Title

Voluntary activation and reflex responses associated with hamstring strain injury

Authors

Buhmann R[1,2], Trajano GS[1], Kerr GK[1], Shield AJ[1].

[1]School of Exercise and Nutrition Sciences and Institute of Health and Biomedical Innovation, Queensland University of Technology, Brisbane, Australia

[2]School of Health and Sport Sciences, University of the Sunshine Coast, Maroochydore, Australia

Corresponding Author

Robert Buhmann

School of Exercise and Nutrition Sciences & Institute of Health and Biomedical Innovation, Queensland University of Technology, Victoria Park Road, Kelvin Grove, 4059, Brisbane, Queensland, Australia.

Email: r.buhmann@qut.edu.au

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Abstract

Introduction: There is a lack of definitive evidence supporting deficits in voluntary activation in participants with prior hamstring injury, moreover it remains unknown if spinal mechanisms contribute to suspected deficits. **Purpose**: To determine the effect of prior hamstring strain injury on knee flexor concentric and eccentric strength, voluntary activation, surface electromyographic activity (sEMG), stretch and tendon reflex amplitudes. **Methods:** Twenty-five participants were recruited, twelve with a history of unilateral hamstring strain injury of at least moderate severity. Voluntary activation, strength and sEMG activity were recorded during maximal eccentric and concentric knee flexor contractions at 60°/s. Stretch and tendon reflexes were also recorded at rest. **Results:** Previously injured limbs exhibited lower levels of voluntary activation (mean difference= -24.1% [95% CI = -34.1 to -14.0%], $p<0.001$) strength (mean difference vs. control group = -0.37 Nm/kg [95%CI = -0.71 to -0.03 Nm/kg], $p = 0.03$) and normalised sEMG (mean difference = -17% [95% CI = -32 to -2%], $p = 0.02$) during maximal eccentric knee flexor contractions compared to control group. No such differences were seen in concentric contractions. Stretch reflexes (mean difference = -3.8% [95% CI = -6.8 to -0.8], $p = 0.02$) and tendon reflexes (mean difference = -13% [95%CI = -26 to 0%], $p = 0.04$) were also lower in previously injured compared to control biceps femoris muscles. **Conclusion:** Moderate to severe hamstring strain injury is associated with long-term deficits in voluntary activation during maximal eccentric contraction. Hamstring injury history is also associated with deficits in stretch reflex and tendon reflex amplitude.

INTRODUCTION

It has been proposed that voluntary activation of the hamstrings is chronically reduced as a consequence of moderate to severe hamstring strain injury (1). It has also been suggested that this deficit in activation may account, at least partly, for preferentially eccentric strength deficits (2, 3) and short hamstring muscle fascicles (4), thereby contributing to high recurrence rates that characterise this injury (1, 5, 6). Preliminary evidence for chronically inhibited hamstrings (up to 24 months) comes from surface electromyography (sEMG) (2, 7, 8) and one functional magnetic resonance imaging (fMRI) study (9). Isokinetic studies have revealed a reduced eccentric to concentric sEMG ratio for biceps femoris long head muscles during maximal voluntary contractions (7), reduced rates of torque development and a slower onset of integrated sEMG activity (2) in athletes with a history of injury to that muscle. Following maximal eccentric exercise, previously injured hamstring muscles also demonstrate smaller changes in transverse relaxation times (T2) assessed via fMRI, which share a direct relationship with exercise intensity (10), leading authors of this study to infer lower levels of voluntary activation in previously injured participants (9). This preliminary evidence suggests previously injured muscles may not receive the required stimulus for strength and fascicle length gains (11) that have been proposed to account for the benefits of eccentric strength training programs (12, 13). However, sEMG and fMRI are limited in their spatial and temporal resolution respectively (14). Twitch interpolation provides a means of assessing the completeness of voluntary activation with high temporal resolution, providing a more valid measure of this parameter than sEMG and fMRI (15, 16). Deficits in voluntary activation have been observed in participants with other injuries, such as anterior cruciate ligament (ACL) reconstruction (17). To date, however, this technique has not been applied to the hamstrings in previously injured athletes.

Determining the presence of, and mechanisms contributing to voluntary activation deficits in participants with prior hamstring injury will assist in restoring these deficits during rehabilitation. Voluntary activation is the net result of facilitatory and inhibitory drive from spinal and supraspinal inputs to the motoneurone pool, with either potentially limiting activation with injury (17, 18). For example, deficiencies in Ia afferent input, which are mediated mainly at the spinal level, have been observed with ACL reconstruction (18). One role of this complex pathway, which comprises the muscle spindle, Ia afferent neurones and spinal alpha motoneurones, is to contract the homonymous muscle in response to stretch (19). Output from this pathway can be assessed through sEMG responses to rapid passive rotations of the joint (stretch reflex) and tendon taps (tendon reflex) (19). While both of these measures are underpinned by similar mechanisms, the stretch reflex contains short and medium latency components due to the discharge of group I and II spindle endings respectively, while tendon reflexes provide a potent stimulus for primary (group 1a) endings (19). Additionally, tendon reflexes assess the Ia afferent pathway in a controlled manner minimizing input from other afferent neurones (for example joint receptors), while stretch reflexes assess this pathway in a dynamic setting that better represents hamstring function during running. Similar findings from both reflexes would provide strong evidence for lower levels of Ia afferent input in participants with prior hamstring injury.

Deficits in voluntary activation and sEMG responses to muscle stretch and tendon taps would, if found, suggest that the Ia afferent pathway may warrant attention in rehabilitation. However, these have, to our knowledge, not been investigated in the context of prior hamstring strain injury. The primary aim of this study was to obtain definitive assessments of voluntary activation using the twitch interpolation technique, in participants with a history of hamstring strain injury. A secondary aim was to assess output from the Ia afferent loop through stretch and tendon tap reflexes. We hypothesized that previously injured hamstrings

would exhibit greater superimposed twitches (and consequently lower levels of voluntary activation) and smaller stretch reflex and tendon tap responses than uninjured groups.

METHODS

Participants

The twenty-five recreationally active males who were recruited for this study participated in sports such as soccer, sprinting, rugby league and rugby union. All were free of major lower limb injury (for example ACL reconstruction) and twelve (22.2 ±2.6 y, 180.3 ±9.7 cm, 79.2 ±10.1 kg) presented with a unilateral history of hamstring strain injury incurred 8.3 ± 6.2 months prior to participation in this study (see Table 1). All reported a complete recovery and had returned to full training and competition schedules. The biceps femoris (n = 9) was the most common site of injury followed by the semitendinosus (n = 3) (see Table 1). Thirteen participants had no history of hamstring strain injury (23.9 ± 4.3 y, 177.0 ± 6.9 cm, 80.0 ± 8.8 kg) and were free of other lower limb injuries. All testing procedures were approved by the Queensland University of Technology Human Research Ethics Committee (approval number: 1600001056) and participants gave written informed consent prior to participating.

Study Design and Overview

Participants visited the laboratory on three occasions, separated by at least one week. The first (familiarisation session) and second sessions involved measures of isokinetic dynamometry and twitch interpolation. The third experimental session was used to assess stretch and tendon reflexes. Prior to all data collection participants performed five minutes of self-paced cycling (50 W) on a Monark bike as a warm up.

Isokinetic dynamometry

Participants attended at least one familiarisation session involving isokinetic dynamometry. These sessions occurred approximately one-week prior to identical data collection sessions. Concentric and eccentric strength of the knee flexors was assessed, for both legs, on a Biodex systems 3 dynamometer (Biodex Medical Systems, Shirley, NY). Knee range of motion was set between 0˚ and 90˚ of knee flexion and participants sat with their hips at ~85˚ of hip flexion. All torque measurements were corrected for limb weight. Participants were secured in the dynamometer with restraints applied to the knee, hips and shoulders. The ankle was secured to the lever arm with the padding on the anterior shin, so participants pulled into the less compliant Velcro strap during concentric and eccentric knee flexor contractions.

As a warm up, participants completed one set of six repetitions of knee extension and flexion at $+240^{\circ}s^{-1}$. Following one-minute rest, another set of six maximal knee extension and flexion repetitions at $+180^{\circ}s^{-1}$ were completed and used for sEMG normalisation. To assess flexor strength, three sets of two maximum voluntary contractions at +60 and $-60^{\circ}s^{-1}$ were completed (twelve repetitions total, six per contraction mode), with one-minute rest separating each set.

Voluntary activation

A constant current stimulator (DS7, Digitimer, Welwyn Garden City, UK) delivered twin electrical stimuli (pulse width 50 μs, inter-pulse interval 10 ms) to the hamstrings via two self-adhesive electrodes (5 cm x 9 cm, Pals Platinum electrode, Axelgaard Manufacturing Co., California). The cathode was placed immediately below the gluteal fold and the anode approximately 5 cm superior to the popliteal fossa on the posterior thigh (20). To determine the stimulus intensity used in assessments of voluntary activation, twin electrical stimuli were applied, at 30º from full knee extension, to the knee flexors during passive knee extension. Stimuli were automatically triggered from the angle output of the isokinetic dynamometer.

Current began at 50 mA and was increased in 25 mA intervals until the maximal twitch response was obtained (confirmed by visual inspection of torque traces).

In each set of maximal concentric and eccentric contractions on the dynamometer, supramaximal stimuli (10% greater than the current generating maximal twitches) were superimposed over one of two efforts. Relaxed muscles were stimulated during passive knee movement in the same direction five seconds after the second voluntary effort, to produce a potentiated resting twitch (see Figure 1). All stimuli were applied at 30° of knee flexion.

To calculate voluntary activation, three torque-time curves for both the eccentric and concentric efforts were averaged. Torque data from the 10° prior to the onset of stimulation was extrapolated, using a second order polynomial function, beyond the peak of the superimposed response. The superimposed response amplitude was then estimated by subtracting the extrapolated voluntary torque from the coinciding peak of the superimposed response (see Figure 1). The superimposed and potentiated resting twitch responses were then substituted into the following equation:

$$\%VA = [1 - (\text{superimposed twitch}/\text{potentiated twitch})] * 100$$

Surface EMG

Surface EMG during isokinetic dynamometry sessions was sampled with a PowerLab26T recording unit (ADInstruments, New South Wales, Australia) at 1KHz, bandpass filtered (20-500Hz), full wave rectified and smoothed across a 100 ms moving window. Bipolar pre-gelled Ag/AgCl electrodes (15 mm inter-electrode distance) were placed over the biceps femoris and medial hamstring muscle bellies, midway between the ischial tuberosity and the knee crease as per SENIAM guidelines. For stretch reflex and tendon tap assessments, electrodes were also placed over the vastus medialis and lateralis to ensure electrical silence

of these muscles during recording. Skin was prepared as per the hamstring muscle group and electrodes were placed at the most prominent portion of the muscle belly along the line connecting the anterior superior iliac spine and the medial/lateral patella for the vastus medialis and lateralis, respectively. The reference electrode was placed over the head of the ipsilateral fibula.

Stretch reflex testing

During session 3, sEMG and force data were sampled at 4 kHz using the same equipment and methods as described above. Surface EMG was assessed during fast hamstring stretches using a modified leg curl machine fitted with a custom made electrogoniometer that provided knee angle data. Participants lay in a standardised prone position (with ~20˚ hip flexion) with the test limb secured to the machine's lever arm. Participants initially performed two four second long maximal isometric knee flexion contractions (at 20˚ of knee flexion), which were used for normalising sEMG stretch reflex responses. The investigator then lifted the machine's lever arm to the end of its range (~90˚ of knee flexion) while participants remained relaxed. The lever arm was then dropped without warning and with variable delay to elicit a rapid hamstring stretch (lever arm load was approximately 2.3 kg, average angular velocity of the lever arm was approximately 210°.s^{-1}) (21). Relaxation of the hamstrings and quadriceps was confirmed by silent sEMG traces before stretches were initiated. Ten responses were recorded from each leg with at least 10 seconds between stretches.

Tendon taps

Responses to 'taps' to the distal biceps femoris tendon were recorded from each leg using a custom-made inverted pendulum apparatus. Reliability of the vertical acceleration at strike was confirmed prior to testing by securing a tri-axial accelerometer to the tendon hammer apparatus (CV= 3.5%). The tendon of each limb was identified by palpation and marked 5

cm superior to the knee crease. The tendon hammer was lifted to vertical and dropped so that it struck the marked position of the intended tendon. Six responses were recorded at rest with approximately 10 seconds rest between taps.

Data Analysis

Three maximal voluntary contractions on the dynamometer were used to determine mean torque and sEMG activity between 15 and 35° of knee flexion during contractions at +/- 60°s^{-1} [5, 7]. Smoothed sEMG was normalised to maximal values (a 100ms average around peak sEMG activity) during knee flexion at +180°s^{-1} and expressed as a percentage ([sEMG during maximal concentric or eccentric efforts at 60°s^{-1} divided by sEMG during contractions at +180°s^{-1}]*100). Concentric and eccentric sEMG activity during contractions at 60°s^{-1} was normalised to concentric sEMG activity at 180°s^{-1} for consistency with other hamstring literature (2, 7).

Stretch reflex latency was defined as the time between the lever falling one degree (observed via goniometry data) and the peak of the smoothed sEMG signal (which was synchronised with goniometry data) of each hamstring muscle. To determine stretch reflex amplitudes, integrated EMG (the area under the rectified signal) recorded during fast hamstring stretches was normalised to two hundred milliseconds of data around the peak integrated EMG activity recorded during isometric knee flexor MVC's. These responses are expressed as a percentage. . Raw peak-to-peak sEMG amplitudes were recorded from tendon taps elicited at rest and normalised to five percent of maximal isometric knee flexor rectified sEMG.

Statistical Analysis

All statistical analyses were conducted using SPSS 22.0 software (SPSS Inc, Chicago, IL), with differences reported as the mean and 95% confidence interval, (CI). Given the potential

for between muscle differences in stretch reflex responses and tendon tap responses, participants with a previous semitendinosus injury (n= 3) were excluded from the comparison between injured and control groups for these variables. The average of the dominant and non-dominant limbs in control participants was calculated for all variables and used for comparison to injured and uninjured limbs of previously injured participants. All variables were assessed for normality using Shapiro-Wilks test and homoscedasticity via Levene's test. Non-normally distributed data were log transformed (stretch reflex responses) for statistical analysis, results were back transformed for reporting. Two way repeated measures analysis of variance (ANOVA) was used to determine statistical differences between limbs (limb × muscle for sEMG comparisons and limb × contraction mode for strength and voluntary activation comparisons), with Bonferroni's post hoc test used to determine statistical difference. Cohen's d values were also determined (calculated as $\sqrt{[(s_1 + s_2)/2]}$, where s= standard deviation) with effect sizes interpreted as small <0.5; moderate 0.5 – 0.8; large >0.8.

RESULTS

Twitch interpolation (percentage voluntary activation)

Descriptive statistics for twitch interpolation data are displayed in Table 2. Voluntary activation in eccentric actions was significantly lower in injured than control groups (mean difference = -24.1% [95% CI = -34.1 to -14.0%], $p<0.001$, Cohen's d = -2.70) and lower in injured than uninjured limbs (mean difference = -10.6% [95% CI = -21.0 to -0.1%], $p = 0.04$, Cohen's d = -1.52). Voluntary activation during concentric knee flexor contractions was not statistically different between control and injured groups (all p's > 0.28, all Cohen's d < -0.61) (Figure 2).

Stretch reflex responses

Stretch reflex responses of the biceps femoris were significantly lower in injured compared to control groups (mean difference = -3.8% [95% CI = -6.8 to -0.8], p = 0.02, Cohen's d = -0.61). There was no difference in stretch reflex responses of the biceps femoris in injured compared to uninjured limbs (mean difference = -1.6% [95% CI = -3.0 to 0.3%], p = 0.07, Cohen's d = -0.30) or between uninjured and control groups (mean difference = 1.6% [95%CI = -0.7 to 3.6%], p = 0.41, Cohen's d = 0.30) (Figure 3). There were no differences in stretch reflex responses of the medial hamstrings between control and injured groups (mean difference = 2.1% [95%CI = -1.6 to 5.8%], p = 0.08, Cohen's d = 0.24) or between injured and uninjured limbs (mean difference = -1% [95%CI = -2.4 to 0.4%], p = 0.18, Cohen's d = -0.14) (Figure 3).

Tendon taps

Tendon tap responses of the biceps femoris at rest were significantly lower in injured limbs compared to control groups (mean difference = -37% [95% CI = -75 to -1%], p = 0.04, Cohen's d= -1.48) (see Figure 3). There were no statistically significant differences in tendon tap responses of the biceps femoris muscle between injured and uninjured limbs (mean difference = -21% [95%CI = -57 to 16%], p = 0.48, Cohen's d = -0.51). There were no statistically significant differences in resting tendon tap responses of the medial hamstrings between injured and control groups (mean difference = -14% [95%CI = -50 to 21%], p = 0.97, Cohen's d = -0.39) or between injured and uninjured limbs (mean difference = 10% [95% CI = -25 to 45%], p = 0.99, Cohen's d = 0.52).

Maximal voluntary contraction torque

Eccentric strength (at $-60^{\circ}s^{-1}$) was significantly lower in injured than control groups (mean difference = -0.37 Nm/kg [95%CI = -0.71 to -0.03 Nm/kg], p = 0.03, Cohen's d = -1.56). There was no statistically significant difference in eccentric strength between injured and

uninjured limbs (mean difference = -0.25 Nm/kg [95%CI = -0.60 to 0.10], p = 0.23, Cohen's d = -0.68). Concentric strength (at $+60^{\circ}s^{-1}$) was not statistically different between injured and control groups (all p's > 0.13, all Cohen's d < -0.27) (Figure 4).

Surface EMG

Normalised sEMG of the biceps femoris during eccentric actions (at $-60^{\circ}.s^{-1}$) was significantly lower in previously injured limbs than control groups (mean difference = -17% [95% CI = -32 to -2%], p = 0.02, Cohen's d = -1.27). There was no difference in normalised sEMG of the biceps femoris during eccentric contractions between injured and uninjured limbs (mean difference = -4% [95%CI = -20 to 11%], p = 0.99, Cohen's d = 0.31). There was no difference in normalised sEMG of the biceps femoris during concentric contractions (at $+60^{\circ}.s^{-1}$) between limbs or groups (all p's > 0.36, all Cohen's d < -0.96).

There were no statistically significant differences in normalised sEMG of the medial hamstrings during eccentric contractions (at $-60^{\circ}.s^{-1}$) between control and injured groups (mean difference = 12% [95%CI = -2 to 26%], p = 0.11, Cohen's d = 0.82) or injured and uninjured limbs (mean difference = 2% [95% CI = -13 to 16%], p = 0.99, Cohen's d = 0.13) There were no differences in normalised sEMG of the medial hamstrings during concentric contractions (at $+60^{\circ}.s^{-1}$) between limbs or groups (all p's > 0.49, all Cohen's d < 0.49).

DISCUSSION

Current literature (2, 7-9) and the results of this study demonstrate an inability of those with prior unilateral hamstring strain injury to completely activate the involved limb, despite full voluntary exertion. It has been suggested that a failure to fully voluntarily activate the previously injured muscle may result in limited gains in eccentric strength and fascicle lengthening during rehabilitation (1). Since this hypothesis was presented (24), there have

been a number of findings that are consistent with it. For example, Opar and colleagues (25) reported that elite Australian footballers with a recent (<12 month) history of hamstring strain injury gained less eccentric strength during preseason training, despite significant emphasis on increasing strength, than players without a recent history of injury. More recently, Timmins and colleagues (26) showed that Australian footballers with a recent history of hamstring injury exhibited shorter biceps femoris fascicles in the preseason period, and less fascicle lengthening as a consequence of pre-season and in-season training than players without such history. It has been suggested that high levels of activation at long muscle lengths is an important stimulus for strength and fascicle length increases (27). Therefore, these limited gains in eccentric strength fascicle lengthening observed in previously injured participants may reflect a failure to activate the muscle sufficiently at during long excursions. Understanding the mechanisms contributing to lower levels of voluntary activation may help create novel interventions leading to greater gains in both of these measures, consequently limiting re-injury risk (5, 6).

Incomplete voluntary activation has been observed in other injuries such as ACL reconstruction, meniscectomy and osteoarthritis (17, 18, 28). It has been proposed that reduced spinal reflex excitability is one of the main contributors to chronic deficits in voluntary activation and strength observed in those with joint injury (18, 28). H-reflexes, which are considered the electrophysiological equivalent of the stretch reflex (19), are lower in the quadriceps of anterior cruciate ligament reconstructed limbs compared to the homonymous contralateral muscle (29). Additionally, smaller H-reflexes are significantly associated with lower levels of knee extensor strength in ACL reconstructed participants (28). Although H-reflexes differ from the stretch and tendon reflex, as the stimulus bypasses the muscle spindle (19), these studies (28, 29) suggest spinal reflex excitability is reduced in ACL reconstructed participants, contributing to lower levels of voluntary activation and

strength. Given stretch and tendon reflexes are indicative of output from the Ia afferent pathway (including the muscle spindle) (19) the findings of the current study also suggest spinal reflex excitability is lowered in participants with prior hamstring strain injury, possibly contributing to lower levels of voluntary activation, and should therefore be targeted during rehabilitation. The importance of Ia afferent input has been demonstrated by the demonstration that motoneuron discharge is reduced when this pathway is blocked with anaesthetic (30). Impaired Ia afferent input may explain the specific deficits in eccentric knee flexor strength observed in limbs with previous hamstring strain injury. Given afferents in this pathway are sensitive to stretch, it appears important for the facilitation of motoneurons during lengthening contractions, while the unloading of muscle spindles limits the discharge of Ia afferents during concentric contractions (31).

There are several mechanisms potentially contributing to lower levels of afferent input and force generation during maximal eccentric contraction in participants with prior hamstring strain injury. Two main presynaptic inhibitory mechanisms for motoneurons exist. Disfacilitation of eccentric actions may occur through homosynaptic post activation depression, where neurotransmitter release at Ia terminals is reduced due to repetitive stimulation, limiting the excitatory output of the Ia afferent pathway (34). There is limited data to support an increase in post activation depression with injury (18) and it is yet to be determined if this mechanism may be involved in hamstring strain injury. Alternatively, primary afferent depolarisation may occur through activation of inhibitory interneurons, which are centrally controlled, presynaptically inhibiting Ia terminals (34). Similarly however, there is limited data available to be able to determine if primary afferent depolarisation is involved in lower levels of motor output in hamstring or other sporting injuries. Future work should employ methods such as tendon vibration or conditioning stimulation protocols to determine the contribution of these mechanisms to

inhibition/disfacilitation of eccentric actions following hamstring injury so that they may be targeted in rehabilitation. Excitability of motoneurones may also be modified several postsynaptic mechanisms, however recurrent inhibition via Renshaw cells may be the most likely (34). Descending pathways may modulate the level recurrent inhibition during eccentric actions, consequently limiting motor unit firing frequencies, however, this also requires further investigation before substantive claims can be made (34). Understanding the mechanisms of inhibition and eccentric weakness may prove valuable in the development of novel hamstring rehabilitation strategies. For example, wide-pulse width electrical stimulation (36) and tendon vibration (37) may be effective in increasing afferent input to the hamstring motoneurone pool.

It is possible that reduced estimates of voluntary activation in injured participants may be due to factors other than limited voluntary drive to the hamstring muscle group (such as differences in potentiated twitch amplitude or muscle-tendon unit stiffness). However, we observed no significant difference in the size of potentiated control twitches between limbs or groups, and greater relative superimposed responses during eccentric efforts in injured compared to the control group. In fact, there was a moderate effect size for potentiated twitches elicited during passive lengthening to be larger in injured than control groups ($p = 0.07$, Cohen's $d = 0.68$). As the potentiated twitch is the denominator in the voluntary activation equation, the effect of a larger potentiated twitch, in injured compared to the control group, would be an increased estimate of voluntary activation of injured limbs. As deficits in voluntary activation were observed in injured compared to the control group this gives us confidence that estimates of impaired voluntary activation are the result of impaired drive during eccentric contraction and not due to other factors. Furthermore, twitch interpolation and biceps femoris sEMG results (from eccentric actions normalised to the sEMG signal from concentric actions at $+180^{0}.s^{-1}$) both demonstrate deficits in injured

compared to the control group, supporting the hypothesis that voluntary drive is limited during eccentric contraction of previously injured limbs.

The current study has some limitations, the first of which is its retrospective design, which makes it impossible to tell whether observed deficits are the cause or result of injury. Secondly, peripheral nerve stimulation is typically used when assessing voluntary activation, as opposed to the muscle belly (nerve branch) stimulation employed here. However, in pilot testing our attempts to stimulate the hamstrings via the sciatic nerve resulted in only very small twitch responses, indicating less complete activation of these muscles than we obtained when stimulating over the muscle belly. Furthermore, muscle and nerve stimulation of the quadriceps produces similar results (38) and the tight match between estimates of voluntary activation, sEMG and strength in eccentric and concentric efforts gives us some confidence in the current results. Previous research has also employed similar methods for hamstring muscle stimulation to examine voluntary activation (20). Additionally, we were unable to use the M-wave for normalisation of reflex measures as this response is obtained via nerve stimulation. However, normalising reflex responses to the sEMG obtained in maximal voluntary contractions has been suggested as a suitable alternative approach (33).

CONCLUSION

Recreational athletes with a history of unilateral hamstring strain injury exhibited reduced voluntary activation, strength and sEMG in eccentric knee flexor actions along with diminished stretch and tendon tap responses. Lower levels of afferent input may contribute to lower levels of voluntary activation in participants with prior hamstring injury and specifically targeting these deficits in rehabilitation may result in improve rehabilitation programs.

AUTHOR CONTRIBUTIONS

Rob Buhmann was supported by the Australian Government Research Training Program Scholarship. Rob Buhmann, Anthony Shield and Gabriel Trajano designed the study, monitored data collection, analysed and interpreted the data and revised the paper. Graham Kerr interpreted the data and revised the paper. The results of the present study do not constitute endorsement by the American College of Sports Medicine. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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FIGURES

Figure 1. A) An example of an eccentric maximal voluntary contraction with a superimposed electrical stimulus and a resting potentiated twitch response. A doublet electrical stimulus (black arrow) was applied 30° from full knee extension. Voluntary torque traces were extrapolated through the stimulus, voluntary activation was calucalted using the equation; %VA= [1 – (superimposed twitch/potentiated twitch)] *100. B) and C) depict a representative stretch reflex responses (grey lines = raw sEMG signal, dark lines = instantaneous velocity of the lever arm) in control and injured biceps femoris muscles respectively. D) and E) depict a representative tendon tap response in control and injured biceps femoris muscles respectively.

Figure 2. Estimates of voluntary activation average torque between 35 and 15° degrees of knee flexion during maximal eccentric and concentric contractions. Error bars represent 95% confidence intervals, *** signifies $p < 0.001$ and * signifies $p < 0.05$. ECC= eccentric, CON= concentric, VA= voluntary activation.

Figure 3. Stretch reflex responses (A) and the responses to distal biceps femoris tendon taps (B) of the lateral and medial hamstrings. The iEMG recorded during stretch reflex responses is normalised to peak iEMG during an MVC and tendon tap responses are normalised to five percent of maximal isometric knee flexor sEMG. Error bars represent 95% confidence intervals, * signifies $p < 0.05$. iEMG= integrated EMG, nEMG= normalised electromyography, SR= stretch reflex, BF= biceps femoris, MH= medial hamstrings.

Figure 4. Average torque between 35 and 15° degrees of knee flexion during maximal eccentric and concentric contractions (A). Average smoothed and rectified surface EMG between 35 and 15° degrees of knee flexion for the lateral (B) and medial (C) hamstrings during maximal concentric and eccentric contractions. Error bars represent 95% confidence

intervals and * signifies $p < 0.05$. ECC= eccentric, CON= concentric, nEMG= normalised electromyography, BF= biceps femoris, MH= medial hamstrings.