PLANTAR FLEXOR CENTRAL AND PERIPHERAL FATIGUE DURING SIMULATED SOCCER MATCH PLAY

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SUMMARY (DANISH)

Formål: I dette studie undersøgte vi indledende, hvorvidt der opstår central og perifer træthed i plantar fleksorerne som et resultat af 90 minutters simuleret fodboldkamp ($SAFT^{90}$), samt hvorvidt central træthed ledsages af en modulering af de spinale kredsløb. $SAFT^{90}$ blev brugt til at genskabe de fysiologiske krav og aktivitetsprofilen fra en fodboldkamp, hvormed det er muligt at undersøge træthed i en kontrolleret eksperimentel kontekst. **Metoder:** Otte amatør fodboldspillere deltog i det nærværende studie. Studiet var designet som et *crossover-studie*, hvor forsøgspersonerne deltog i én eksperimentel-session og én kontrol-session, der blev udført i en randomiseret rækkefølge. Forsøgspersonerne udførte $SAFT^{90}$ i eksperimentel-sessionen, hvor der blev foretaget neuromuskulære test før kampen (T_0), i halvlegen (T_{45}) og efter kampen (T_{90}). Forsøgspersonerne foretog de samme målinger i kontrol-sessionen, men i modsætning til at udføre $SAFT^{90}$, så hvilede forsøgspersonerne i det samme tidsrum. De følgende variable blev indhentet ved T_0 og T_{90}: Maksimal voluntær kraft (MVC), voluntær aktivering (VA), peak kraft fra et kontrol twitch fremkaldt i den afslappede muskel, maksimal overflade elektromyografi (sEMG) aktivitet fra soleus (SOL), statisk og dynamisk Hoffmann-reflekser (H-reflex) and volitional-waves (V-wave). Herudover blev MVC, VA, peak kraft fra kontrol twitch og maksimal sEMG fra SOL indhentet ved T_{45}. **Resultater:** MVC var faldet med -13,7 % ($p = 0,042$) fra T_0 til T_{90} i eksperimentel-sessionen, hvorimod den forblev stabil i kontrol-sessionen. Peak kraft fra kontrol twitch var faldet i eksperimentel-sessionen fra T_0 til T_{45} (-23,4 %, $p < 0,001$). Der blev ikke observeret nogen signifikante ændringer for VA, maksimal sEMG aktivitet fra SOL (RMS/M_{MAX}), statisk og dynamiske H-reflekser samt V-waves. **Konklusion:** Dette studie viste at $SAFT^{90}$ forårsager træthed i plantar fleksorerne, hvilket kunne ses i form af reduktioner i den maksimale kraft. Denne nedgang i maksimal kraft var ledsaget af perifer træthed, hvilket var indikeret af en nedgang i peak kraft af kontrol twitchet. Ydermere viste vores resultater tendenser til central træthed, hvilket kunne ses i form af ikke-signifikante reduktioner i VA og V-waves. Det var dog ikke muligt at se nogle indikationer af en modulering af de spinale kredsløb, som defineret ud fra ændringer i amplituden på H-reflekser.

PREFACE

This master thesis was conducted by group 1031 at Aalborg University, as part of the fourth semester masters-program in Sports Science. The thesis was conducted during a four month period and includes work equivalent to 30 ECTS points.

With this thesis we initially investigated whether plantar flexor central and peripheral fatigue would occur as a result of 90minutes of simulated soccer match play (soccer-specific aerobic field test). Furthermore, we sought to elaborate on the central components of fatigue, by investigating if central fatigue of the plantar flexors would be accompanied by indications of spinal and/or supra-spinal fatigue. A more comprehensive understanding of the factors that contribute to fatigue in soccer may be beneficial in understanding the cause of musculoskeletal injuries, which might help in the design of future injury prevention strategies.

We would like to thank Dr. Natalie Mrachacz-Kersting, Professor Uwe G. Kersting & MSc. Priscila de Brito Silva for their supervision and help throughout the study. Additionally, we would like to thank the soccer players who volunteered to take part in the study.

READER GUIDELINES

The master thesis consists of a manuscript and supplemental material. The manuscript is written based on the present study and consists of an introduction to the area of research that includes the aim of the study, a description of the methods, a results section, a discussion of these results and finally a conclusion.

The supplemental material contains theoretical background information and a more detailed description of the applied methods, which is helpful for the understanding of the manuscript. More precisely, the supplemental material contains a description of the soccer-specific aerobic field test, fatigue, electromyography measurements, the twitch interpolation technique, the Hoffmann-reflex and Volitional-wave.

References are made using the Harvard method.

PLANTAR FLEXOR CENTRAL AND PERIPHERAL FATIGUE DURING SIMULATED SOCCER MATCH PLAY

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ABSTRACT

Purpose: In the current study it was investigated whether plantar flexor central and peripheral fatigue would occur as a result of 90 min of simulated soccer match play ($SAFT^{90}$), and further if central fatigue would be accompanied by modulations of spinal loop properties. The $SAFT^{90}$ was used to replicate the physiological response and activity demands of soccer match play, to investigate fatigue in a controlled experimental context.

Methods: Eight amateur soccer players participated in the present study. The study was designed as crossover study, where the participants took part in one experimental session and one control session, performed in a randomized order. The participants performed the $SAFT^{90}$ in the experimental session, where neuromuscular tests were conducted prior to the match (T_0), at half-time (T_{45}) and after the match (T_{90}). In the control session the participants performed the same measurements, however, instead of performing the $SAFT^{90}$ they rested for the same duration. The following variables were assessed at T_0 and T_{90}: Maximal voluntary force (MVC), voluntary activation (VA), peak force of the control twitch evoked in the relaxed muscle, maximal surface electromyography (sEMG) activity of soleus (SOL), static and dynamic Hoffmann-reflexes (H-reflex) and volitional-waves (V-wave). Additionally, MVC, VA, peak force of the control twitch and maximal sEMG of SOL were also assessed at T_{45}.

Results: MVC deceased by -13.7% ($p = 0.042$) from T_0 to T_{90} in the experimental session, while it remained stable in the control session. The peak force of the control twitch decreased in the experimental session from T_0 to T_{45} (-23.4%, $p < 0.001$). No significant changes were observed for VA, maximal SOL sEMG activity (RMS/M_{MAX}), static and dynamic H-reflexes and V-waves.

Conclusion: This study showed that the $SAFT^{90}$ causes fatigue of the plantar flexors, seen as decreases in maximal force. This decline was accompanied by peripheral fatigue, as indicated by a decrease in the peak force of the control twitch. The results showed tendencies towards central fatigue, seen as non-significant reductions in VA and V-waves. It was not possible to see indications of modulations of spinal loop properties, as defined from changes in the amplitude of the H-reflex.

Key words: Simulated soccer match play, plantar flexion, central fatigue, peripheral fatigue, H-reflex, V-wave

INTRODUCTION

According to Fédération Internationale de Football Association (FIFA) more than 15 million adults play soccer in registered clubs, while 110,000 players are professional. (FIFA, 2006) Both at amateur (Schmikli et al., 2009) and elite level (Junge & Dvorak, 2004), a great number of these players

experience injuries. Lower leg injuries are common in professional players, with ankle injuries accounting for 17% of all injuries (Woods et al., 2003) and 13% of muscle injuries occurring to the calf muscles (Ekstrand, 2011). Muscular fatigue has been related to the incidence of injuries in soccer, as the injuries often occur during the latter stages of each half during a match. (Woods et al., 2003; Hawkins & Fuller, 1999; Hawkins et al., 2001) For instance, Woods et al. (2003) found that nearly half of all ankle sprains were sustained during the last 15 min of each half during a match. Investigations into the factors that contribute to fatigue may thus provide insight regarding the cause of musculoskeletal injuries in soccer.

Muscular fatigue of the plantar flexors may be caused by both central and peripheral factors. Central fatigue can be defined as the progressive reduction in the voluntary activation (VA) of a muscle, whereas peripheral fatigue is caused by changes occurring at or distal to the neuromuscular junction. (Gandevia, 2001, p. 1733) While central fatigue is commonly investigated using the twitch interpolation technique (TIT; Shield & Zhou, 2004), measurements of the Hofmann reflex (H-reflex) and the volitional wave (V-wave) can be used to determine whether central fatigue originates at the spinal and/or supra-spinal level. To date only one study has investigated plantar flexor central and peripheral fatigue following soccer match play using the conventional TIT method. (Nybo et al., 2013) By employing measurements 30 min following the termination of a soccer match, Nybo et al. (2013) showed reductions in VA and the control twitch, indicating the presence of both central and peripheral fatigue. However, based on these results it is not known whether central fatigue originated at the spinal and/or supra-spinal level. Results from studies where plantar flexor central and peripheral fatigue were investigated following continuous running (90 min; Racinais et al., 2007), intermittent running (90 min; Racinais et al., 2007), five km running (Girard et al., 2012) and tennis match play (3 h match protocol; Girard et al., 2011) suggest that central fatigue may be caused by modulations of spinal loop properties. For example, decreases in the amplitude of the H-reflex (Racinais et al., 2007; Girard et al., 2011; Girard et al., 2012) and V-wave (Racinais et al., 2007; Girard et al., 2011) indicate that central fatigue is mediated by changes occurring at both the spinal and supra-spinal level.

Based on these results, the current study was designed to address whether central fatigue following soccer match play is accompanied by modulations of spinal loop properties. As the locomotor profile varies significantly between and within soccer matches (Rampinini et al., 2007; Gregson et al., 2010), a protocol was implemented (soccer-specific aerobic field test ($SAFT^{90}$)) to replicate the physiological response and activity demands of soccer match play (Lovell, Knapper & Small, 2008). In the present study, fatigue was defined as a reduction in maximal voluntary force (MVC) (Gandevia, 2001). The aim of this study was to determine whether plantar flexor central and peripheral fatigue would be present as a result of the $SAFT^{90}$, and to further investigate if central fatigue would be accompanied by modulations of spinal loop properties.

METHODS

PARTICIPANTS

Eight young healthy male amateur soccer players (age; 24.3 years ± 2.8, height; 178.1 cm ± 6.3, body mass; 75 kg ± 4.8) took part in the study. None of the players had experienced any musculoskeletal injuries to the lower extremities within the last six months. The study was carried out during the competitive season and players usually trained two times per week including one official match. Written informed consent was obtained after the participants had received verbal and written information concerning the experiment. The study was approved by the local ethics committee and carried out in accordance with the declaration of Helsinki.

EXPERIMENTAL OVERVIEW

The study was designed as a crossover study, where the participants took part in one experimental and one control session, which were performed in a randomized order and separated by a minimum of seven days. Additionally, participants were familiarized with the experimental procedures of the study a minimum of two days prior to the control or experimental session. The study was performed in a climate controlled athletics hall (Aalborg, Denmark, Temperature = 19 °C). In the 24 hour period prior to each test, participants refrained from consuming any alcohol or caffeine and did not perform any

vigorous exercise or exercise they were unaccustomed to. The participants were further asked not to consume large quantities of food within one hour prior to the experimental or control session and to be hydrated upon arrival.

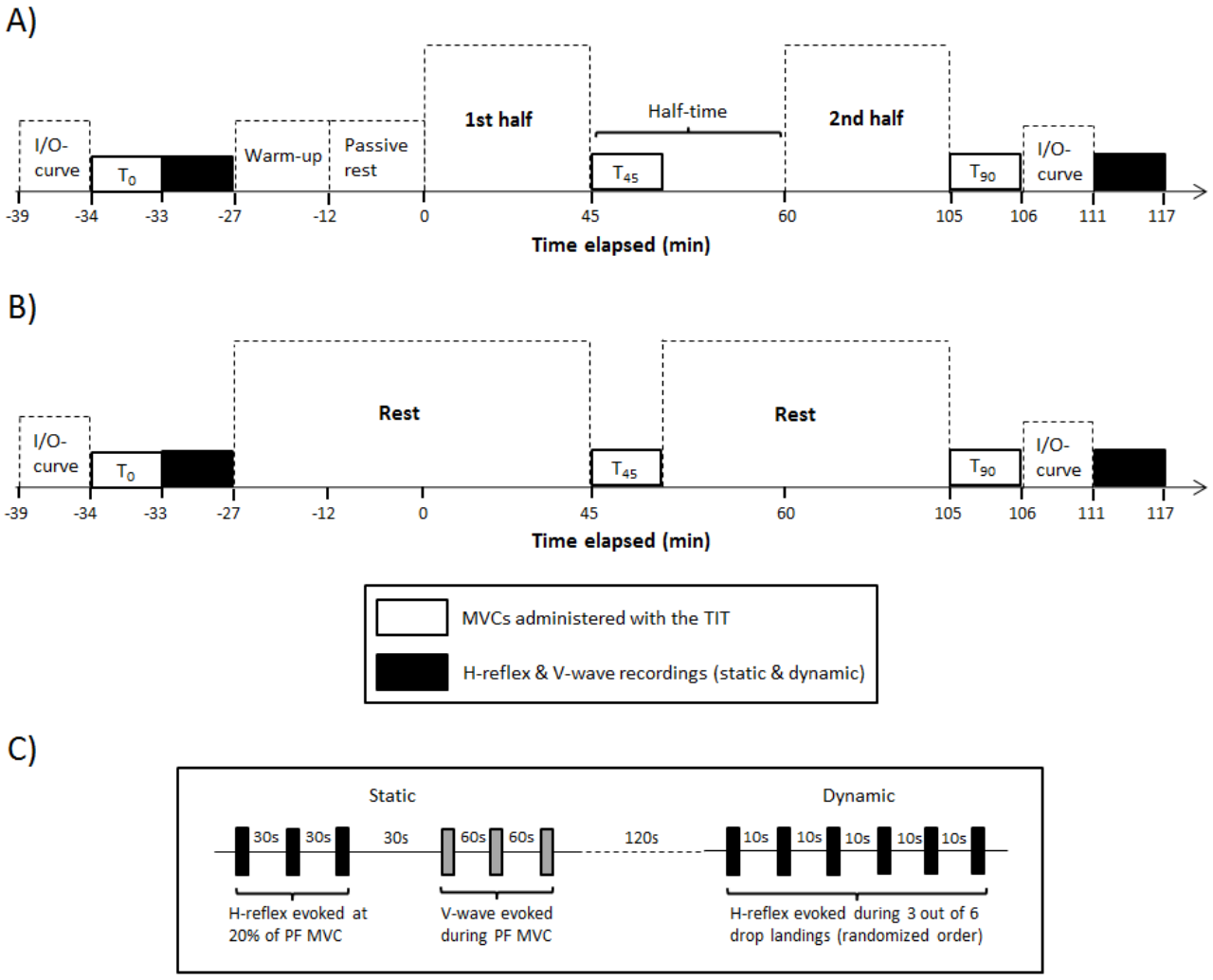
TESTING SESSIONS

The familiarization included habituating the participants with the testing procedures and the SAFT^{90} (Small et al., 2010) (see section *Fatigue Protocol)*.

In the experimental session participants completed the SAFT^{90}, where neuromuscular tests (see section *Neuromuscular tests*) were conducted prior to the start of SAFT^{90} (T_0), at the end of the first half (T_{45}) and post SAFT^{90} (T_{90}). The half-time interval was conducted as a 15 min passive rest period, where the participants remained seated throughout the break.

In the control session the participants performed the same measurements, however, instead of performing the SAFT^{90} they rested for the same duration. The sequence of measurements and events is illustrated in Figure 1 for the experimental and control session, respectively.

Figure 1. An overview of the time course of the experimental (A) and control session (B), as well as an overview of the Hoffmann reflex (H-reflex) and volitional wave (V-wave) recordings (C) performed pre and post the soccer-specific aerobic field test (SAFT^{90}).



ELECTROMYOGRAPHY

Following appropriate skin preparation, pairs of AG/AgCl surface electrodes (Ambu Neuroline 720, Ambu A/S, Ballerup, Denmark) were placed on the belly of the muscle on soleus (SOL), gastrocnemius medialis (GM) and tibialis anterior (TA) of the preferred kicking leg (Right leg, n = 5; Left leg, n = 3) in accordance to the recommendations by SENIAM. The electrodes were placed in a bipolar configuration with an inter-electrode distance of 20 mm (from center to center). Due to individual differences, the placements of the electrodes were carefully checked by palpation of the borders of SOL, GM and TA. A reference electrode (Ambu Neuroline 720, Ambu A/S, Ballerup, Denmark) was placed on the medial surface of the tibia. The specific position of the electrodes was marked on a reference sheet during the first testing session (experimental or control) to ensure correct electrode placement between sessions. All electrodes were carefully taped down to ensure that they remained in the same position as the participants started to sweat. Surface electromyography (sEMG) signals were sampled at 2000 Hz, analog to digital converted (National Instruments, NI USB-6221, USA), amplified (gain = 1000) and filtered between 0.5-1000 Hz. During offline analysis, signals were further rectified and filtered between 30-500 Hz.

STIMULATION

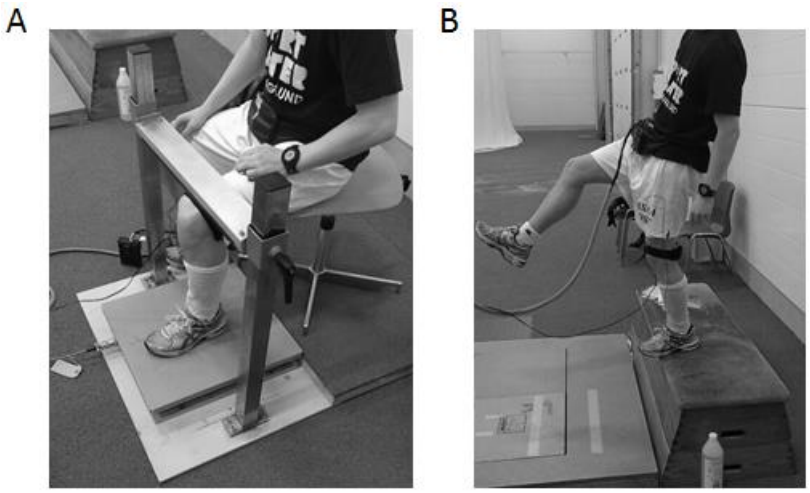
The tibial nerve was stimulated using a cathode electrode (3.2 cm in diameter; PALS Platinum, Axelgaard Manufacturing, USA) positioned in the popliteal fossa and a rectangular anode electrode (5×9 cm; PALS Platinum rectangular electrode, Axelgaard Manufacturing, USA) placed proximal to the patella. Prior to positioning of the cathode electrode, the optimum site of stimulation was located. The site was chosen based on the criterion that the SOL Ia afferents would be stimulated selectively at low stimulation intensities. This was controlled by ensuring an H-wave appeared prior to an M-wave. The cathode electrode was fixated with a strap to ensure the same stimulation intensities during different tasks. The stimulation consisted of a 1 ms rectangular pulse delivered by a constant current stimulator (Isolated Stimulator, NoxiTest, IES 230). With the participant standing, the amperage was gradually increased in 10 mAmp increments until a plateau in the measured peak-to-peak M-wave of SOL was

attained. This intensity was then used as a guideline to perform the input-output curve (I/O-curve) and to establish the supra-maximal (120%) stimulation intensity. The supra-maximal stimulation intensity was maintained throughout the experimental (50.9 mA ± 10.8 [42-70.8 mA]) and control session (51.5 mA ± 6.8 [42-60 mA]). The I/O-curve consisted of 45 stimuli divided into 15 classes of varying intensity and was performed with the participant seated with a constant SOL background sEMG activity corresponding to 10±2.5% of maximal SOL sEMG activity.

MAXIMAL VOLUNTARY AND EVOKED CONTRACTIONS

All voluntary and evoked isometric contractions of the plantar flexors were performed using a purpose built aggregate (Figure 2A), where force was measured using a force plate (AMTI, OR6-7-1000, Watertown, MA, USA). MVCs were performed on the preferred kicking leg with the participants seated with a hip, knee and angle joint angle at approximately 90°. The participants placed their hands on their shoulders during contractions and were instructed to perform each contraction as "fast and forceful as possible". Verbal encouragement was provided to encourage a maximal effort. Two sub-maximal contractions at 25, 50 and 75% of self-perceived MVC were performed prior to T_0, in order to prepare. These contractions were repeated prior to the measurements at T_{45} and T_{90} in the control session. Additionally, two MVCs were performed prior to T_0 to obtain a reference point for the I/O-curve (background SOL sEMG; see section *Stimulation*) and the *static H-reflex recordings* (20% of MVC; see section *Neuromuscular tests*). Force signals were recorded at 2000 Hz using an analog to digital converter (National Instruments, NI USB-6221, USA), amplified (AMTI DigiAmp DSA-6, Watertown, MA, USA; Gain = 5000) and filtered during offline analysis using a 2nd order digital 10 Hz low-pass filter.

Figure 2. The aggregate used to obtain maximal voluntary (MVC) and evoked contractions in the present study (A) and a representative participant preparing to perform a drop landing (B).



NEUROMUSCULAR TESTS

MVC with TIT: Two 3-4 s duration plantar flexor MVCs (separated by 30 s of rest) were administered with the TIT and performed at T_0, T_{45} and T_{90}. One doublet (2 twitches, 10ms interval) at supra-maximal stimulation intensity (120%) was evoked and superimposed on the MVC when the force was at its maximum and had reached a subsequent plateau (determined visually). A control twitch (potentiated doublet) was further evoked in a relaxed state 3-4 s after the MVC was stopped. The following parameters were extracted from each MVC with respect to force and sEMG; 1) MVC was determined as the greatest force value recorded during the contraction (prior to stimulation) and 2) maximal sEMG of SOL was determined as the greatest 50 ms root-mean-square value obtained in the 100 ms time interval leading up to the maximal force (prior to stimulation). The maximal sEMG of SOL was normalized to the corresponding M-wave evoked during the contraction. Furthermore, peak force and the index of VA was determined from the control twitch, where the index of VA was calculated based on the ratio between the superimposed doublet over the size of the control twitch as follows; %VA = [1 – (superimposed twitch/control twitch)] × 100 (Shield & Zhou, 2004). The superimposed twitch was determined as the peak force detected within 200 ms after the stimulation subtracted with the mean force during the 50 ms preceding the stimulation (M-wave). (Nordlund et al., 2004)

H-reflex recordings: *Static H-reflex recordings* were obtained from SOL at T_0 and T_{90}, where the participants performed three 2-3 s duration isometric plantar flexor contractions at 20% of MVC (obtained in non-fatigued state prior to T_0), each separated by 30 s of rest. A single stimulation was evoked when the participants were able to keep the force stable at 20% of MVC. The stimulation intensity used to evoke H-reflexes (both static and dynamic) was found by inspecting the M-wave and corresponding H-reflex amplitudes in the I/O-curve. The class where the H-reflex was on the ascending part of the recruitment curve was chosen as the H-reflex stimulation intensity. This procedure was performed pre and post $SAFT^{90}$ and it was ensured that the stimulation intensity evoked an M-wave of equivalent percentage of M-max both pre and post $SAFT^{90}$. The peak-to-peak amplitude of the H-reflex was extracted from each of the three trials, normalized to the first M-wave evoked during the *V-wave recordings* and subsequently averaged.

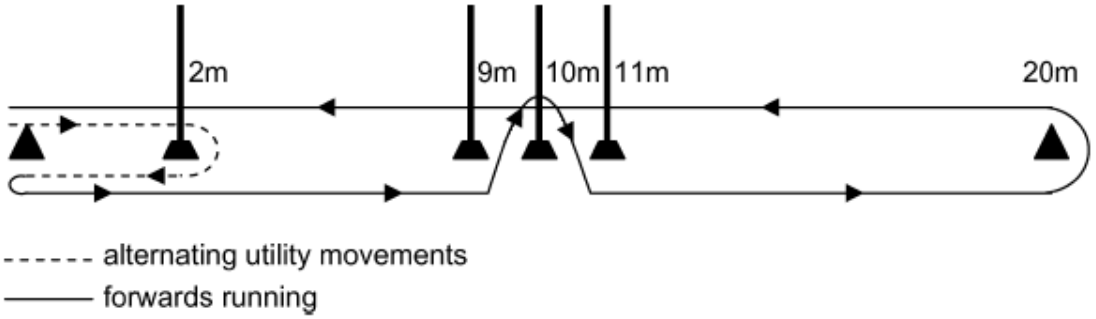
Dynamic H-reflex recordings were obtained from SOL at T_0 and T_{90} during a drop landing. The participants performed a unilateral drop from a box (30 cm in height) to a force plate (AMTI, OR6-7-1000, Watertown, MA, USA) and subsequently maintained their balance on the landing leg (kicking leg) for three seconds. The drop was performed by raising the non-landing leg (non-kicking leg) to an angle of 45°, enabling them to displace their center of mass forward and drop to the force plate. Figure 2B shows a participant preparing to perform a drop landing. During the drop, the participants focused straight ahead and kept their arms across the chest. A single stimulation was delivered as the front foot of the kicking leg made ground contact, triggered by a force sensitive resistor (foot switch) placed on the bottom of the shoe in line with the 1st metatarsal bone. The participants performed a total of six drop landings, three with stimulation and three without stimulation performed in a randomized order. Each drop landing was separated by a 10 s rest period. Additional trials were carried out if the drop landing was performed incorrectly or if the foot switch was triggered unintentionally. The peak-to-peak amplitude of the H-reflex was extracted from the three trials with stimulation, normalized to the first M-wave evoked during the *V-wave recordings* and averaged.

V-wave recordings: V-waves were obtained from SOL during three 2-3 s duration plantar flexor MVCs performed at T_0 and T_{90}. Each MVC was separated by a one min rest period. A single supra-maximal stimulation was evoked and superimposed on the MVC when the force was at its maximum and had reached a subsequent plateau. The peak-to-peak amplitude of the V-wave was extracted from each of the three MVCs in which the corresponding amplitude of the M-wave was ≥ 95% of M-max (Aagaard et al., 2002), where M-max was defined as the M-wave evoked during the first MVC performed at T_0. V-waves were subsequently averaged and normalized to the corresponding M-wave evoked during the contraction.

FATIGUE PROTOCOL

The SAFT^{90} is developed based on time-motion analysis from English Championship level match play and has been validated to simulate the physiological responses of soccer match play (Lovell et al., 2008). The protocol is intermittent in nature and incorporates multidirectional and utility movements, including frequent accelerations and decelerations. (Lovell et al., 2008) The SAFT^{90} is performed over a 20 m course that incorporates four poles the participant has to navigate using utility movements. (Small et al., 2010; Figure 3) The participant navigates around the first pole by either backwards running or sidestepping and continues by running forwards through the course, while navigating through the three middle poles. The protocol is developed as a 15 min activity profile (typically repeated six times to mimic 90 min of soccer match play), where the intensity and movement activity is maintained using verbal signals from an audio file. (Small et al., 2010) Before commencing the SAFT^{90}, a 15 min standardized warm-up consisting of 12 different soccer-specific warm-up exercises was performed. The warm-up was followed by a 12 min passive rest period prior to the start of the SAFT^{90} to resemble the pre-match routine of a professional soccer match (Towlson et al., 2013). Heart rate (Garmin Forerunner 210, Garmin Ltd., England) was measured continuously throughout the SAFT^{90} and participants were allowed to drink water ad libitum before, during half-time and after the SAFT^{90}.

Figure 3. A diagrammatic representation of the field course for the soccer-specific aerobic field test (SAFT^{90}). (Small et al., 2010)



STATISTICAL ANALYSIS

All statistical analysis were performed using SigmaStat statistical software (Systat Software Inc©, version 3.5), where statistical significance was set to $p < 0.05$. Data were normally distributed, as assessed by a Kolmogorov-Smirnov Test. Results are presented as mean values ± standard deviation (SD) unless stated otherwise. Two way repeated-measures analysis of variance (ANOVA) were used to analyze depended variables over time, with two levels of session (experimental and control) and either two (T_0 and T_{90}) or three (T_0, T_{45} and T_{90}) levels of time. If a main effect was observed in the ANOVA, post-hoc comparisons were conducted using a Bonferroni correction. If session, time or interaction effects were observed in the ANOVA, mean changes and SD, and 95% confidence intervals (CI) were calculated. Additionally, a paired t-test was used to compare heart rate between the first and second half during the SAFT^{90}.

RESULTS

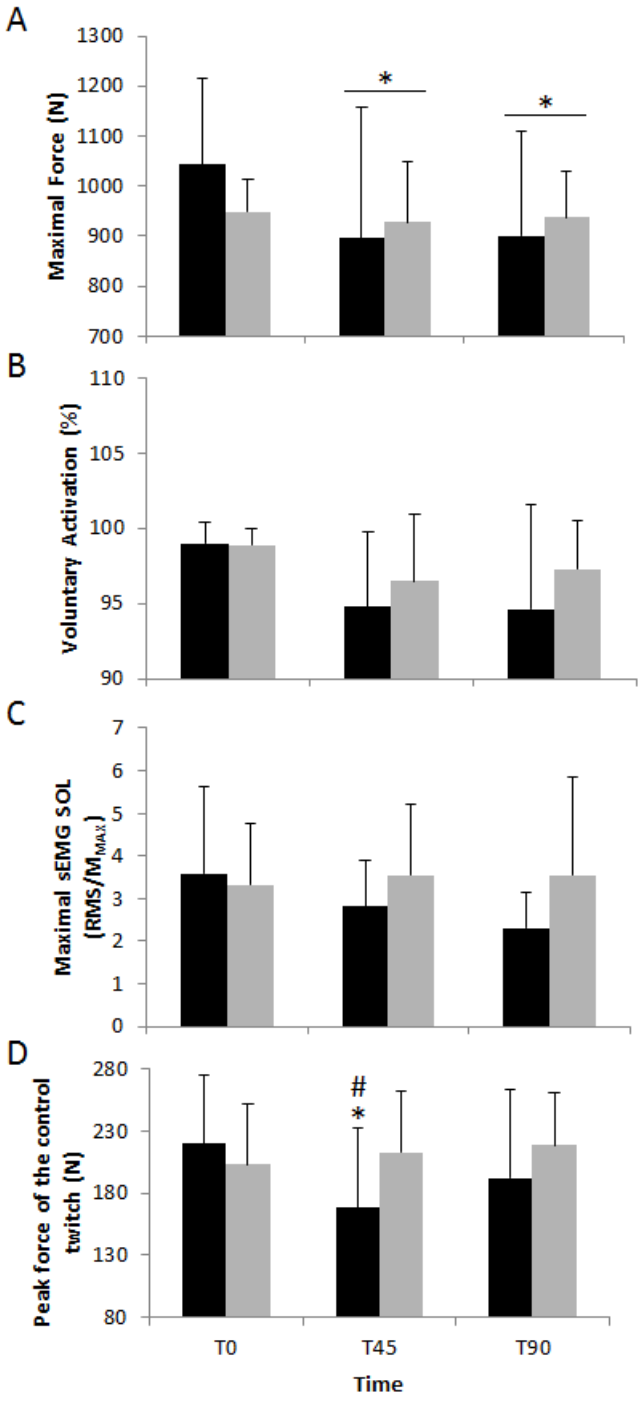
All eight participants completed both testing sessions. One participant was excluded from all statistical analysis, given that the particular participant's maximal force increased as a result of the SAFT^{90}, and the aim of the study was to investigate whether central fatigue is accompanied by modulations of spinal loop properties. Also, two participants were excluded from the statistical analysis of the V-wave recordings, as there was insufficient trials that met the criteria regarding the amplitude of the corresponding M-wave evoked during the contraction (see section *Neuromuscular tests*).

Maximal voluntary contraction force and muscle activation: A main effect of time was observed for maximal force ($F_{(1, 12)}$ = 6.047; p =0.015; Figure 4A), with the maximal force decreased at T_{45} (p = 0.026) and T_{90} (p = 0.042), as compared to pre values. In the experimental session, maximal force was decreased by -14.1% at T_{45} (mean change from T_0 of -146.8 N ± 149.2, 95% CI -250.2 to -43.4) and -13.7% at T_{90} (mean change from T_0 of -142.6 N ± 90.9, 95% CI -205.6 to -79.6). In the control session, maximal force was decreased by -2.1% at T_{45} (mean change from T_0 of -19.8 N ± 126.6, 95% CI -107.5 to 67.9) and -1.1% at T_{90} (mean change from T_0 of -10 N ± 105.8, 95% CI -83.3 to 63.3). For maximal force, no main effects of session or session by time were observed (p =0.103).

VA was reduced by -4.4% at T_{90} in the experimental session and by -1.6% at T_{90} in the control session, compared to pre values. There was no effects of session or session by time interactions for VA (p = 0.131). However, there was a trend towards a decrease over time (p = 0.067; Figure 4B).

The maximal sEMG of SOL (RMS/M_{MAX}) was 3.6 ± 2.0 at T_0 and 2.3 ± 0.8 at T_{90} in the experimental session and 3.3 ± 1.4 at T_0 and 3.6 ± 2.3 at T_{90} in the control session. No main effects of session, time or session by time interactions were observed for maximal sEMG of SOL (RMS/M_{MAX}) throughout the trials (p = 0.233; Figure 4C).

Figure 4. The development in maximal force (A), VA (B), maximal surface electromyography of soleus (C) and peak force of the control twitch (D). Measurements were obtained prior to the soccer-specific aerobic field test (SAFT^{90}; T_0), at half-time (T_{45}) and after the SAFT^{90} in the experimental session (*black bars*), and at equivalent time points during the control session (*grey bars*), where the participants rested contrary to performing the SAFT^{90}. *Significantly different from T_0. #Significantly different from control session.



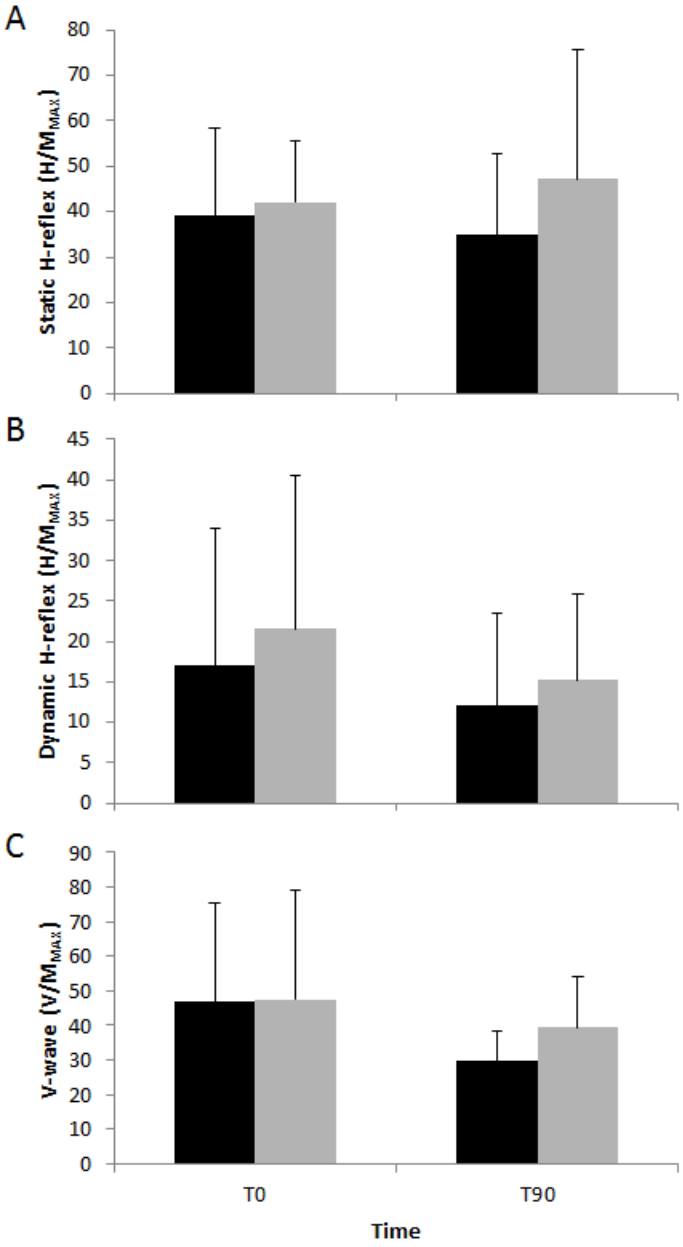
Evoked potentials: There was a significant decrease with time for the peak force of the control twitch ($F_{(1, 12)}$ = 5.598; p = 0.019; Figure 4D), as well as a session by time interaction ($F_{(1, 12)}$ = 5,863; p = 0.017) where the experimental and control session developed differently at T_{45}. In the experimental session, the peak force decreased by -23.4% from T_0 to T_{45} (mean change from T_0 of -51.5 N ± 34.8, 95% CI -75.6 to -27.4, p < 0.001), whereas the peak force did not change over time in the control session (p = 0.552). Additionally, the peak force of the control twitch almost decreased from T_0 to T_{90} in the experimental session (p = 0.066).

Static SOL H-reflexes (H/M_{MAX}) decreased by -10.4% from T_0 to T_{90} in the experimental session, and increased by 12.1% from T_0 to T_{90} in the control session, with no significant effects of session, time or session by time interactions (p = 0.371; Figure 5A). Both in the experimental and control session, dynamic SOL H-reflexes (H/M_{MAX}) were reduced by ~29% from T_0 to T_{90}, with no significant effects (p = 0.146; Figure 5B).

SOL V-waves (V/M_{MAX}) were decreased by -36.7% from T_0 to T_{90} in the experimental session, and -17.1% from T_0 to T_{90} in the control session, with no significant effects of time or session by time interactions (p = 0.285; Figure 5C). However, there was almost a significant difference between the experimental and control session (p = 0.089).

There was a significant increase with time for M-waves from SOL corresponding to the superimposed supra-maximal stimulation during V-wave recordings ($F_{(1, 4)}$ = 8.434, p = 0.044). In the experimental session, M-waves increased by 17.5% from T_0 to T_{90} (mean change from T_0 of 1024.8 µV ± 1344.6, 95% CI 1956.5 to 93.1), whereas M-waves increased by 7.6% from T_0 to T_{90} (mean change from T_0 of 554.7 µV ± 2166, 95% CI 2055.6 to 946.3) in the control session. M-waves from SOL corresponding to the superimposed supra-maximal stimulation during MVCs administered with the TIT, the control twitch and the static H-reflex recordings remained unchanged (p = 0.139).

Figure 5. The development in static Hoffmann-reflexes (H-reflex) (A), dynamic H-reflexes (B) and volitional-waves (V-wave) (C). Measurements were obtained prior to the soccer-specific aerobic field test ($SAFT^{90}$; T_0), at half-time (T_{45}) and after the $SAFT^{90}$ in the experimental session (*black bars*), and at equivalent time points during the control session (*grey bars*), where the participants rested contrary to performing the $SAFT^{90}$.



Heart rate: The peak heart rate decreased significantly from the first half to the second half of the $SAFT^{90}$ ($p = 0.04$; table 1). The average heart rate showed a trend that did not reach statistical significance from the first half to the second half of the $SAFT^{90}$ ($p = 0.09$; table 1).

Table 1. Development in heart rate from the first to the second half of the soccer-specific aerobic field test ($SAFT^{90}$). *Significantly different from the first half.

	Heart rate (bpm)	
	1st half	**2nd half**
Average	157 ± 13	153 ± 13
Peak	178 ± 8	173 ± 9*

DISCUSSION

In this study 90 min of simulated soccer match play caused significant fatigue in all participants, as shown by a significant decrease in the maximal force. This was accompanied by a -23.4% decrease in the peak force of the control twitch at half-time, indicating the presence of peripheral fatigue. There was no clear indication of central fatigue, as no differences were observed in VA and efferent neural drive to SOL. However, VA showed a trend towards a decrease over time, although this failed to reach significance. Furthermore, it was not possible to show any significant differences in both static and dynamic H-reflexes and thus no indications of modulations in spinal loop properties. The V-wave measures although not significantly different between the experimental (-36.7%) and control session (-17.1%), showed a trend towards greater decrease following the SAFT, suggesting a greater decline in supra-spinal output in the experimental session.

Maximal force

In the experimental session, the maximal force was reduced by -13.7% at the end of the simulated soccer match, while the maximal force remained relatively stable (-1.1%) in the control session. However, there were no significant differences or interactions between the two sessions, which may be due to large variability in the data. To date only Nybo et al. (2013) have measured plantar flexor fatigue following soccer match play. Nybo et al. (2013) showed a tendency towards a -5% decrease in

maximal torque as measured 30 min following soccer match play, although it failed to reach significance. The lower reduction in maximal torque in the study by Nybo et al. (2013) may be explained by discrepancies between studies. Nybo et al. (2013) measured maximal torque 30 min after the termination of the soccer match, allowing time for recovery. This is in contrast to the present study, where maximal force was measured immediately at the termination of the simulated soccer match. Additionally, Nybo et al. (2013) used elite soccer players, contrary to the amateur soccer players used in the present study. Other studies investigating plantar flexor fatigue following intermittent running (90 min, Racinais et al., 2007), five km running (~18 min, Girard et al., 2012), continuous running (90 min, Racinais et al., 2007) and prolonged running (two hours, Saldanha et al., 2008) have shown decreases in maximal torque between -11% to -27%. However, it is difficult to compare results between studies, due to large variations in the protocols used to induce fatigue. For instance, Girard et al. (2012) showed a -27% reduction in maximal torque following a five km run, performed as fast as possible. The nature of the fatigue protocol used by Girard et al. (2012) is different from the $SAFT^{90}$ used in the present study. The $SAFT^{90}$ is intermittent in nature, where participants spend ~49 min walking at 5.0 km h^{-1} and ~40 min running at different speeds (10.3 to ≥20.4 km h^{-1}) (Small et al., 2009), allowing recovery between intense bouts. The difference in fatigue protocols may explain the different reductions in maximal strength between the present study and the abovementioned studies.

Muscle activation

In the present study a tendency towards a decrease in VA occurred over time, where VA was decreased by -4.4% after the $SAFT^{90}$ in the experimental session and by -1.6% from pre to post values in the control session, although not significant. In comparison, Nybo et al. (2013) showed a reduction of -1.6% in VA 30 min following soccer match play, indicating the presence of central fatigue. However, Nybo et al. (2013) tested VA following two soccer matches in different climate conditions (Hot vs. temperate) and did not use any control group. The -1.6% decrease in VA found by Nybo et al. (2013) is equivalent to the reduction seen in the control session in the present study. This could suggest that the

decrease in VA seen by Nybo et al. (2013) is not due to fatigue induced by the two soccer matches, implying that soccer match play may not induce central fatigue. However, the low decrease in VA observed by Nybo et al. (2013) could also be a result of the 30 min time period from the end of the match to the measurement of VA, which could allow time for recovery. In contrast, a non-significant -4.4% reduction in VA was observed immediately following the SAFT^{90} in the present study, which might suggest that the time period from the end of the match to the measurement of VA has an influence. When measured directly following simulated soccer match play, the results of the present study suggest that central fatigue may occur. Furthermore, the lack of a significant reduction in the present study may be attributed to a high variability in data, as well as the low number of participants. Nybo et al. (2013) tested 17 participants compared to the seven participants in the present study, which may suggest that a greater number of participants are needed in the present study to reduce the variability. Racinais et al. (2007) employed an intermittent and continuous running protocol of a duration of 90 min, which is comparable to the running profile and duration of the SAFT^{90} employed in the present study. Racinais et al. (2007) did not observe any significant reductions in VA with a sample size of 11 participants, which further imply a need for a greater number of participants in the present study.

The maximal sEMG of SOL (RMS/M_{MAX}) did not change over time and was not different between sessions. However, when looking at the percent change in the experimental and control session, a large reduction (-35.9%) was observed in the experimental session and an increase (6.7%) in the control session. The large variability in data and the low number of participants may thus have masked any significant differences. No studies have explored how and if maximal sEMG of SOL changes as a result of plantar flexor fatigue in soccer. Based on other studies (Girard et al., 2011; Girard et al., 2012; Racinais et al., 2007) investigating the effect of fatigue on maximal sEMG of SOL (RMS/M_{MAX}), a reduction was expected. Racinais et al. (2007) showed a -11% reduction following 90min of both intermittent and continuous running. Similarly, Girard et al. (2012) showed a -13% reduction following five km running, while Girard et al. (2011) showed a greater reduction of -26.2% following

90min of tennis match play. Firstly, the fatigue protocols employed by Girard et al. (2011 & 2012) and Racinais et al. (2007) are different from the SAFT^{90}, as e.g. Girard et al. (2012) used a five km running event to induce fatigue. As previously mentioned, the five km running was performed continuously and as fast as possible, while the SAFT^{90} is intermittent in nature and allows for recovery between intense bouts. In contrast, the intermittent fatigue protocol employed by Racinais et al. (2007) is somewhat comparable to the SAFT^{90}. However, the intermittent protocol is performed on a treadmill, while the SAFT^{90} includes multidirectional and utility movements, which could imply that the SAFT^{90} is more demanding than the intermittent protocol employed by Racinais et al. (2007). Taking the differences in the employed fatigue protocols into account, comparisons between studies are difficult. Secondly, the three abovementioned studies (Girard et al., 2011; Girard et al., 2012; Racinais et al., 2007) tested a greater number of participants (11 to 12 participants), which could explain the lack of a significant decrease in maximal sEMG of SOL with seven participants in the present study.

Control twitch

Peripheral fatigue was present as a result of the 90 min of simulated soccer match play. This was seen as a -23.4% reduction in the peak force of the control twitch from the start of the match to half-time, and a tendency towards a -12.6% decrease from the start of the match to the end of the match. Peripheral fatigue was expected based on the study by Nybo et al. (2013), as they showed a decrease in the peak torque of an evoked twitch 30 min following soccer match play, which remained reduced 24 and 48 hours after the match. Similarly, peripheral fatigue of the plantar flexors has been observed following 90 min of intermittent and continuous running (-10.1%, Racinais et al., 2007), five km running (-16%, Girard et al., 2012) and three hours of tennis match play (-10.6%, Girard et al., 2011). The reduction in peak force of the control twitch in the present study and thus peripheral fatigue, implicates an impairment of excitation-contraction coupling mechanisms. The peripheral fatigue might be a result of impairments in the release and reuptake of calcium from the sarcoplasmic reticulum and the interaction of calcium with the actin-myosin contractile apparatus, which may lead to less forceful contractions (Brooks et al., 2005). Additionally, the release rate of calcium from the sarcoplasmic

reticulum may be impaired by low glycogen levels (Ørtenblad et al., 2011), which has been shown to be decreased following soccer match play (Bangsbo et al., 2006; Mohr et al., 2005).

Spinal loop modulation

To our knowledge, this is the first study to address the effect of fatigue on the modulation of the H-reflex in a dynamic task. The dynamic H-reflex was reduced by ~29% from T_0 to T_{90} in both the experimental and control session, although not significant. This may indicate that the dynamic H-reflex is not affected by fatigue. However, due to large variability in the data, more participants are needed to fully understand the impact of fatigue. Additionally, more studies on the impact of fatigue on the dynamic H-reflex are needed, as static situations does not reflect the actual movement patterns in soccer and sports in general. Injuries also occur during dynamic situations in soccer (Woods et al., 2003), which further implicates the need for investigations into this aspect.

In the present study no differences were observed in the static H-reflex. However, there was a tendency towards a decrease (-10.4%) in the experimental session, and more participants are thus needed to evaluate this tendency. The observed tendency in the present study may be explained by a decrease in motor neuron excitability of the α-motor neuron pool or an increase in pre-synaptic inhibition of Ia afferent synapses. (Aagaard et al., 2002) A decrease in the static H-reflex was expected, as studies have shown decreases in the H-reflex evoked during MVCs following five km running (-46%, Girard et al., 2012) and tennis match play (-48%, Girard et al., 2011) and at rest following intermittent and continuous running (-61%, Racinais et al., 2007). However, there are methodical differences in how H-reflexes are evoked between studies. Besides the different fatigue protocols, H-reflexes were evoked during sub-maximal contractions and on the ascending part of the H-reflex recruitment curve in the present study, whereas Girard et al. (2011 & 2012) evoked H-reflexes during MVCs at H_{MAX} intensity. More research is thus needed to evaluate how the spinal loop is affected by fatigue during soccer match play.

There was a tendency towards a greater decline in V-waves in the experimental session (-36.7%) as compared to the control session (-17.1%). A decline in the normalized amplitude of the V-wave may reflect a decrease in the level of efferent neural drive from spinal α-motor neurons during maximal contractions (Aagaard et al., 2002). Additionally, the V-wave may reflect changes in reflex excitability and pre-synaptic inhibition of Ia afferents (Aagaard et al., 2002). Thus, the results indicate a greater decline in the supra-spinal output and/or changes in reflex excitability and pre-synaptic inhibition in the experimental session compared to the control session. However, more participants are needed to confirm this tendency.

Conclusion

In conclusion, this study has shown that 90 min of simulated soccer match play causes fatigue of the plantar flexors, seen as decreases in maximal force. This decline was accompanied by peripheral fatigue, as indicated by a decrease in the peak force of the control twitch. Furthermore, our results show tendencies towards central fatigue, which was seen as non-significant reductions in VA and V-waves. Lastly, it was not possible to see any indications of a modulation of spinal loop properties, as defined from changes in the amplitude of the H-reflex. It is recommended that more participants are included to address whether central fatigue of the plantar flexors occurs as a result of simulated soccer match play.

REFERENCES

Aagaard, P., Simonsen, E. B., Andersen, J. L., Magnusson, P., & Dyhre-Poulsen, P., (2002). Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. Journal of Applied Physiology. Vol. 92, No. 6, p. 2309-2318.

Bangsbo, J., Mohr, M., & Krustrup, P., (2006). Physical and metabolic demands of training and matchplay in the elite football player. Journal of Sports Sciences. Vol. 24, No. 7, p. 665-674.

Brooks G. A., Fahey T. D., & Baldwin K. M., (2005). Exercise Physiology: Human Bioenergetics and Its Applications. 4th edition, [New York], McGraw-Hill.

Ekstrand, J., Hägglund, M., & Waldén, M., (2011). Epidemiology of Muscle Injuries in Professional Football (Soccer). The American Journal of Sports Medicine. Vol. 39, No. 6, p. 1226-1232.

Fédération Internationale de Football Association (FIFA), (2006). FIFA Big Count 2006: 270 million people active in football. Available: http://www.fifa.com/mm/document/fifafacts/bcoffsurv/bigcount.statspackage_7024.pdf. Accessed May 27, 2014.

Gandevia, S. C., (2001). Spinal and Supraspinal Factors in Human Muscle Fatigue. Physiological Reviews. Vol. 81, No. 4, p. 1726-1771.

Girard, O., Millet, G. P., Micallef, J. P., & Racinais, S., (2012). Alteration in neuromuscular function after a 5 km running time trial. European Journal of Applied Physiology. Vol. 112, No. 6, p. 2323-2330.

Girard, O., Racinais, C., Micallef, J. P., & Millet, G. P., (2011). Spinal modulations accompany peripheral fatigue during prolonged tennis playing. Scandinavian Journal of Medicine & Science in Sports. Vol. 21, No. 3, p. 455-464.

Gregson, W., Drust, B., Atkinson, G., Salvo, V., (2010). Match-to-match variability of high speed activities in premier league soccer. International Journal of Sports Medicine. Vol. 31, p. 237-242.

Hawkins, R. D., & Fuller, C. W., (1999). A prospective epidemiological study of injuries in four English professional football clubs. British Journal of Sports Medicine. Vol. 33, No. 3, p. 196-203.

Hawkins, R. D., Hulse, M. A., Wilkinson, C., Hodson, A., & Gibson, M., (2001). The association football medical research programme: an audit of injuries in professional football. British Journal of Sports Medicine. Vol. 35, No. 1, p. 43-47.

Junge, A., & Dvorak, J., (2004). Soccer Injuries: A Review on Incidence and Prevention. Journal of Sports Medicine. Vol. 34, No. 13, p. 929-938.

Lovell, R., Knapper, B., & Small, K., (2008). Physiological responses to SAFT90: a new soccer-specific match simulation. Verona-Ghirada Team Sports Conference Proceedings, June 2008.

Mohr, M., Krustrup, P., & Bangsbo, J., (2005). Fatigue in soccer: A brief review. Journal of Sports Sciences. Vol. 23, No. 6, p. 593-599.

Nordlund, M.M., Thorstensson, A., & Cresswell, A.G., (2004). Central and peripheral contributions to fatigue in relation to level of activation during repeated maximal voluntary isometric plantar flexions. Applied Physiology. Vol.96, p.218-225.

Nybo, L., Girard, O., Mohr, M., Knez, W., Voss, S., & Racinais, S., (2013). Markers of Muscle Damage and Performance Recovery after Exercise in the Heat. Medicine & Science in Sports & Exercise. Vol. 45, No. 5, p. 860-868

Racinais, S., Girard, O., Micallef, J. P., & Perrey, S., (2007). Failed Excitability of Spinal Motoneurons Induced by Prolonged Running Exercise. Journal of Neurophysiology. Vol. 97, p. 596-603.

Rampinini, E., Coutts, A., Castagna, C., Sassi, R., & Impellizzeri, F., (2007). Variation in top level soccer match performance. Internatiol Journal of Sports Medicine. Vol. 28, p. 1018-1024.

Saldanha, A., Nordlund Ekblom, M. M., & Thorstensson, A., (2008). Central fatigue affects plantar flexor strength after prolonged running. Scandinavian Journal of Science and Medicine in Sports. Vol. 18, No. 3, p. 383-388.

Schmikli, S. L., Backx, F. J. G., Kemler, H. J., & Mechelen, W. V., (2009). National Survey on Sports Injuries in the Netherlands: Target Populations for Sports Injury Prevention Programs. Clinical Journal of Sports Medicine. Vol. 19, No. 2, p. 101-106.

Shield, A., & Zhou, S., (2004). Assessing Voluntary Muscle Activation with the Twitch Interpolation Technique. Sports Medicine. Vol. 34, No. 4, p. 253-267.

Sidhu, S. K., Bentley, D. J., & Carroll, T. J., (2009). Locomotor exercise induces long-lasting impairments in the capacity of the human motor cortex to voluntarily activate knee extensor muscles. Journal of Applied Physiology. Vol. 106, p. 556-565.

Small, K., McNaughton L. R., Greig M., & Lovell, R., (2010). The effects of multidirectional soccer-specific fatigue on markers of hamstring injury risk. Journal of Science and Medicine in Sport. Vol. 13, No. 1, p. 120-125.

Small, K., McNaughton, L. R., Greig, M., Lohkamp, M., & Lovell, R., (2009). Soccer Fatigue, Sprinting and Hamstring Injury Risk. International Journal of Sports Medicine. Vol. 30, p. 573- 578.

Towlson, C., Midgley, A. W., & Lovell, R., (2013). Warm-up strategies of professional soccer players: practitioners' perspectives. Journal of Sports Sciences. Vol. 31, No. 13, p. 1393-1401.

Woods, C., Hawkins, R., Hulse, M., & Hodson, A., (2003). The Football Association Medical Research Programme: an audit of injuries in professional football: an analysis of ankle sprains. British Journal of Sports Medicine. Vol. 37, No. 3, p. 233-238.

Ørtenblad, N., Nielsen, J., Saltin, B., Holmberg, H. C., (2011). Role of glycogen availability in sarcoplasmic reticulum Ca2+ kinetics in human skeletal muscle. The Journal of Physiology. Vol. 589, No. 3, p. 711–725.

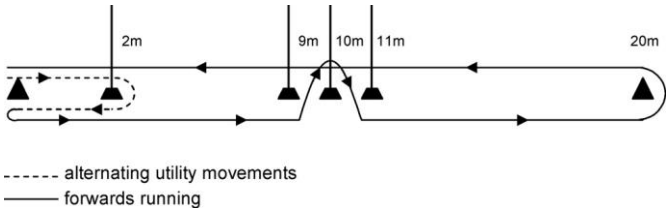
SUPPLEMENTAL MATERIAL

INDEX

SOCCER-SPECIFIC AEROBIC FIELD TEST

The activity profile of the 90 min soccer-specific aerobic field test (SAFT^{90}) is developed based on time-motion data from English Championship level. (Lovell et al., 2011; Small et al., 2009a) The test is based on the average activity of all outfield players, and it is therefore not specific to one position. The SAFT^{90} mimics the fatigue response of soccer match-play, as the activity profile and physiological demands of match-play is comparable to actual soccer match-play. (Lovell et al., 2008) The agility course is 20 m long and based on a shuttle run that incorporates four poles, which the participants has to navigate around (Figure 1).

Figure 1. An overview of the 90min soccer-specific aerobic field test course. (Small et al., 2010)



The participants navigate around the first pole by either sidestepping or forwards and backwards running, and then continue forwards through the three middle poles. (Small et al., 2009b) The activity profile proceeds for 15 minutes and is repeated six times to simulate 90 minutes of soccer match play. The test consists of sidestepping, cutting, backwards and forwards running and frequent accelerations and decelerations. In total, the test involves 1269 changes in speed and 1350 changes in direction over the 90 min period. To ensure that the intensity and duration of the test is standardized and reproducible, the intensity (walking, jogging, striding or sprinting) and movement activity (forwards and backwards running or sidestepping) is given using verbal signals from an audio file. (Lovell et al., 2011; Small et al., 2009a; Small et al., 2009b) Table 1 shows the distances covered at each activity during the test as compared to match-play data. During the SAFT^{90}, the participants cover a total distance of 10.78 km, with 1.8 km (17%) of the distance being performed at high-speed (15.0 km h^{-1}) (Table 1). (Lovell et al., 2011)

Table 1 depicts the different activity intensities, the distance covered at different intensities and data concerning the distance covered during match-play. (Small et al., 2010)

Activity	Distance during SAFT^{90} (km)	Distance from match-play data (km)
Standing (0.0 km h^{-1})	0	0.02
Walking (5.0 km h^{-1})	3.36	3.60
Jogging (10.3 km h^{-1})	5.58	5.81
Striding (15.0 km h^{-1})	1.50	1.46
Sprinting (≥20.4 km h^{-1})	0.34	0.27
Total distance (km)	10.78	11.08

FATIGUE

Fatigue is frequently described as either an inability to "continue working at a given exercise intensity" (Gandevia, 2001; p. 1732) or as an inability "to maintain the required or expected force" (Gandevia, 2001; p. 1732). However, both of these definitions suggest that there is a specific time point where fatigue would prevent the individual from performing a given task, whereas fatigue might occur almost at the onset of exercise and develop progressively throughout the exercise (Gandevia, 2001). According to Gandevia (Gandevia, 2001), a more accurate definition would be "any exercise-induced reduction in the ability to exert muscle force or power, regardless of whether or not the task can be sustained". (Gandevia, 2001; p. 1732) Several factors has an influence on fatigue, including the age and fitness level of the individual, dietary status, fiber type composition of the muscles of interest and the duration, intensity and characteristics of the exercise (Fitts, 2012). Thus, fatigue is a complex problem that might result from both deleterious alterations occurring in the muscle itself (peripheral fatigue) and from changes occurring in the nervous system (central fatigue) (Fitts, 2012). It may further be difficult to unequivocally determine the factors causing fatigue, as it can result from the effects of multiple factors acting at different sites, which often interact synergistically. (Fitts, 2012) However, Bigland-Ritchie (1984) has identified the following major potential sites of fatigue, which can be divided into central and peripheral sites.

The central sites include;

1. Excitatory input to higher motor centers
2. Excitatory drive to lower motor neurons
3. Motor neuron excitability
4. Neuromuscular transmission

Whereas the peripheral sites include;

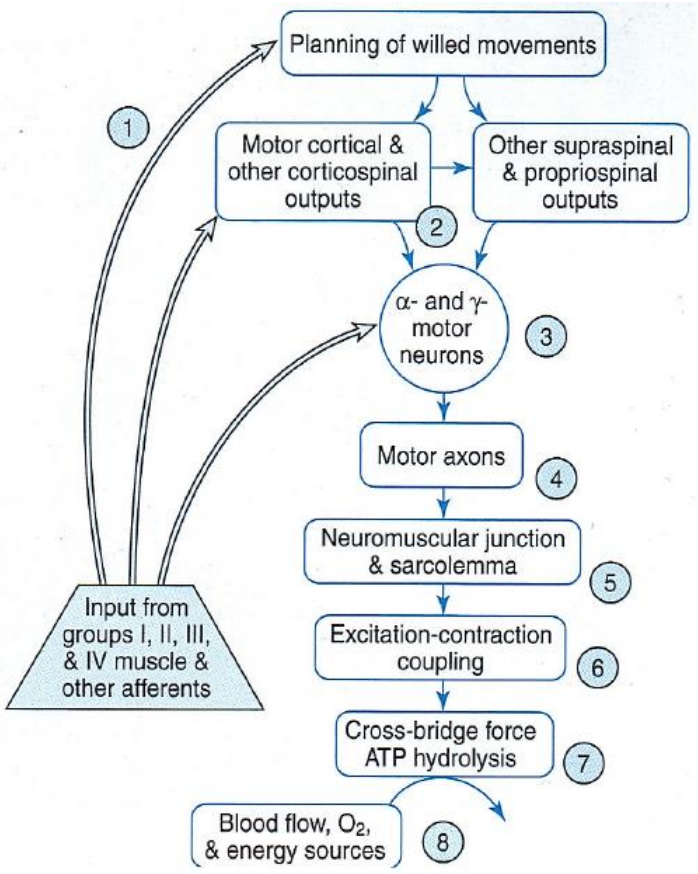
5. Sarcolemma excitability
6. Excitation-contraction coupling
7. Contractile mechanisms

8. Metabolic energy supply and metabolite accumulation

(Bigland-Ritchie, 1984)

The abovementioned sites are depicted in figure 2 and represent all of the steps involved in the production of voluntary force. (Fitts, 2012) Figure 2 further illustrates the potential sites at which central and peripheral factors could mediate fatigue, where number one to four illustrates the sites at which central factors could contribute to fatigue and number five to eight illustrates the sites where peripheral factors could contribute to fatigue.

Figure 2. Illustrates the potential sites of central (number one to four) and peripheral fatigue (number five to eight). The feedback from skeletal muscle is indicated by number one, and acts at three levels of the central nervous system. (Fitts, 2012)



The following sections will focus on the central and peripheral factors that may contribute to the cause of fatigue.

CENTRAL FATIGUE

Central fatigue comprises all supra-spinal and spinal physiological factors that can cause a decrease in motor neuron excitation. According to Boyas & Guével (2011), central fatigue can be defined as "a progressive, exercise induced degradation of the muscle voluntary activation" (Boyas & Guével, 2011; p. 89), where voluntary activation involves the recruitment and increase in firing of motor neurons, and the subsequent translation of motor neuron firing into force production in muscle fibers. Central fatigue may occur at a number of different sites of the central command, and can for instance be caused by muscular afferent fibers and the activity of cerebral neurotransmitters (Boyas & Guével, 2011). Additionally, central fatigue occurs at both the supra-spinal (less optimal output from motor cortex) and spinal level, where the spinal level involves several factors (Gandevia, 2001). Some of the factors at the spinal level comprise the intrinsic behavior of motor neurons and recurrent inhibition and reflex inputs that terminate on α and γ-motor neurons (Gandevia, 2001). The following has been highlighted by Boyas & Guével (2011) as the likely causes of central fatigue, and comprises both supra-spinal and spinal factors.

1. Propagation of axonal action potentials may be blocked at axonal branching sites, inducing a loss of activation of the muscle fiber. The significance of this factor remains to be determined
2. Motor neuron command may be influenced by reflex activities induced by the muscle afferents. Hence, central fatigue could (to some extent) be compensated for by reflexes due to mechanoreceptors (neuromuscular spindles and Golgi tendon organs)
3. The stimulation of type III and IV nerves (chemoceptive and nociceptive afferents) may induce a drop in the motor neuron discharge rate and an inhibition of motor cortex command
4. The excitability of the cells within the motor cortex may vary during a sustained motor task
5. The synaptic effects of serotoninergic neurons could augment and, thus, induce an increase in the sensation of fatigue. This could occur after an increase in the brain's uptake of the serotonin precursor tryptophan. During prolonged exercise, this type of increase could be related to the drop in the plasma concentration of branched chain amino acids

6. Exercise could lead to the release of cytokines such as interleukin-6, which is associated with the sensation of fatigue (proximal to muscle)

(Boyas & Guével, 2011; p. 90)

SUPRA-SPINAL FATIGUE

This section focuses on some of the supra-spinal factors that may cause central fatigue. The decrease in motor cortex excitation that occurs during prolonged exercise may cause a reduction in the output from central command, and thus cause central fatigue. It is further known that neurotransmitters and muscle afferents has an influence on supra-spinal fatigue. (Boyas & Guével, 2011) Other factors that may be linked to supra-spinal fatigue include the depletion and accumulation of certain neurotransmitters in the brain, as well as decreases in descending corticospinal excitation. As mentioned, certain neurotransmitters are proposed to be involved in the development of fatigue, which include dopamine (limit serotonin synthesis), serotonin and catecholamine's (adrenaline and noradrenaline). (Boyas & Guével, 2011) Furthermore, neurotransmitters such as acetylcholine, adenosine, glutamate and gamma-aminobutyric acid [GABA] may also affect the development of fatigue. (Boyas & Guével, 2011) Serotonin is one of the most studied neurotransmitters and is believed to limit central command and thus the recruitment of motor units, by increasing the brain's serotoninergic activity. (Boyas & Guével, 2011) An increase in the serotoninergic activity in the brain may lead to a loss of motivation and lethargy that could reduce the time to exhaustion. (Gandevia, 2001; Boyas & Guével, 2011) Supra-spinal fatigue has further been linked to muscle afferents, which are related to the biochemical status and force generating capacity of the muscles, and it has been suggested that these afferents may limit cortical activity. (Gandevia, 2001; Boyas & Guével, 2011) Additionally, this demonstrates the association between peripheral and central fatigue, as these muscle afferents are sensitive to changes in pH and force changes (peripheral fatigue). (Boyas & Guével, 2011) A change in the muscles biochemical status may as well stimulate group III and IV muscle afferents, which are activated by the extracellular accumulation of potassium (K+) and lactate, hypoxemia and ischemia. Changes in the muscle biochemical status will likely occur with peripheral

fatigue, which in turn may activate group III and IV muscle afferents that could decrease the output to motor neurons and thus cause central fatigue. (Boyas & Guével, 2011) Decreases in the glycogen level of the brain are further thought to mediate central fatigue, as it may affect the serotonin activity, which as previously mentioned might impact on the athlete's perceived workload and motivation. (Boyas & Guével, 2011) However, it should be noted that despite the brains glycogen reserves are small and rapidly exhausted, these reserves are renewed rapidly. Nevertheless, a depletion of glycogen might influence the function of the brain. (Boyas & Guével, 2011)

SPINAL FATIGUE

A number of factors at the spinal level may cause central fatigue, which include decreases in motor neuron activity, intercortical inhibition, inhibition by renshaw cells and inhibition of neural activity by golgi tendon organs. Decreases in motor neuron activity may as well be linked to the accumulation of lactate and K^+, as metaboreceptors (group III and IV muscle afferents) might inhibit the activity of the alpha motor neurons and thus cause fatigue at the spinal level. (Boyas & Guével, 2011)

Golgi tendon organs (group Ib afferents) are mechanoreceptors that provide the central nervous system with feedback about the intramuscular tension, and these receptors are thought to inhibit neural activity. However, because of the difficulties in isolating these afferents, as their interneurons receive signals from both Ib and Ia afferents, little is known about how Golgi tendon organs affect neural activity. Nevertheless, they are believed to influence the development of central fatigue at the spinal level. (Boyas & Guével, 2011) Renshaw cells are another inhibitory mechanism, which influences motor neuron activity. These cells are stimulated by the same motor neurons as they inhibit, as well as by descending and peripheral influences. (Boyas & Guével, 2011) Studies have suggested that the inhibition from renshaw cells is greatest during maximal contractions (increases during the initial 30seconds) as compared to submaximal contractions (inhibition decreases at 20% of MVC), and that this inhibition decreases with the occurrence of central fatigue at both contraction intensities. However, little is known about the inhibition from renshaw cells at different contraction intensities and further research is required. (Gandevia, 2001; Boyas & Guével, 2011)

PERIPHERAL FATIGUE

Peripheral fatigue occurs as a result of factors acting at the neuromuscular junction and within the muscle. (Brooks et al., 2005; Boyas & Guével, 2011; Bigland-Ritchie, 1984) It is often linked to the depletion or accumulation of different metabolites that eventually will lead to a disturbance in the muscle homeostasis and cause fatigue (Brooks et al., 2005; Boyas & Guével, 2011; Bigland-Ritchie, 1984). The exact cause of peripheral fatigue is difficult to determine, as it involves many different concurrently processes, where one system affect others. (Brooks et al., 2005) The following sections will focus on how the depletion and accumulation of key metabolites may influence peripheral fatigue.

DEPLETION OF KEY METABOLITES

The depletion of adenosine tri phosphate (ATP) has been linked to peripheral fatigue (Brooks et al., 2005). However, identifying ATP depletion can be difficult, as ATP might be depleted at certain sites within the muscle, but not elsewhere (Brooks et al., 2005). A possible site for ATP depletion may be on the head of myosin cross bridges. Unfortunately, it is not yet possible to identify depletion at such a specific site and the site of fatigue could thus be masked (Brooks et al., 2005). The following paragraph will focus on the link between ATP and creatine phosphate (CP) depletion, as this is important in order to sustain sufficient ATP levels.

During exercise, ATP levels are maintained at a stable level until the CP level is greatly reduced. At this point, the production of ATP cannot be maintained, and will eventually lead to muscle fatigue. (Brooks et al., 2005) Only small quantities of ATP and CP are stored in the resting muscle and it is thus important that any utilization is matched by a restoration to ensure that exercise can continue (Brooks et al., 2005). Additionally, some studies have observed a relation between the concentration of CP and the development of tension in muscles, implying that as CP depletion occurs it gets increasingly harder to develop tension, which have linked CP depletion to fatigue (Brooks et al., 2005).

Another factor that may influence peripheral fatigue is the availability of glycogen. Glycogen might be depleted in skeletal muscles during exercise and is especially associated with prolonged submaximal exercise. (Brooks et al., 2005) The depletion of glycogen is depended on both the muscle fiber type and

the type of exercise. (Brooks et al., 2005) For instance, slow twitch fibers are more likely to be recruited during work at a low resistance and therefore suffer from glycogen depletion. (Brooks et al., 2005) Because glycogen depletion is work rate depended, muscle fibers can further be depleted unequally. In order to restore the glycogen reserves it is necessary with a rise in epinephrine, as this will stimulate glycolysis, glycogenolysis, production and release of lactate and energy (lactate) exchange via the lactate shuttle. (Brooks et al., 2005)

The amount of stored glycogen and the activity of the hepatic glycogenolytic and glyconeogenic enzymes have an influence on the ability to maintain a high rate of blood glucose release over time. During prolonged exercise, the availability of hepatic glycogen may not be sufficient, which will limit the glucose production to gluconeogenesis. This might be insufficient in order to maintain the required level of glucose to supply the working muscles, and may thus lead to peripheral fatigue.

Accumulation of Metabolites

During short term high intensity exercise, lactic acid accumulates as a result of a higher production than removal. Furthermore, the accumulation of lactic acid in the blood is directly related to the accumulation of hydrogen ions (H^+) in the blood. A part of the energy production during high intensity exercise is derived from glycolysis that produces the byproduct lactic acid, which dissociates H^+. It is the accumulation of H^+ that causes a decrease in the pH level in the muscle, rather than the accumulation of lactic acid itself. Thus, it is the H^+ ion that causes difficulties for the athlete. (Brooks et al., 2005) A lowering of the pH level in the muscle as a result of H^+ accumulation might slow down glycolysis by inhibiting phosphorfructokinase. Additionally, a lower pH level may interfere with muscle contractions by displacing Calcium (Ca^{2+}) from troponin, leading to less forceful contractions. A low pH level may further stimulate pain receptors, which can have an influence on the athletes' ability to continue exercising. (Brooks et al., 2005) However, some studies suggest that a lowering in the pH level is insufficient to cause a cessation of exercise, and argue that it is more likely due to a depletion of CP, which lactate accumulation is a marker of. Another factor proposed to be related to peripheral fatigue is the accumulation of phosphate, which is the result of the depletion of CP. An accumulation of

phosphate has a negative effect on the ability to sustain exercise, as it may act in the same manner as H^+. Phosphate might interfere with excitation-contraction coupling by affecting Ca^{2+} binding to troponin, and can further interfere with glycolysis in the muscles. (Brooks et al., 2005) Furthermore, H^+ and phosphate may combine to produce hydrogen phosphate, which is known to be the most deleterious metabolite to accumulate in muscles. This will further have a negative effect on the ability to sustain exercise. An accumulation of hydrogen phosphate can occur when the muscles are working hard to provide sufficient energy as phosphate gets depleted, and when the muscles are suffering from acidosis. A rise in both H^+ and hydrogen phosphate indicates that the muscles are not working at a steady state, which is due to an inability of the oxidative metabolism to sustain high levels of CP and ATP. Combined with low levels of phosphate this situation will result in imminent fatigue. (Brooks et al., 2005)

Ca^{2+} significantly influences peripheral fatigue as it affects several processes that have an influence on the efficiency of muscle contraction. A loss of Ca^{2+} from the sarcoplasmic reticulum (SR) during excitation-contraction may diffuse into the mitochondria, which could affect the efficiency of the mitochondrial function. High levels of Ca^{2+} may cause an additional O_2 consumption, which can interfere with the phosphorylating of adenosine di phosphate (ADP) to ATP, and thus have implications on the mitochondrial function. (Brooks et al., 2005) Furthermore, as previously mentioned H^+ can interfere with the Ca^{2+} binding to troponin, which will further interfere with the sensitivity of the actin-myosin contractile apparatus to Ca^{2+} and lead to less forceful contractions and thus fatigue. Lastly, the reuptake of Ca^{2+} by the SR could be slowed, which will lead to prolonged contractions and slower relaxation of the muscle. (Brooks et al., 2005)

FATIGUE DURING SOCCER

According to Bangsbo and colleagues (Bangsbo et al., 2006), the cause of fatigue during soccer is a complex phenomenon with several contributing factors. However, as Bangsbo and colleagues (Bangsbo et al., 2006) argue that the cause of fatigue is most likely muscular in nature when players are well-motivated, they have investigated the influence of peripheral factors on fatigue (Bangsbo et

al., 2006). Bangsbo and colleagues (Bangsbo et al., 2006; Mohr et al., 2005) highlights disturbances in muscle ion homeostasis and impaired excitation of the sarcolemma as a possible cause of the temporary fatigue experienced during a soccer match (Mohr et al., 2005), while fatigue towards the end of a match might be caused by low glycogen concentrations in individual muscle fibers (Bangsbo et al., 2006; Mohr et al., 2005). It has further been shown that the concentration of plasma free fatty acids increase towards the end of the game whereas the lactate concentration declines (Bangsbo et al., 2006; Mohr et al., 2005), indicating a shift in intensity and substrate utilization towards the end of the game (Mohr et al., 2005). An increase in the concentration of plasma free fatty acids may increase the serotoninergic activity in the brain, which could cause the player to be less motivated and to experience lethargy, which is a sign of supra-spinal fatigue (Boyas & Guével, 2011). Based on these findings, it could be argued that both central and peripheral fatigue might be present during a soccer match.

ELECTROMYOGRAPHY MEASUREMENTS

In order to perform a muscle contraction and produce force, a number of events have to take place. At first, central nervous system activity initiates a depolarization of motor neurons that is conducted from the motor neuron to the motor end plate of the muscle fibers. At the motor endplate, a chemical substance is released that causes a depolarization of the synaptic membrane, which is also referred to as a muscle action potential. Using recording electrodes, the muscle action potential can be detected as it spreads across the muscle fibers. (Türker & Sözen, 2013) This creates a signal called an electromyogram (EMG) that can be used to evaluate the electrical activity of muscles (Türker & Sözen, 2013). EMG recordings can be acquired by the use of either needles or wires (invasive), which are inserted directly into the muscle, or by using electrodes (non-invasive) that are placed over the skin of the muscle. (Türker & Sözen, 2013; Day, 2013) The latter is denoted surface EMG (sEMG), and as it is non-invasive it is a commonly used method for measuring muscle activity, especially within sport science. Contrary to invasive methods, sEMG does not represent any discomfort or risk to the subject and can further be conducted by personnel other than medical doctors. (Türker & Sözen, 2013; Day, 2013) As sEMG is used to collect EMG data in the current project, the following sections will focus on this method of measuring muscle activity.

The sEMG signal is usable within a frequency range of 0 to 500 Hz, where the main frequency is in the range of 50 to 150 Hz. Furthermore, the amplitude (peak-to-peak) can vary from 0 to 10 mV or 0 to 1.5 mV (root mean squared). (De Luca, 2002) The signal can be influenced by factors such as the placement of electrodes, type of electrodes, preparation of the skin and the properties of the overlying tissue, type of muscle contraction and the amplifier. For instance, the distance from the electrode to the active muscle and the amount of adipose tissue can influence the sEMG signal. (Türker & Sözen, 2013; Day, 2013) Additionally, preparation of the skin is important to ensure optimal contact between skin and electrode. The following paragraphs will focus on how to ensure optimal collection of sEMG signals and different means of signal processing.

COLLECTION OF SEMG SIGNALS

It is important to be aware of any noise that could influence the signal in order to measure and accurately represent a sEMG signal. (Day, 2013) Generally, two types of noise may influence the sEMG signal – that is ambient noise and transducer noise. Ambient noise is caused by the surroundings, while transducer noise is caused by the interaction at the skin-electrode junction. (Day, 2013) Ambient noise arises from the surroundings, where it can be generated by components that are plugged into outlets, such as force plates, computers, lights etc. Noise from such devices has a dominant noise frequency at 50 or 60 Hz, corresponding to the frequency of the A/C power supply. (Day, 2013) Transducer (conversion of ionic currents to an electronic current) noise may be caused by oxidative and chemical reactions occurring at the area of contact between the electrode and the conductive gel causing impedance differences between the skin and electrode. (Day, 2013)

Filters are often used to remove unwanted frequencies that may be a result of the abovementioned noise, and thus ensure signals with minimal noise (a high signal to noise ratio). (Day, 2013) These filters remove some ranges of frequencies, while allowing others to pass. (De Luca, 2001) Generally, three filter types are commonly used, which include a high-pass filter (typically between 10 and 20 Hz), low-pass filter (typically between 500 and 1000 Hz) and a band pass filter. A high-pass filter is used to remove movement artifacts, which is typically less than 10 Hz. Contrary, a low-pass filter removes high frequency components, which ensures that it is possible to distinguish between signals, and thus avoid signal aliasing. (Day, 2013; De Luca, 2001) Additionally, a band pass filter removes both high and low frequencies and attenuates these values towards zero. (De Luca, 2001) Other types of filters include the Butterworth, Thompson/Bessel and Chebyshev. For instance, the Butterworth filter is used to maximally smoothen out pass band frequencies (frequencies that are let through), which will minimize pass band ripple. (De Luca, 2001)

To maximize the signal to noise ratio it is important to amplify the signal, which is done by optimizing the resolution. (Day, 2013) A commonly used method is differential amplification, which can be used to remove the noise signal from power line sources. The method involves detecting the signal at two

sites (bi polar sEMG electrodes), after which the two signals are subtracted from each other and the difference is amplified. When this method is used, all signals that are common to both sites will be removed, whereas signals that are different will have a differential that is amplified. (De Luca, 2002) Thus, this method ensures that signals originating far away from the detection site (noise) are removed, whereas the local EMG signal is amplified. (De Luca, 2002)

PROCESSING OF SEMG SIGNALS

The first step in the processing of EMG signals is often rectification of the signal, which converts the negative amplitudes to positive amplitudes. By doing so, it will be possible to interpret standard amplitude parameters like mean and peak/max (e.g. raw EMG data has a mean value of zero). (Konrad, 2006) Digital smoothing algorithms are often applied to the signal, as the interference pattern of EMG is random of nature, which implies that the raw EMG bursts cannot be reproduced to the exact shape from time to time. These algorithms are used to cut away steep amplitude spikes, and thereby minimizing the non-reproducible part of the signal. Commonly employed smoothing algorithms include a *'moving average'* and *'root mean squared'*. (Konrad, 2006) The moving average is applied within a time window defined by the user, in which the data is averaged using a technique called the gliding window. This technique is also known as the average rectified value when it is used on rectified signals, where it relates to information of the area under the signal. Contrary, the root mean squared calculation is a reflection of the power of the signal, and it is often recommended for smoothing of EMG signals (Konrad, 2006). Because these smoothing algorithms are defined in relation to certain time windows, it is imperative to consider the length of the time window in relation to the type of study. (Konrad, 2006) Lastly, it may be useful to normalize the signal, as it can vary between subjects, electrode sites, and from day to day measures at the same muscle site. For instance, EMG signals can be normalized to a MVC value of a reference contraction. Generally, the purpose of all normalization methods are to remove the influence of a given detection condition. (Konrad, 2006)

TWITCH INTERPOLATION TECHNIQUE

A commonly employed method to investigate central fatigue during voluntary contractions is the twitch interpolation technique (TIT). When supra-maximal stimulation is applied to intramuscular nerve branches or a nerve trunk of an active muscle during voluntary contractions, motor units will respond with a twitch respond in force, if they are not already recruited voluntary. (Shield & Zhou, 2004) This will activate motor neurons that are not in a refractory state and activate motor units firing at sub-maximal rates. When the neural drive to the muscle increases, fewer motor units will be available for recruitment, eventually diminishing the twitch response. If a muscle is fully activated voluntarily, the superimposed twitch response will be undetectable. (Shield & Zhou, 2004) Merton (Merton, 1954) reported a negative linear relationship between the twitch response and voluntary isometric force, during his initial work with the TIT. Based on this, it was concluded that full voluntary activation could be achieved, as there was no twitch response during maximal voluntary contractions (MVC). This was confirmed by other studies, who reported that the majority of healthy individuals were able to fully activate most muscles. However, later studies using more sensitive techniques have reported that full activation is not always achieved. (Shield & Zhou, 2004)

The previous reported linear relationship between voluntary force and the evoked twitch response enables a quantification of the degree of inactivation. This can be calculated by scaling a single interpolated twitch as a percentage of an evoked control twitch in the relaxed muscle (Shield & Zhou, 2004). Thus, voluntary activation is often calculated using the following linear equation (Shield & Zhou, 2004):

$$Voluntary\ activation\ (\%) = \left[1 - \left(\frac{superimposed\ twitch}{control\ twitch}\right)\right] * 100$$

In the equation, the superimposed twitch can be seen as the force increment at the time of the supra-maximal stimulation, while the control twitch is the muscle response evoked 1.5-5 sec after the voluntary contraction. (Shield & Zhou, 2004) Indications of central fatigue can be seen if the

superimposed twitch causes a force increment that exceeds the MVC, reflecting an insufficient drive to produce tetanus in a number of motor units (Sidhu et al., 2009). Contrary, if the amplitude of the control twitch decreases it is an indication of peripheral fatigue. This decrease can be caused by e.g. the accumulation of intramuscular metabolites and disturbances in excitation-contraction coupling (Sidhu et al., 2009). When the cause of central fatigue is investigated using nerve or direct muscle stimulation it is difficult to determine the site of central fatigue, as this might be mediated at several sites proximal to the motor neuron axons, including supra-spinal, brainstem, spinal and reflex circuits (Sidhu et al., 2009). However, methods such as transcranial magnetic stimulation could help determine the site of central fatigue, by providing knowledge regarding the corticospinal cell output to the motor neurons (Sidhu et al., 2009).

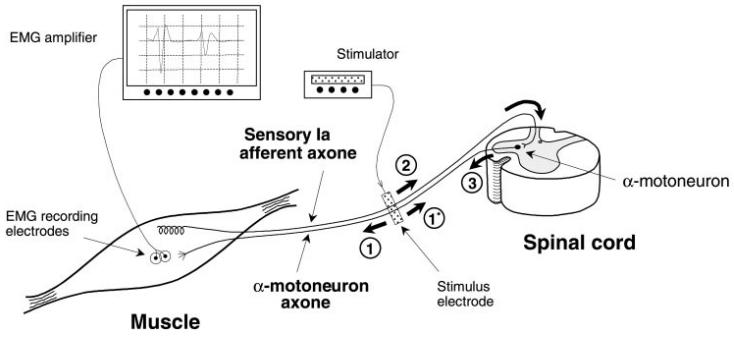
There have been some disagreements as to whether the linear relationship can be used to quantify voluntary activation based on a single interpolated twitch ratio, as some studies have shown both concave upward and asymptotic relationships. (Merton, 1954) The non-linear relationship may be a result of factors such as collisions between antidromic and orthodromic potentials and spinal effects of the stimulation, as well as mechanical factors such as series elastic slackness. (Shield & Zhou, 2004) However, these factors exert their greatest influence during voluntary contractions between 40-80% of MVC, and at forces higher than 80% of MVC the superimposed response is only slightly reduced because of the collision effect.

When careful attention is paid to proper experimental techniques, the scaling of an interpolated twitch response to an evoked control twitch is a valid measurement of voluntary activation according to Shield & Zhou (Shield & Zhou, 2004). To ensure proper experimental techniques in this study, attention was paid to choosing the proper number of interpolated stimuli (twin stimuli separated by 10ms), stimulation intensity (supra-maximal) and familiarizing the participants with performing MVC with and without stimulation. (Shield & Zhou, 2004)

HOFFMANN-REFLEX

The Hoffmann-reflex (H-reflex) is one of the most studied reflexes and is widely used to investigate spinal pathways, as it engages a simple reflex circuit that bypasses both the fusimotor activity and the muscle spindle system, which may influence the sensitivity of the Ia afferents (Knikou, 2008). The H-reflex can be evoked by applying low-intensity electrical stimulation of the Ia afferent nerve fibre (peripheral motor nerve), which will lead to a monosynaptic excitation of α-motoneurons. (Knikou, 2008) The pathways and technique is displayed in Figure 3. The H-reflex has previously been used as a measure of motor neuron excitability, while also reflecting presynaptic inhibition of Ia afferents. (Aagaard et al., 2002)

Figure 3. An illustration of the pathways and parts involved when eliciting an H-reflex. 1) Antidromic and orthodromic volleys from stimulation flowing via the axon. 2) Ia afferent signals running towards the spinal cord. 3) An H-reflex travelling to the muscle. (Aagaard et al., 2002)



If electrical stimulation above motor threshold is applied to a mixed peripheral nerve, two responses in the homonymous muscle are produced. (Knikou, 2008) The first response is a short-latency motor response that results from stimulation of the motor axons - this response is called an M-wave (Figure 4C). The second response is an H-reflex, which is absent at supra-maximal stimulation intensities, due to collisions between antidromic and orthodromic volleys. (Knikou, 2008) At low stimulation intensities only the H-reflex is visible, due to the lower threshold of Iα fibers compared to the motoaxons (Figure 4A). Furthermore, these two responses do not recruit the same α-motoneurons, which leads to the recruitment from small (easily excited) to larger (less excitable) motor neurons. (Knikou, 2008) The recruitment from small threshold α-motoneurons to larger can be seen in the recruitment curve (input-output curve), as seen in Figure 5.

Figure 4. An illustration of three different responses to nerve stimulation. A) Low intensity stimulation eliciting an H-reflex. B) Submaximal stimulation eliciting both an M-wave and an H-reflex. C) Maximal stimulation eliciting only an M-wave. (Aagaard et al., 2002)

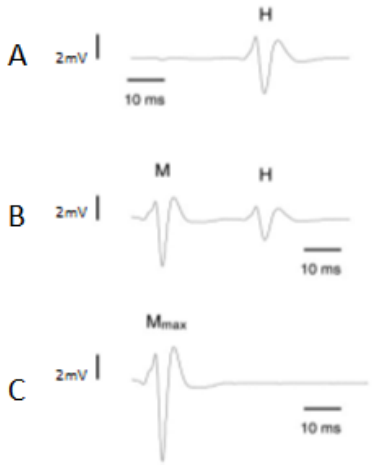
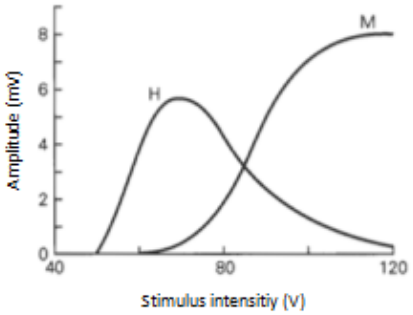


Figure 5. Illustration of an input-output curve, where the H—reflex response is marked with an H and the M-wave with an M. (Kandel et al., 2000)



Many factors can affect the size of the H-reflex and the mechanisms affecting the H-reflex, and it is therefore important to control these factors as much as possible, when H-reflexes are collected. When investigations are conducted on the effect of fatigue on the H-reflex, it is important to ensure that the amplitude of the M-wave remains stable, as the size of the M-wave can be used to ensure that the modulation of the reflex is due to mechanisms acting to facilitate or depress the H-reflex and not due to changes in the test afferent volley. (Knikou, 2008) It is further important to ensure that the level of motoneuron excitability is stable, which can be done by recording H-reflexes during voluntary muscle contractions. When a stable level of motoneuron excitability is ensured, it will further minimize postsynaptic influences. (Knikou, 2008) However, when H-reflexes are recorded during voluntary

contractions it is likely to be affected by a decreased presynaptic inhibition, changes in recurrent inhibition, decreased Ib inhibition, contraction-associated sensory feedback and motoneuron excitability due to descending excitation. The H-reflex should further be elicited on the ascending part of the recruitment curve to avoid recurrent and Ib inhibitory pathways as well as oligosynaptic inputs that can affect the response. (Pierrot-Deseilligny et al., 1981) Lastly, if consecutive H-reflexes are elicited at short inter-stimulus intervals of 1-2 sec, the depression is dramatic. However, this depression decreases progressively as the inter-stimulus interval increases, and vanishes completely after 10 sec. (Knikou, 2008)

INHIBITION OF THE H-REFLEX

In order to successfully execute a motor task, various sensory feedback should be controlled, which can be done through disfacilitation or inhibition. The continuous afferent input from muscles, tendons, skin and joints is controlled through these forms of inhibition. (Knikou, 2008) The following sections describe some of the inhibitory mechanisms that can influence the H-reflex.

PRESYNAPTIC INHIBITION

Presynaptic inhibition may effectively control sensory feedback from the periphery, as this inhibition occurs at the synapses of afferent terminals on the motor neurons. During a motor task, presynaptic inhibition can change the reflex amplitude and is associated with modulation of monosynaptic reflexes. (Knikou, 2008) It has been shown in the cat that pre synaptic inhibition can be caused by axo-axonal gamma-aminobutyric (GABA) synapses that depolarize primary afferents, and consequently these GABA synapses decrease the size of the presynaptic impulse, which will lead to a reduced monosynaptic transmission of the Ia excitatory effects (Knikou, 2008). The modulation of the soleus H-reflex during passive ankle dorsiflexion, standing and leg movements has been related to changes in the amount of presynaptic inhibition, which act on Ia afferent terminals. Furthermore, presynaptic inhibition has also been related to the modulation of the soleus H-reflex amplitude during walking and running. (Knikou, 2008)

RECIPROCAL INHIBITION

During human movements, a reciprocal activation pattern between an agonist and corresponding antagonist is essential to ensure fluent movements. Reciprocal inhibition is influenced by neural pathways that include the Ia afferents. These pathways inhibit the antagonist motor neurons, which ensure an inhibition of the antagonist muscle. (Knikou, 2008) The neural pathways may involve an Ia inhibitory interneuron that is excited by group II, III and IV muscle afferents, as well as the flexion reflex and vestibulospinal, rubrospinal and corticospinal tracts. Reciprocal inhibition may be initiated by supra-spinal mechanisms as well. This can for instance be seen during the transition from dorsiflexion to plantar flexion, where the muscle contraction in tibialis anterior is inhibited 50 ms prior to the initiation of the plantar flexion. (Knikou, 2008)

IB INHIBITION

Ib afferents from Golgi tendon organs can cause Ib inhibition, which may be used to both inhibit synergists motor neurons and to facilitate antagonists motor neurons. It has further been shown that the information from Ib afferents in a single muscle almost reaches all motor nuclei of the particular limb. (Knikou, 2008) For instance, in the ankle extensors this form of inhibition may play an important role during the loco motor cycle, where timing of the different phases is important.

RECURRENT INHIBITION

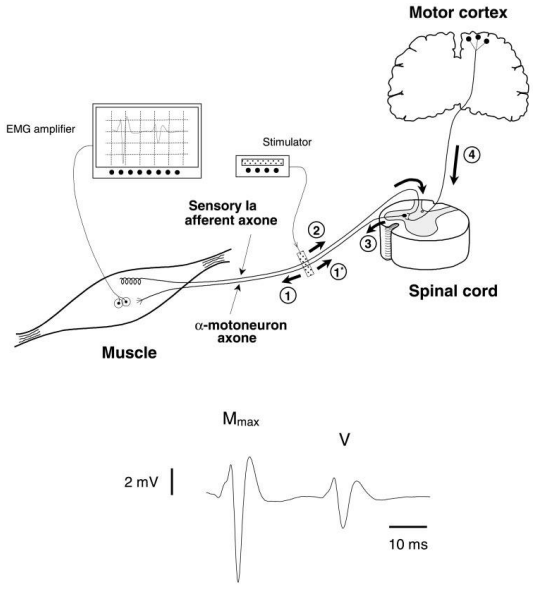
The recurrent inhibition of α-motoneurons that project to the same or the synergist muscle, is partly due to renshaw cells, which are excited by axon collaterals from motor neurons. Recurrent inhibition does not follow a stereotype pattern but is adaptable to the situation, as a number of segmental reflex pathways are involved in this form of inhibition. (Knikou, 2008) Recurrent inhibition plays an important role in the neural control of movements, as recurrent inhibition restricts motor neuron discharge. A number of mechanisms are involved in the restriction of motor neuron discharge, which for instance is done by stabilizing the discharge frequency from tonically firing motor neurons, by inhibiting motor neurons to slow twitch fibers during rapid muscle contractions. Furthermore, a

restriction of motor neuron discharge can be done through synchronization of motor neuron discharge rate and increasing short-term synchronization of α-motoneuron discharges. (Knikou, 2008)

VOLITIONAL-WAVE

The volitional wave (V-wave) is the electrophysiological variant of the H-reflex and can be evoked during MVCs with supra-maximal nerve stimulation. (Aagaard et al., 2002; Solstad et al., 2011) When a MVC is performed, the orthodromic action potentials resulting from the activation of motor neurons by descending neural drive will collide with the antidromic action potentials produced by the stimulation of the motor axons. (Solstad et al., 2011) This results in a cancellation of the two signals allowing the H-reflex volley to passage the muscle, where it can be recorded as a V-wave. (Aagaard et al., 2002) This is shown in Figure 6.

Figure 6. An illustration of the pathways and parts involved when eliciting a V-wave. Activation of motor neurons from descending pathways (number 4 to 3) collide with antidromic potentials (number 1), which allows parts of the evoked reflex response to pass to the muscle (number 2 to 3), which is denoted a V-wave. (Aagaard et al., 2002)



The V-wave can be used as a measure of efferent neural drive, while also reflecting H-reflex excitability, which includes presynaptic and postsynaptic inhibition as well as motor neuron excitability. This implies that the H-reflex and V-wave are affected by the same neural mechanisms. However, the H-reflex is more sensitive to spinal mechanisms (presynaptic inhibition and motor

neuron excitability), while the V-wave is more sensitive to supra-spinal mechanisms (input to the motoneural pool). (Vila-Chã et al., 2012)

REFERENCES

Aagaard, P., Simonsen, E.B., Andersen, J.L., Magnusson, P., & Dyhre-Poulsen, P., (2002). Neural adaption to resistance training: changes in evoked V-wave and H-reflex responses. Journal of Applied Physiology. Vol. 92, p. 2309-2318.

Bangsbo, J., Mohr, M., & Krustrup, P., (2006). Physical and metabolic demands of training and matchplay in the elite football player. Journal of Sports Sciences. Vol. 24, No. 7, p. 665-674.

Bigland-Ritchie, B., (1984). Muscle fatigue and the influence of changing neural drive. Clinics in Chest Medicine. Vol. 5, No. 1, p. 21-34.

Boyas, S., & Guével, A., (2011). Neuromuscular fatigue in healthy muscle: Underlying factors and adaptation mechanisms. Annals of Physical and Rehabilitation Medicine. Vol. 54, No. 2, p. 88-108.

Brooks G. A., Fahey T. D., & Baldwin K. M., (2005). Exercise Physiology: Human Bioenergetics and Its Applications. 4th edition, [New York], McGraw-Hill.

Day, S., (2013). Important Factors in Surface EMG Measurement. Bortec Biomedical Ltd. Available: http://www.andrewsterian.com/courses/214/EMG_measurement_and_recording.pdf. Accessed May 23, 2014.

De Luca, C.J., (2002). SURFACE ELECTROMYOGRAPHY: DETECTION AND RECORDING. DelSys Incorporated.

De Luca, G., (2001). Fundamental Concepts in EMG Signal Acquisition. DelSys Incorporated.

Fitts, R.H., (2012). Chapter 6 – The muscular System: Fatigue Processes in: Farrell, P.A., Joyner, M.J., & Caiozzo, V.J., (2012). ACSM's Advanced Exercise Physiology. 2th edition, Lippincott Williams & Wilkins.

Gandevia, S. G., (2001). Spinal and Supraspinal Factors in Human Muscle Fatigue. Physiological Reviews. Vol. 81, No. 4, p. 1725-1789.

Kandel, E.R., Schwartz, J.H. & Jessell, T.M., (2000). Principles of Neural Science. 4th edition, McGraw-Hill.

Knikou, M., (2008). The H-reflex as a probe: Pathways and pitfalls. Journal of Neuroscience Methods. Vol. 171, No. 1, p. 1-12.

Konrad, P., (2006). The ABC of EMG – A Practical Introduction to Kinesiological Electromyography. Noraxon INC. USA. Available: http://www.noraxon.com/docs/education/abc-of-emg.pdf?sfvrsn=0. Accessed May 23, 2014.

Lovell, R., Knapper, B., & Small, K., (2008). Physiological responses to SAFT90: a new soccer-specific match simulation. Verona-Ghirada Team Sports Conference Proceedings, June 2008.

Lovell, R., Midgley, A., Barrett, S., Carter, D., & Small, K., (2011). Effects of different half-time strategies on second half soccer-specific speed, power and dynamic strength. Scandinavian Journal of Medicine & Science in Sports. Vol. 23, No. 1, p. 105-113.

Merton, P. A., (1954). Voluntary strength and muscle fatigue. Journal of Physiology. Vol. 123, p. 553-564.

Mohr, M., Krustrup, P., & Bangsbo, J., (2005). Fatigue in soccer: A brief review. Journal of Sports Sciences. Vol. 23, No. 6, p. 593-599.

Pierrot-Deseilligny, E., Bergego, C., Katz, R., & Morin, C., (1981). Cutaneous depression of Ib reflex pathways to motoneurones in man. Experimental Brain Research. Vol. 42, p. 351-361.

Shield, A., & Zhou, S., (2004). Assessing Voluntary Muscle Activation with the Twitch Interpolation Technique. Sports Medicine. Vol. 34, No. 4, p. 253-267.

Sidhu, S. K., Bentley, D. J., & Carroll, T. J., (2009). Locomotor exercise induces long-lasting impairments in the capacity of the human motor cortex to voluntarily activate knee extensor muscles. Journal of Applied Physiology. Vol. 106, p. 556-565.

Small, K., McNaughton L. R., Greig M., & Lovell, R., (2010). The effects of multidirectional soccer-specific fatigue on markers of hamstring injury risk. Journal of Science and Medicine in Sport. Vol. 13, No. 1, p. 120-125.

Small, K., McNaughton, L. R., Greig, M., & Lovell, R., (2009) (a). Effect of Timing of Eccentric Hamstring Strengthening Exercises During Soccer Training: Implications for Muscle Fatigability. Journal of Strength and Conditioning Research. Vol. 23, No. 4, p. 1077-1083.

Small, K., McNaughton, L. R., Greig, M., Lohkamp, M., & Lovell, R., (2009) (b). Soccer Fatigue, Sprinting and Hamstring Injury Risk. International Journal of Sports Medicine. Vol. 30, p. 573-578.

Solstad, G.M., Fimland, M.S., Helgerud, J., Iversen, V.M., & Hoff, J., (2011). Test-Retest Reliability of V-Wave Responses in the Soleus and Gastrocnemius Medialis. Journal of Clinical Neurophysiology. Vol. 28, No. 2, p. 217-221.

Türker, H., & Sözen, H., (2013). Surface Electromyography in Sport and Exercise. Electrodiagnosis in New Frontiers of Clinical Research, Associate Prof. Dr. Hande Turker (Ed.). ISBN: 978-953-51 1118-4. Available: http://www.intechopen.com/books/electrodiagnosis-in-new-frontiers-of-clinical-research/surface-electromyography-in-sports-and-exercise. Accessed May 23, 2014.

Vila-Chã, C., Falla, D., Correia, M.V., & Farina, D., (2012). Changes in H reflex and V wave following short-term endurance and strength training. Journal of Applied Physiology. Vol. 112, No. 54, p. 54-63.