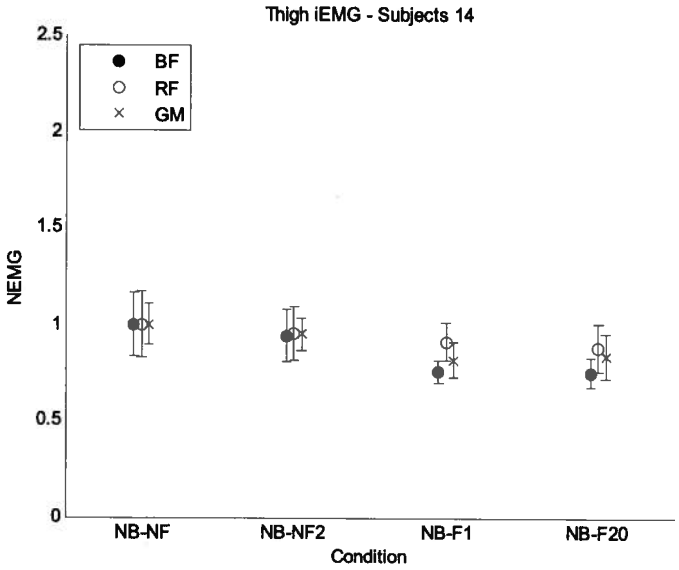
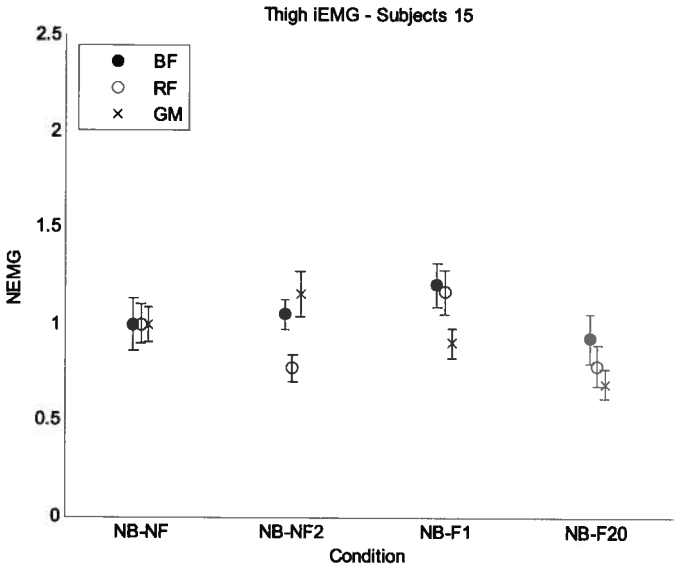
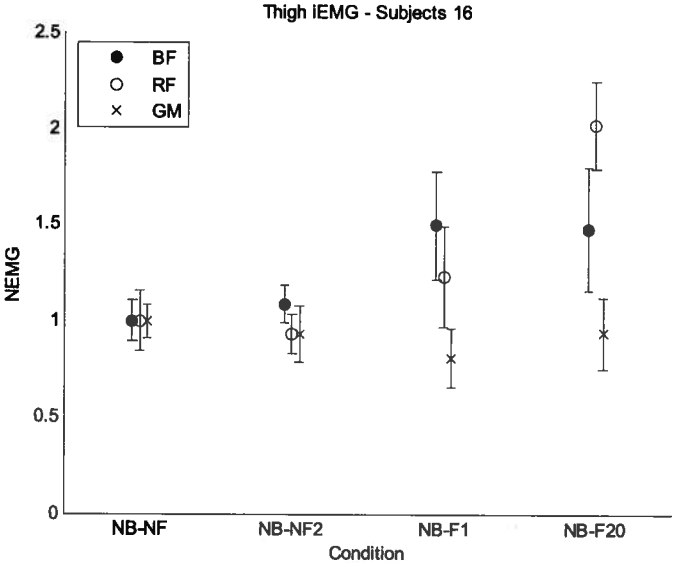
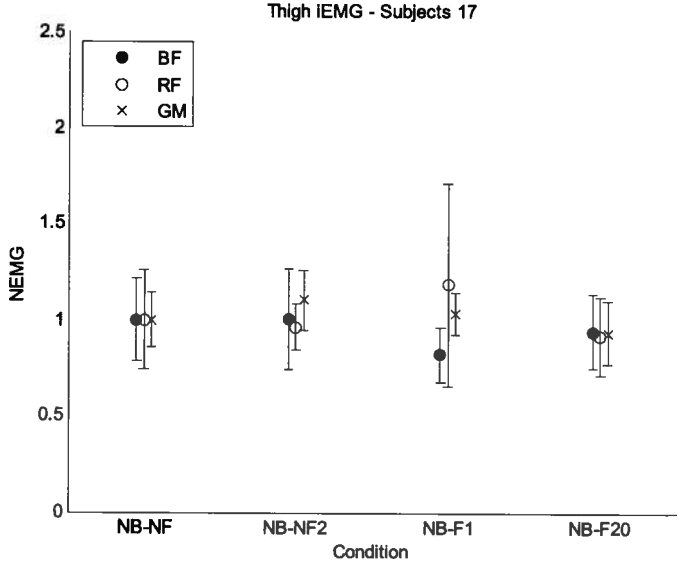
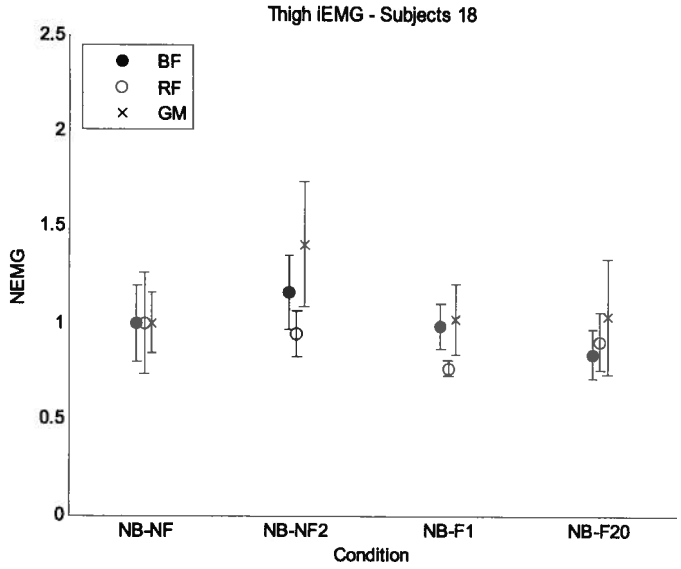
NEMG
2.5
2
1.5
1
0.5
0
NB-NF
NB-NF2
NB-F1
NB-F20
Condition


Thigh iEMG - Subjects 14
BF
RF
GM
NEMG
2.5
2
1.5
1
0.5
0
NB-NF
NB-NF2
NB-F1
NB-F20
Condition

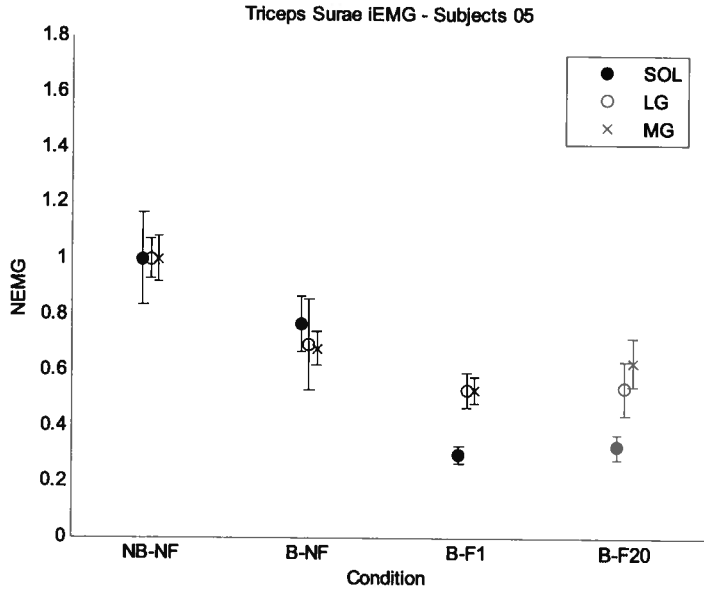

Thigh iEMG - Subjects 15
BF
RF
GM
NEMG
2.5
2
1.5
1
0.5
0
NB-NF
NB-NF2
NB-F1
NB-F20
Condition

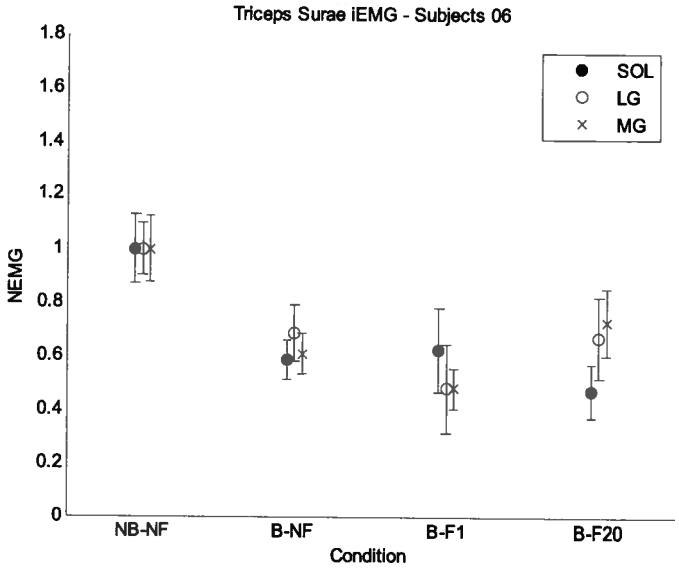

Thigh iEMG - Subjects 16
BF
RF
GM
NEMG
2.5
2
1.5
1
0.5
0
NB-NF
NB-NF2
NB-F1
NB-F20
Condition

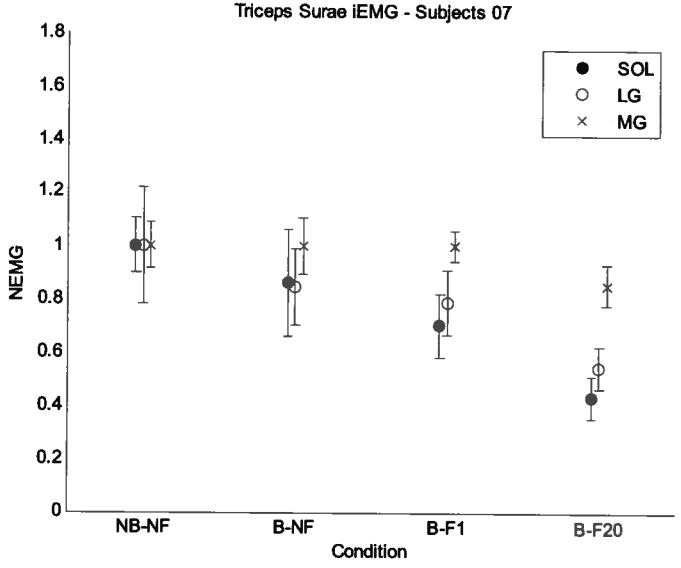

Thigh iEMG - Subjects 17
BF
RF
GM
NEMG
2.5
2
1.5
1
0.5
0
NB-NF
NB-NF2
NB-F1
NB-F20
Condition

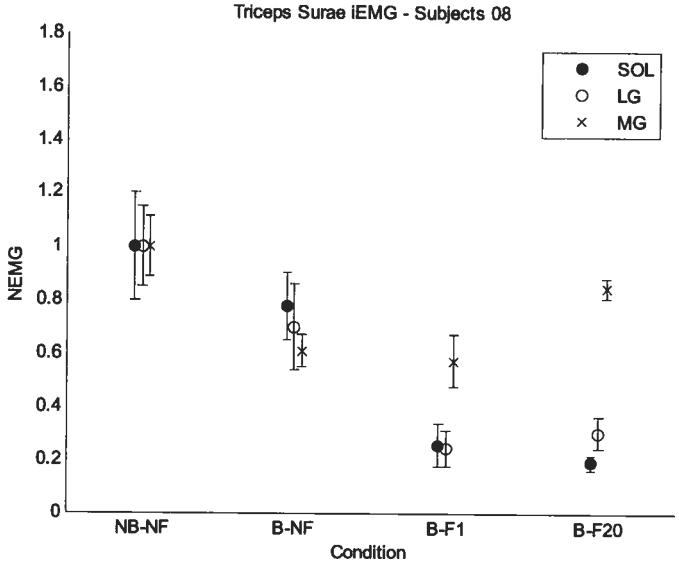

Thigh iEMG - Subjects 18
BF
RF
GM
NEMG
2.5
2
1.5
1
0.5
0
NB-NF
NB-NF2
NB-F1
NB-F20
Condition

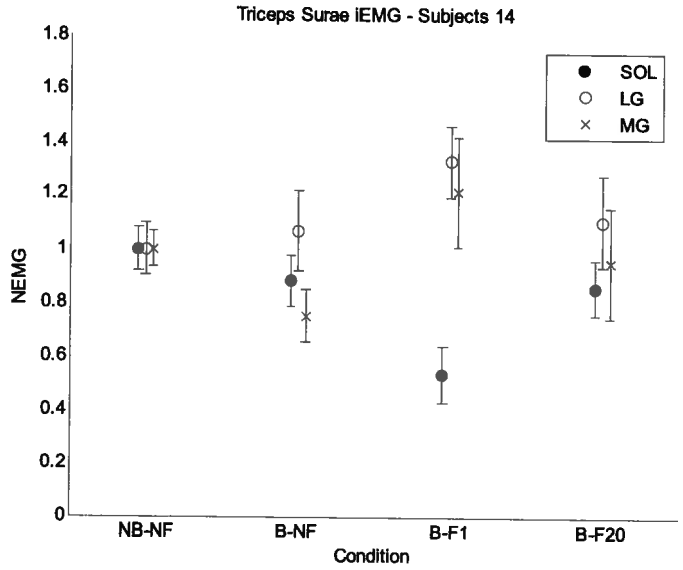
BRACED CONDITION

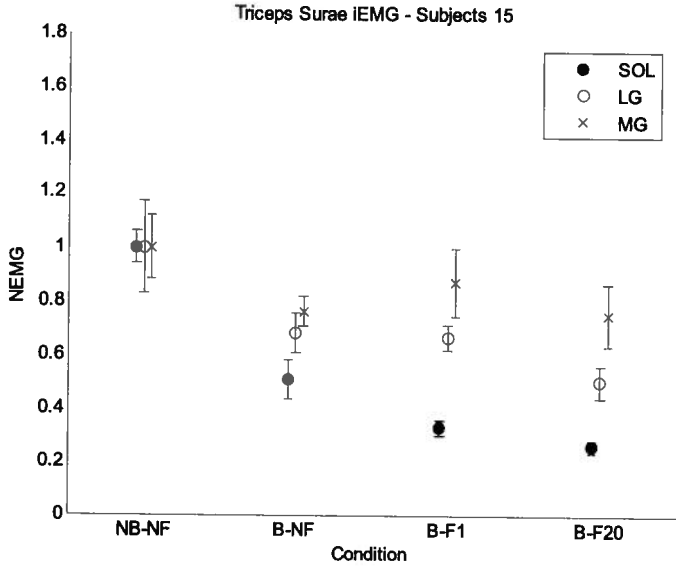


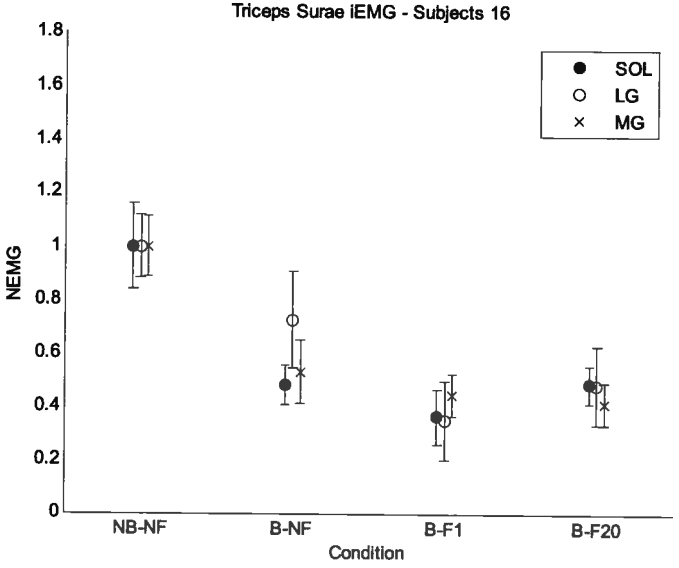


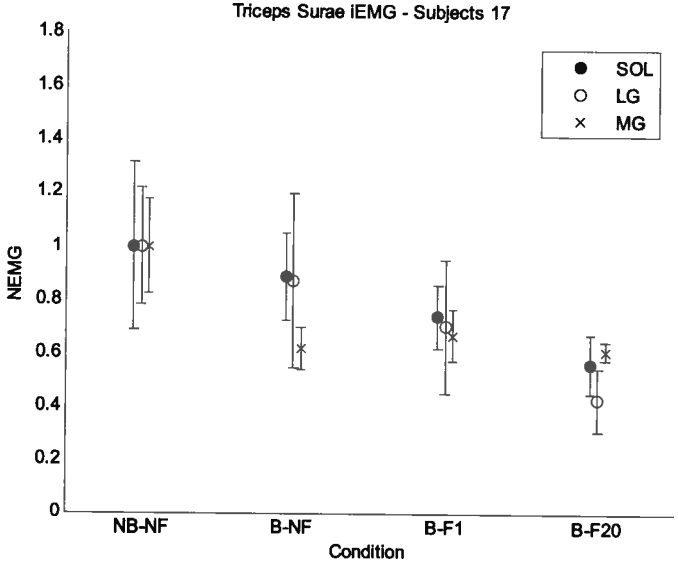


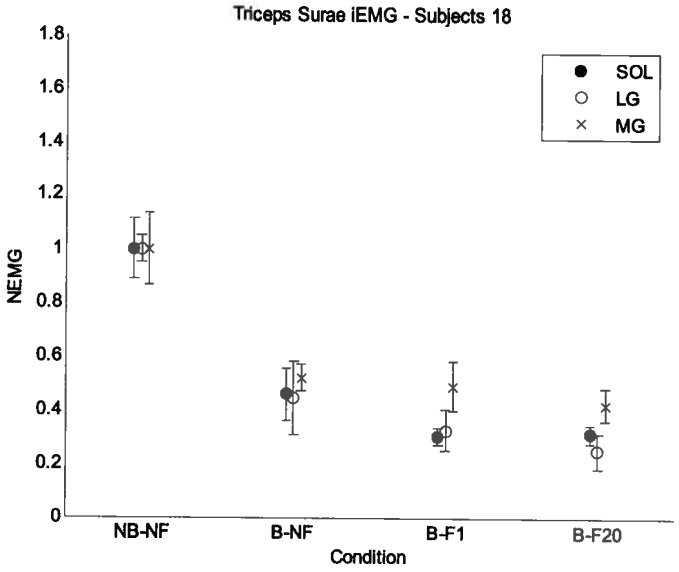


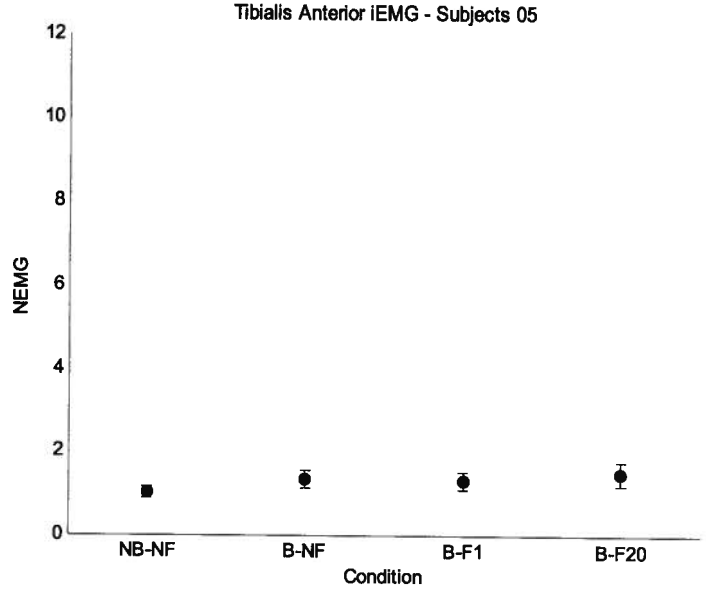

Triceps Surae iEMG - Subjects 14
NEMG
Condition
NB-NF
B-NF
B-F1
B-F20
SOL
LG
MG
0
0.2
0.4
0.6
0.8
1
1.2
1.4
1.6
1.8

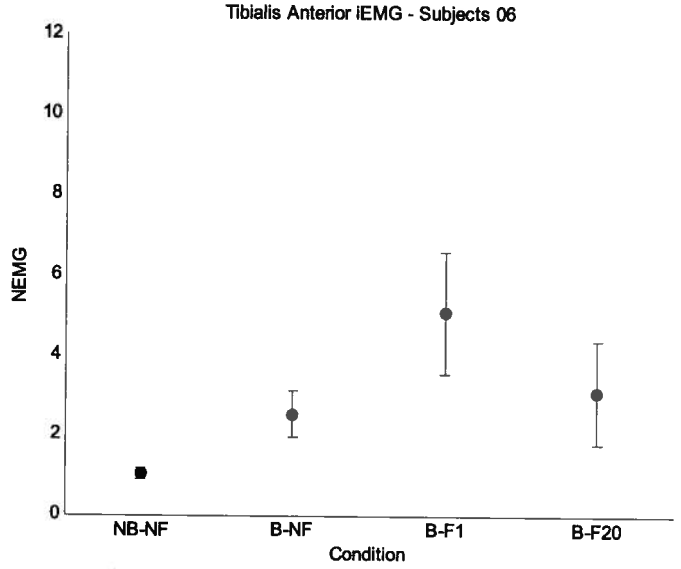

Triceps Surae iEMG - Subjects 15
NEMG
Condition
NB-NF
B-NF
B-F1
B-F20
SOL
LG
MG
0
0.2
0.4
0.6
0.8
1
1.2
1.4
1.6
1.8

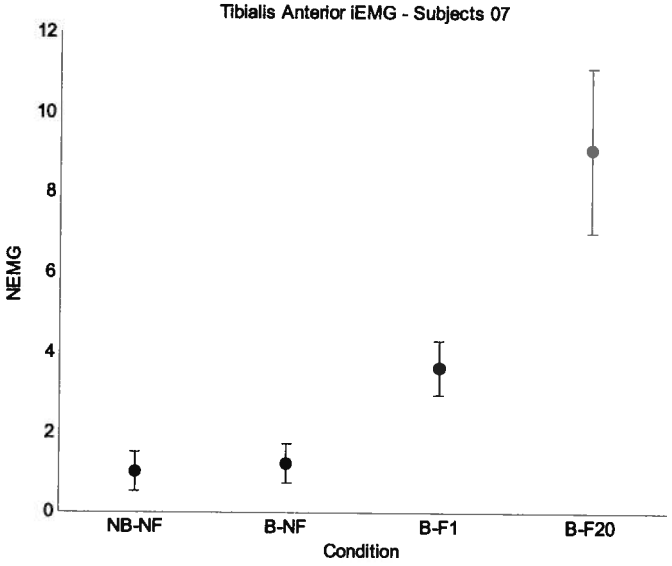

Triceps Surae iEMG - Subjects 16
NEMG
Condition
NB-NF
B-NF
B-F1
B-F20
SOL
LG
MG
0
0.2
0.4
0.6
0.8
1
1.2
1.4
1.6
1.8

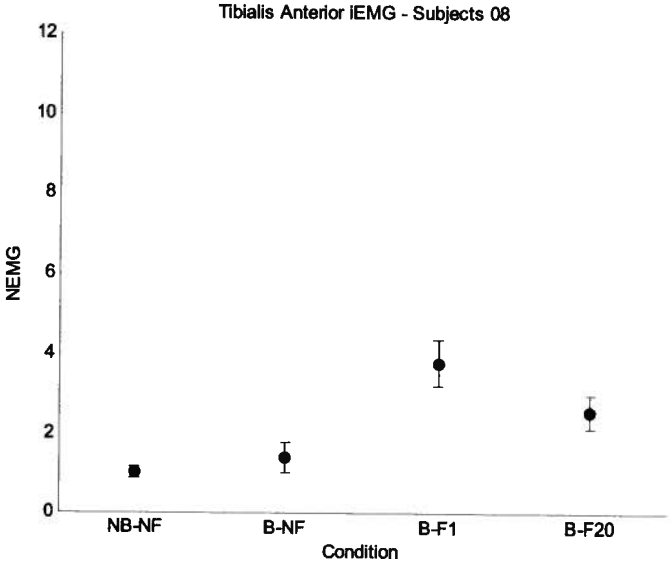

Triceps Surae iEMG - Subjects 17
NEMG
Condition
NB-NF
B-NF
B-F1
B-F20
SOL
LG
MG
0
0.2
0.4
0.6
0.8
1
1.2
1.4
1.6
1.8

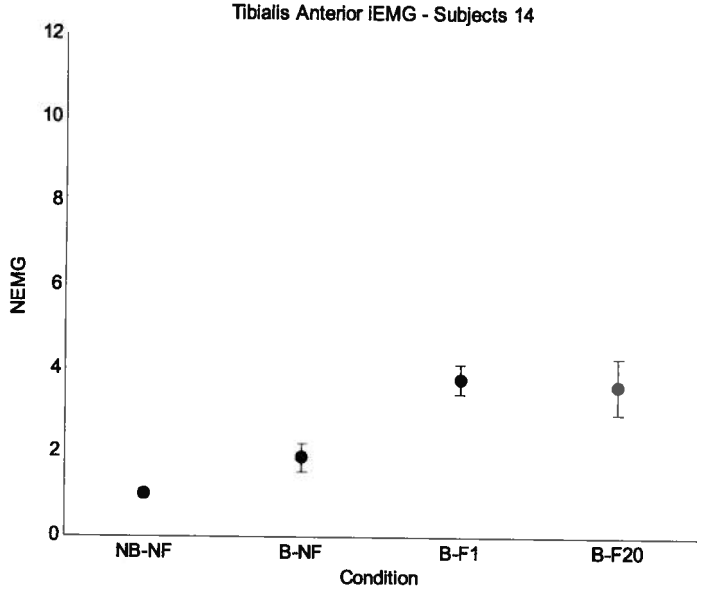

Triceps Surae iEMG - Subjects 18
SOL
LG
MG
NEMG
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
NB-NF
B-NF
B-F1
B-F20
Condition

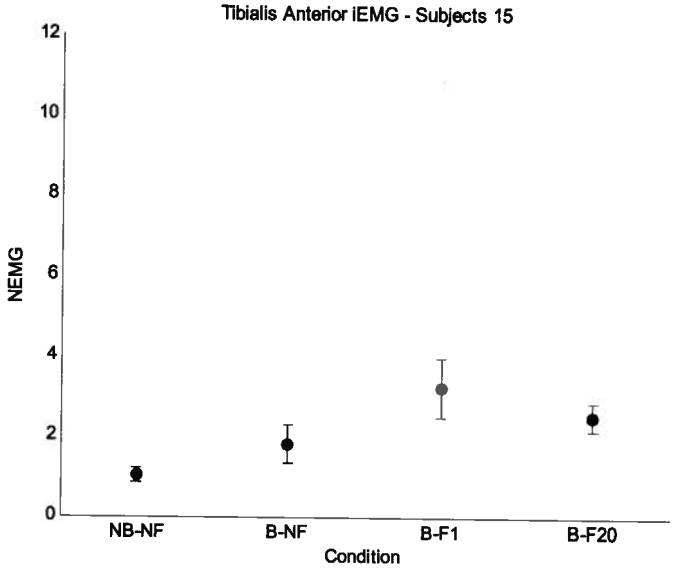

Tibialis Anterior iEMG - Subjects 05
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition

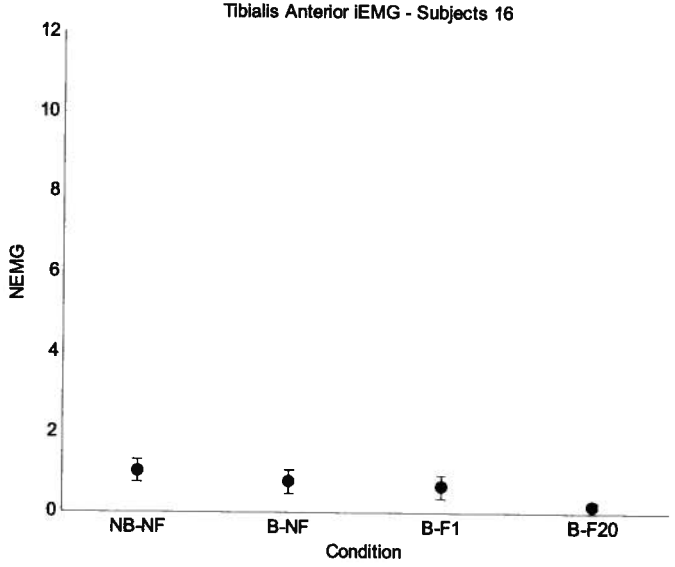

Tibialis Anterior iEMG - Subjects 06
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition

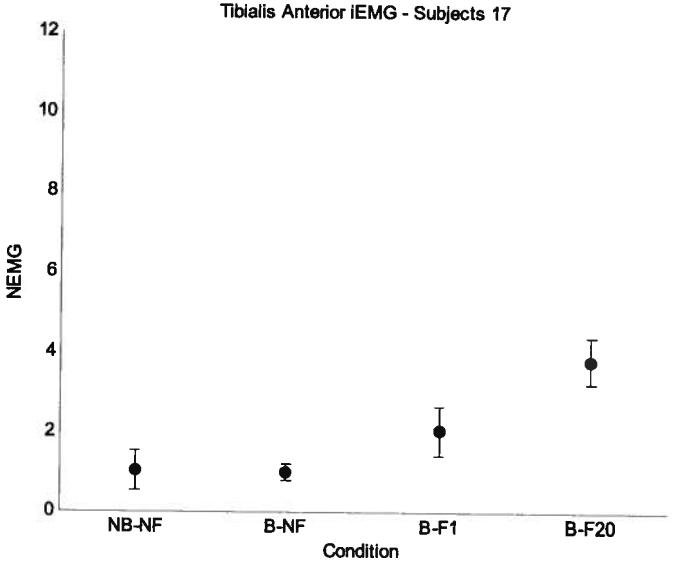

Tibialis Anterior iEMG - Subjects 07
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition

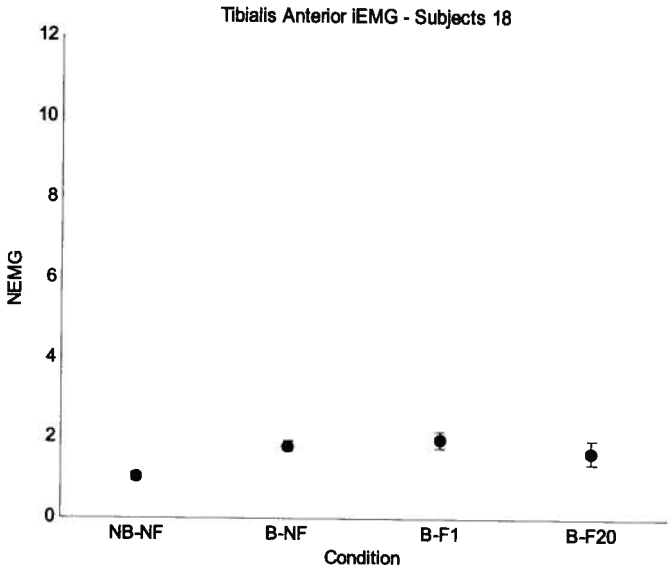

Tibialis Anterior iEMG - Subjects 08
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition

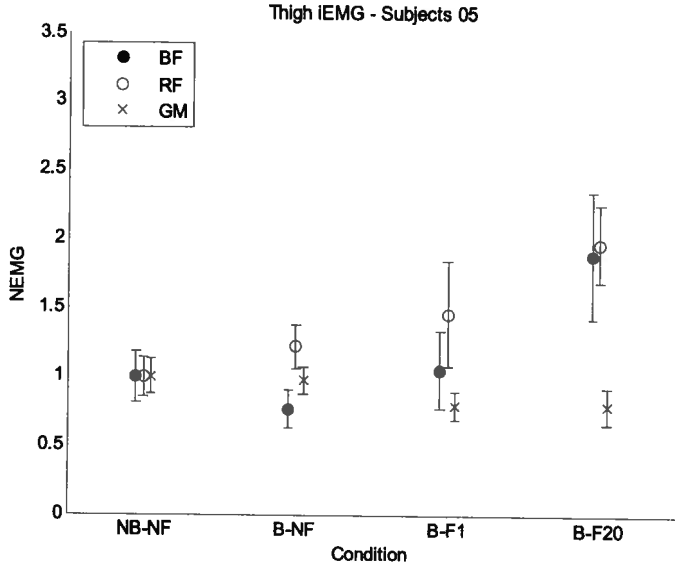

Tibialis Anterior iEMG - Subjects 14
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition

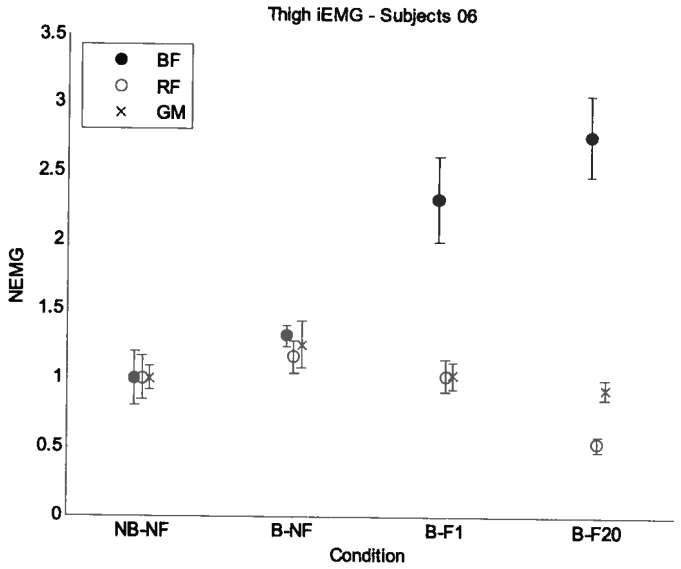

Tibialis Anterior iEMG - Subjects 15
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition


Tibialis Anterior iEMG - Subjects 16
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition


Tibialis Anterior iEMG - Subjects 17
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition


Tibialis Anterior iEMG - Subjects 18
NEMG
12
10
8
6
4
2
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 05
BF
RF
GM
NEMG
3.5
3
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 06
BF
RF
GM
NEMG
3.5
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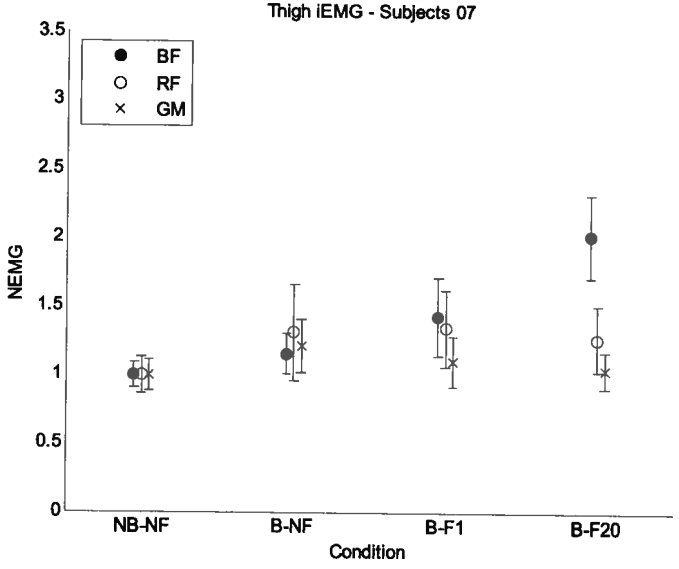
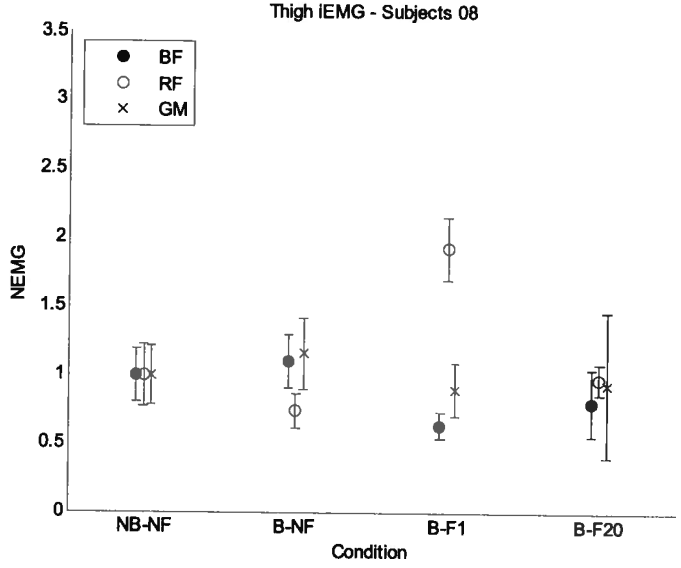
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 07
BF
RF
GM
NEMG
3.5
3
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 08
BF
RF
GM
NEMG
3.5
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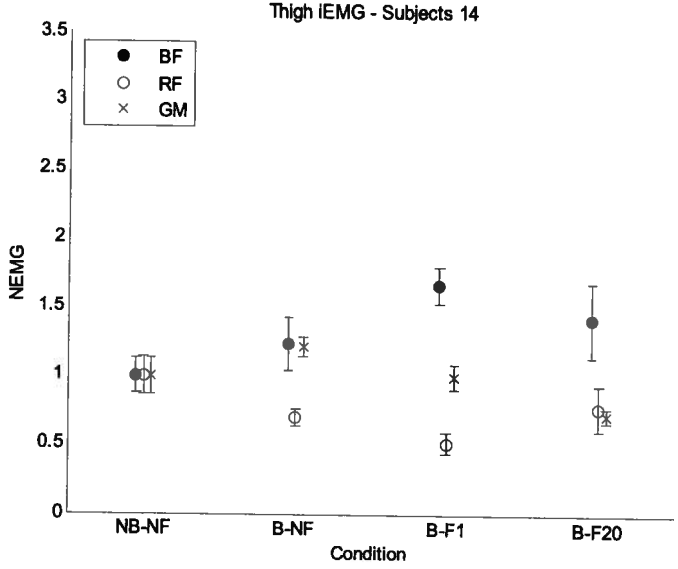
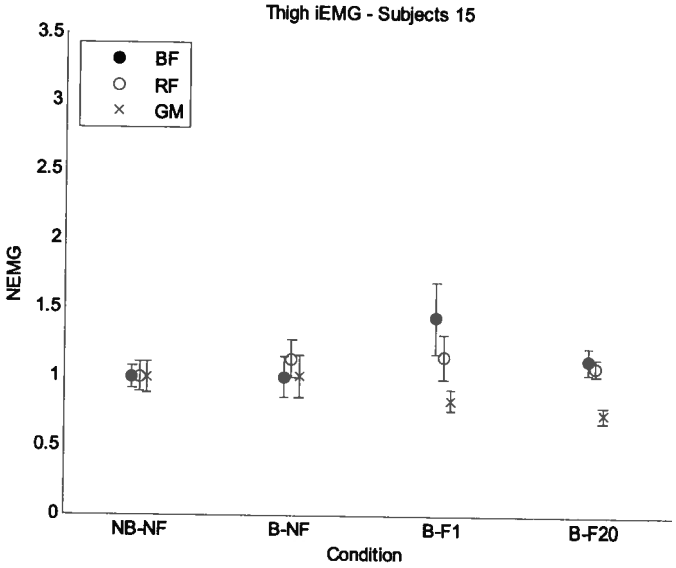
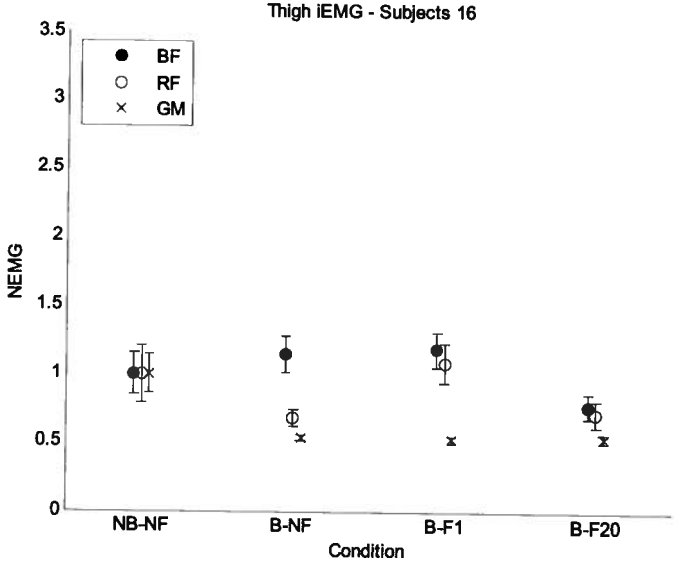
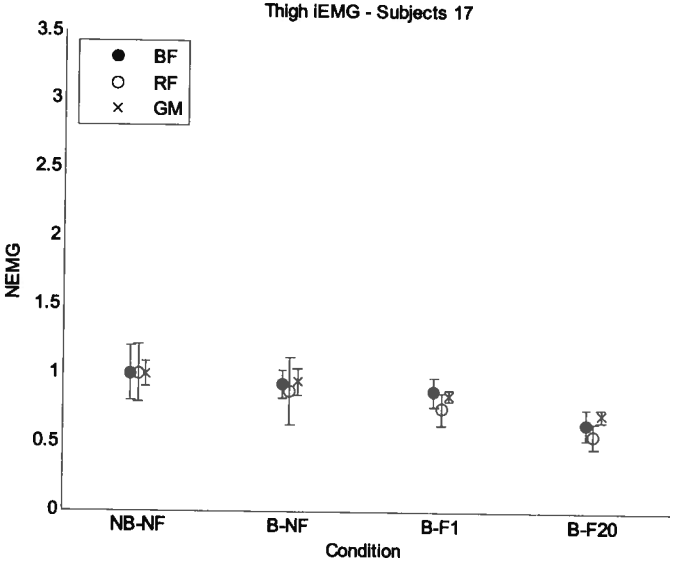
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 14
BF
RF
GM
NEMG
3.5
3
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 15
BF
RF
GM
NEMG
3.5
3
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 16
BF
RF
GM
NEMG
3.5
3
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 17
BF
RF
GM
NEMG
3.5
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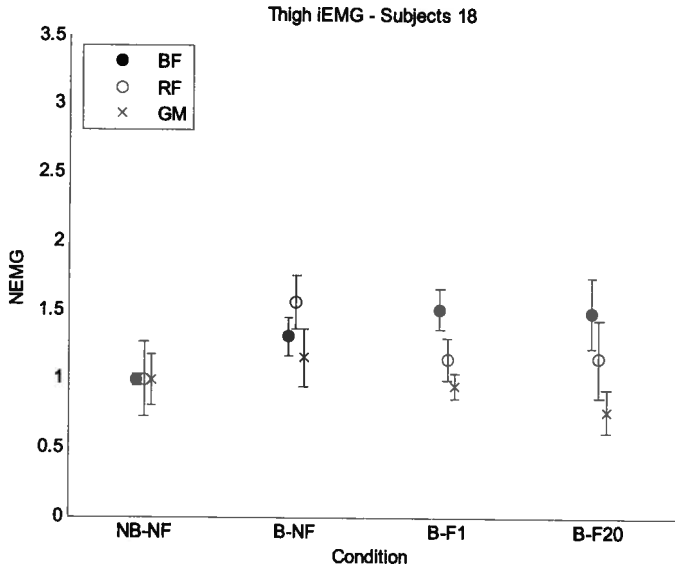
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition


Thigh iEMG - Subjects 18
BF
RF
GM
NEMG
3.5
3
2.5
2
1.5
1
0.5
0
NB-NF
B-NF
B-F1
B-F20
Condition