

Neuromuscular Inhibition, Hamstring Strain Injury, and Rehabilitation: A Review

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ABSTRACT

Objective: The purpose of this review was to summarize the neuromuscular inhibition of the hamstring and lumbopelvic musculature following a hamstring strain injury (HSI) and its subsequent implications on the rehabilitation of the injury.

Design: Narrative review.

Results: A search of electronic databases and references within the articles found 18 articles suitable for review. A critical review of the literature revealed that despite returning to the sport, athletes demonstrated an impairment in the myoelectric activity in the hamstring muscle group, particularly the biceps femoris (BF). Additionally, altered activation patterns in the lumbopelvic musculature (gluteus medius and maximus, external and internal oblique, thoracic and lumbar erector trunci) of healthy athletes were also associated with increased HSI risk.

Conclusion: Despite athletes completing evidence-based rehabilitation programs, there is persistent neuromuscular inhibition present in the hamstring and lumbopelvic musculature on return to sport. Clinicians should be aware of this phenomenon as a return of athletes to sport with neuromuscular inhibition may increase their risk of hamstring strain re-injury. Measures such as surface electromyography (EMG) may be a useful tool to detect neuromuscular changes following HSI and help guide clinicians in the return to play process following an HSI.

Keywords: Hamstring, Inhibition, Muscle injury, Neuromuscular, Strain injury, Surface electromyography.

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INTRODUCTION

The prevention of HSI and its recurrence remains the Holy Grail of sports medicine. It is the most common muscle injury in Australian rules football (ARF)¹ and soccer² whilst also problematic in American football,³ rugby league,⁴ and rugby union.⁵ Over a period of 10 Australian Football League (AFL) seasons HSIs accounted for an average of 16.5% of all new injuries with a recurrence rate of 20%.⁶ The average yearly financial cost of games missed due to HSIs for each AFL club in 2012 was AUD 245,842⁶ and €250,000 for a player to miss 2 weeks due to injury in elite European soccer clubs.⁷ Compounding the significant financial burden of HSI, is the adverse mental health impact that injury can trigger including depression, anxiety, eating disorders, and substance use.⁸

The majority of hamstring strain injuries affect the long head of the BF muscle due to the consensus belief that peak muscle activation and elongation occur simultaneously, during the late swing phase of high-speed running, in order to decelerate the hip and knee.^{9,10}

The diagnosis and prognosis of muscular injuries have traditionally been based on clinical findings¹¹ including hamstring range of motion,¹² manual muscle resistance testing,¹³ active slumps tests,¹⁴ and palpation.¹² However, with the exception of the “taking off the shoe test,” there is a low diagnostic value observed for most clinical tests.¹⁵ Technological advancement has seen magnetic resonance imaging (MRI) become the gold standard assessment technique¹⁶ and is often used to visualize hamstring tendon involvement and help guide rehabilitation.¹⁷ However due to MRI access and cost,¹⁸ ultrasonography is an alternative for diagnosing HSI and provides information on the length, width, and cross-sectional area of the muscle injury.

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Similar to the assessment of HSI, traditional rehabilitation strategies have evolved over the years and currently try to replicate the eccentric load the hamstring muscles undergo in the swing phase in sprinting.¹⁹ The most popular form of hamstring eccentric strengthening is the nordic hamstring exercise (NHE) which was shown to reduce first-time HSI occurrence by more than 60%²⁰ but was less protective in players with a prior HSI.^{7,19} More functional training methods have included sprint training, which appeared to benefit both eccentric hamstring strength and sprint performance.²¹ However, despite the positive effects of these evidence-based preventative and rehabilitation protocols, the recurrence rate of HSI ranges anywhere from 16 to 60%²² and suggests that these current strategies are still relatively ineffective.

A theoretical explanation for the high rate of recurrence may be the neuromuscular inhibition that occurs after injury in the hamstrings²³ and their synergistic muscles in the lower limb kinetic chain. Neuromuscular inhibition refers to a reduction in muscular activity and/or force output due to a reduction in neural stimulation.²³ It is thought to initially protect the previously injured muscle but if prolonged can impair the efficacy of rehabilitation programs and muscle function on return to sport.²³ Surface EMG has been commonly utilized in research studies to identify neuromuscular inhibition post-HSI²⁴ and detects the myoelectric activity formed by physiological variations in the state of muscle fiber membranes.²⁵ Recently, novel approaches such as transcranial magnetic stimulation (TMS) and twitch interpolation have been used to highlight neuromuscular changes post HSI.^{26,27}

Numerous studies^{24,28,29,30} have demonstrated altered muscle activation patterns in the hamstrings and its synergistic muscles, which control pelvic girdle position, following HSI. Indeed, a recent strain-injured limbs systematic review by Presland et al. that was limited to predominantly hamstring muscle activation concluded that athletes with a history of BF injury had decreased muscular activation when comparing injured and uninjured limbs during eccentric contraction. However, whilst neuromuscular inhibition has been acknowledged in the research literature, primary health care professionals who formulate and execute the rehabilitation programs, may not be familiar with the concept. Therefore, this paper aims to summarize neuromuscular inhibition of the hamstring and lumbopelvic musculature associated with HSI by undertaking a narrative review and determining if there is a need to address this phenomenon in rehabilitation programs to minimize the recurrence of injury.

MATERIALS AND METHODS

Four electronic databases [PubMed, SPORTDiscus, SCOPUS (Elsevier), Google Scholar] were searched using the following constructs: hamstring (strain* OR rupture* OR tear*) AND neuromuscular inhibition (Electromyography OR EMG or assessment*) published in English up to June 2022. Journal articles from peer-reviewed journals were selected by reading abstracts and the full article if required. Further articles were derived from the references of the retrieved articles and systematic reviews. Articles that focused on injury to muscle groups not exclusive to the hamstrings, published in a language other than English, and conference proceedings were excluded from this review.

RESULTS

Eighteen articles were obtained and deemed suitable for analysis in this review. It should be noted that the measures of how neuromuscular inhibition was detected included EMG, isokinetic dynamometry, force plates, functional MRI, twitch interpolation, and TMS. The procurement of surface EMG data was also inconsistent, as was the way it was sampled, filtered, and analyzed. Furthermore, the statistical analysis of this data also differed between studies.

Hamstring Muscle Activity after HSI

Amateur league soccer players who had returned to play following an HSI in the preceding 6–24 months, exhibited significantly lower BF myoelectric activity at varying speeds (30°/s and 120°/s) using isokinetic dynamometry.²⁴ A 5–10% reduction in EMG activity in the BF muscle was noted with a large effect size³¹ during eccentric knee flexion at 30, 50, and 100 ms after the onset of contraction.

Opar et al. observed a similar result with running-sport athletes who had suffered an HSI within the last 18 months but were fully active in their chosen sport. A 20% reduction in surface EMG activity of the previously injured BF muscle was detected during fast (180°/s) and slow (60°/s) eccentric contractions using isokinetic dynamometry.²⁸ Likewise, Sole et al. also demonstrated impaired BF and medial hamstring muscle activity in recreational multisport athletes who had returned to full training. Significantly lower surface EMG myoelectric activity was noted during eccentric isokinetic contractions in the previously injured muscles in mid to outer-range knee flexion (from 65° to 5° of flexion).²⁹ Contrastingly, fully active, elite long jumpers and sprinters showed no significant difference during an isometric knee flexor maximum voluntary contraction (MVC) (164.3 ± 37.8 Nm and 171.3 ± 28.5 Nm for the injured and uninjured limb, respectively, $p = 0.20$).³² It was noted that during a 20% isometric knee MVC, a significantly higher semimembranosus/hamstring ratio for the injured limb compared with the uninjured limb was seen with a small to moderate effect size ($p = 0.018$, $d = 0.37$).³² Furthermore, in the same athletic cohort peak MVC torque did not significantly differ between limbs (164.3 ± 37.8 Nm and 171.3 ± 28.5 Nm for the injured and uninjured limb, respectively, $p = 0.20$). However, the contribution of BF torque over the total hamstring torque (BF/Hams) was lower in the injured than in the uninjured limb at 20% MVC ($-5.6 \pm 10.2\%$, $p = 0.038$, $d = 0.49$) and inversely the contribution of semimembranosus was higher in the injured than in the uninjured limb ($5.6 \pm 7.5\%$, $p = 0.007$, $d = 0.47$).³²

Previously injured hamstring muscles displayed a significant reduction in average torque (-9.9% $p = 0.025$), compared to their uninjured counterparts, during eccentric, outer range knee flexion (from 25° to 5°).²⁹ Lower peak torque values following HSI were also observed in fully active, running-sport athletes.²⁸ A 7–11% reduction in torque during both fast (180°/s) and slow (60°/s) concentric and eccentric isokinetic contractions were noted in previously injured hamstring muscles with a moderate effect size ($d = 0.74$, $d = 0.57$, respectively).²⁸ Additionally, prior HSI also inhibits torque production in “fully recovered” multisport recreational athletes.³³ A near 40% reduction was seen with large to very large effect size, in the rate of torque development at 50 ($p = 0.008$, $d = 1.12$) and 100 ms ($p < 0.001$, $d = 1.20$) after the onset of an anticipated isokinetic eccentric contraction at 60°/s when comparing previously injured and uninjured hamstrings.³³

Furthermore, voluntary hamstring muscle activation in previous HSI measured *via* twitch interpolation during slow (60°/s) eccentric contraction showed significant impairment.²⁶ Fully competing recreational athletes demonstrated a lower percentage of voluntary activation both between injured and uninjured limbs (mean difference = -10.6% , $p = 0.04$, $d = 1.52$) and between injured and control groups (mean difference = -24.1% , $p < 0.001$, $d = 2.70$).²⁶ The same cohort of athletes also displayed lower stretch reflexes (average difference = 3.8% , $p = 0.02$, $d = 0.61$) and tendon tap responses (average difference = -37% , $p = 0.04$, $d = 1.48$).²⁶

Finally, previous HSI is associated with short interval cortical inhibition (SICI) in the BF in recreational multisport athletes.²⁷ The BF of previously injured limbs exhibited greater levels of SICI compared with control limbs (average difference = 19% , $p = 0.007$, $d = 1.33$).²⁷ Furthermore, the BF of contralateral uninjured limbs exhibited greater levels of SICI compared with control limbs (mean difference = 17% , $p = 0.03$, $d = 1.20$).²⁷ It was also noted that the isometric knee flexor strength (in 30° knee flexion) was lower in previously injured limbs compared with control limbs (average

difference = -26% , $p = 0.04$, $d = -1.27$) and contralateral uninjured limbs (average difference = -17% , $p = 0.01$, $d = -0.57$).²⁷

Hamstring Muscle Activity during Functional Movements

Whilst most surface EMG hamstring literature has utilized isokinetic dynamometry^{28,29,33} researchers have also been able to demonstrate the neuromuscular inhibitory effects of prior HSI during functional movements such as high-speed running. Posterior chain musculature surface EMG in elite athletes who had a history of acute sprint-related HSI, showed reductions of between 12.5 and 23% in muscle activity ratios of the BF compared to the ipsilateral gluteus maximus, erector spinae, and external oblique and the contralateral rectus femoris whilst running at 20 km/h in the late swing phase [BF: ipsilateral gluteus maximus (maximum difference -12.5% , $p = 0.03$), BF: ipsilateral erector spinae (maximum difference -12.5% , $p = 0.01$), BF: ipsilateral external oblique (maximum difference -23% , $p = 0.01$), and BF: contralateral rectus femoris (maximum difference -22% , $p = 0.02$)].³⁰ Furthermore, decreased long head of BF muscle activity was also noted in male college sprinters during a maximal sprint effort.³⁴ In the late-swing phase, 80% of the athletes demonstrated reduced myoelectric activity in the BF long head in the previously injured limb compared to the uninjured limb ($p < 0.05$, $d = 0.95$).³⁴ Previously, Silder et al. had investigated a group of 18 multisport athletes with a history of HSI and showed no significant limb difference in the magnitude of BF or medial hamstring activity during treadmill running at 60%, 80%, 90%, and 100% of maximum sprinting speed.³⁵

The conflicting results of hamstring myoelectric activity in the hamstring musculature during functional tasks are also observed in the temporal characteristics of muscle activation. There were no significant differences in hamstring muscle onset, offset or duration of muscle activation during overground running at 60%, 70%, or 80% of maximum speed amongst 15 elite AFL footballers, 7 of whom had suffered an HSI within the preceding six months.³⁶ However, altered contraction patterns in previously injured hamstring muscles were observed in marching movements.³⁷ In athletes who had suffered an HSI within the preceding 12 months but had returned to training, the onset of BF and medial hamstring myoelectric activity was significantly earlier ($p < 0.05$) than their injury-free counterparts.³⁷ This resulted in a significantly faster (54 ms, $p = 0.049$) initiation of movement, which the authors attribute to changed proprioception.³⁷ These findings are corroborated in amateur league soccer players with previous HSI who displayed worse knee joint position sense.²⁴ The previously injured hamstring muscles displayed a significantly higher absolute angle error with a very large effect size (Injured group = 4.6° vs Control group = 1.8° , $p = 0.014$).

Altered Hamstring Synergist Muscle Activation and HSI Risk

Researchers have shown that reducing hamstring myoelectric activity following HSI creates asymmetrical pelvic and lower limb movement patterns.^{30,34} Accordingly, differences in the neuromuscular activation of the hamstring synergistic muscles during high-speed running appear to have an association with HSI.^{38,39} Increased average ($p = 0.001$) and peak surface EMG activity ($p = 0.06$) of the gluteus medius in elite footballers running between 12 and 15 km/h was associated with an increased in-season hamstring injury risk.³⁸

Conversely, surface EMG recordings in elite soccer players during airborne phases of sprinting suggest that the risk of HSI decreased by 20% and 6% with increased gluteus maximus and trunk muscle activity (external and internal obliques, thoracic and lumbar erector trunci) in initial front swing and back swing, respectively.³⁹ Furthermore, surface EMG has shown that recruitment sequences of the hamstring synergists can influence HSI risk in amateur soccer players.⁴⁰ In this group, activation of lumbar musculature, prior to the hamstring muscle group, could significantly estimate injury occurrence with a sensitivity of 0.80 and a specificity of 0.23. The risk of sustaining a hamstring injury significantly increased when there was a delay in hamstring activity onset ($p = 0.018$) in an activation sequence in which the lumbar erector muscles were recruited prior to the hamstrings ($p = 0.009$) during the prone hip extension exercise.⁴⁰ Similarly, professional soccer players who have previously suffered an HSI exhibited increased myoelectric activity noted in the medial hamstring ($p = 0.01$) and the gluteus maximus muscles ($p = 0.04$) during the prone hip extension exercise.⁴¹

Neuromuscular Inhibition of the Hamstring Musculature and its Impact on Rehabilitation

Hamstring strength appears to have a direct link to HSI risk with many studies investigating the NHE due to its ability to improve eccentric hamstring strength.⁷ Functional MRI has demonstrated that previously injured hamstring muscles were on average approximately 7% less active whilst performing the NHE.⁴² This neuromuscular inhibition due to prior HSI appears to adversely impact the effectiveness of the NHE in increasing eccentric hamstring strength.⁴³ Comparisons made at the start and end of pre-season training in elite footballers revealed on average a 40N difference in strength gain between previously injured athletes and those that had never sustained an HSI ($p = 0.012$).⁴³

DISCUSSION

Hamstring strain injury (HSI) is a frequent and expensive injury in field sports that have a high-speed running component.⁴⁴ Current evidence-based HSI rehabilitation interventions focus on a progressive return to high-speed running and sprinting.⁴⁵ This is facilitated by a progression from concentric to eccentric hamstring strengthening as well as trunk stability exercises.¹⁶ However, despite this evidence-based approach, HSI still remains the most common injury in professional soccer⁴⁶ and ARF and has the highest recurrence rate.⁴⁷ It is common knowledge amongst sports medicine practitioners that apart from age, the greatest risk factor for a new HSI, is a previous HSI.⁴⁸ Whilst clinicians may use the NHE as a preventative measure they may be unfamiliar with the concept of persisting neuromuscular inhibition of the hamstrings and the lumbopelvic musculature after athletes return to sport following HSI.

Muscle strains occur when there is excess tension on the contracted muscle.⁴⁹ Furthermore, activated muscles have more elasticity and can store more elastic energy when stretched, whereas relaxed muscles cannot.⁴⁹ It has been widely accepted that during a 100 ms window in the late swing phase of high-speed running, the long head of BF contracts eccentrically,^{9,10,33,50} puts it at risk for HSI. This has been challenged by Van Hooren and Bosch, who suggest that there is no eccentric contraction, but rather an isometric contraction of the hamstrings during the swing phase of high-speed running.⁵¹ They go on to suggest that current

rehabilitation strategies, which focus on eccentric strengthening, are therefore incorrectly training the muscles and hence may be a reason for the high rate of prevalence in HIS.⁵¹

It has been demonstrated in this narrative review that previous HSI causes decreased myoelectric activity during eccentric hamstring contraction. This is in accordance with a recent systematic review⁵² which essentially focused only on hamstring muscle activation post HSI and did not assess the effects on the lumbopelvic musculature. There is conflicting evidence regarding muscle activation during isometric contraction in previously injured hamstring muscles. However, in the study by Avrillon et al. in which no difference was demonstrated during an isometric knee flexor MVC, there was increased semimembranosus myoelectric activity relative to BF.³² This increased contribution of the semimembranosus was also seen during torque generation and may reflect the semimembranosus working harder to compensate for the previously injured BF and its suboptimal activation levels. The more novel approaches to identify neuromuscular inhibition *via* twitch interpolation²⁶ and TMS²⁷ have demonstrated that neural inhibition after HSI could have its origins from cortical and spinal cord inhibition.

Furthermore, the harmful effects of neuromuscular inhibition post HSI are not exclusive to the hamstring musculature and cause asymmetries in pelvic and lower limb movement patterns during high-speed running.^{30,34} There are conflicting results regarding BF activity during high-speed running with both the temporal and magnitude of muscle activation. This may have been due to different population characteristics. For example, Crow et al. utilized a group of elite AFL footballers who would have likely undergone the most up-to-date evidence-based rehabilitation protocols.³⁶ However, what has been established is that lumbopelvic musculature exerts influence on the BF strain and that well-timed neuromuscular control of the gluteal and trunk muscles provides stability during running and can mitigate HSI risk.^{39,53} The reduced activation of these proximal muscles creates an unhelpful anterior pelvic tilt, increased hip flexion, and increased medial knee rotation³⁰ and compounds the strain in the BF muscle, likely increasing the risk of injury.

When considering the aforementioned factors, it becomes apparent that a previously injured BF which has lost its proprioceptive ability, has reduced myoelectric activation and consequently cannot generate torque rapidly, will certainly be at a high risk of strain injury. These findings were observed in athletes that had returned to training or sport and had recovered. However, traditional guidelines do not incorporate EMG analysis as part of mainstream rehabilitation for HSI.⁴⁶ Given the findings of this review, this may need to be considered as there are clearly aberrant muscle activity patterns that could be detected both in the hamstrings and the lumbopelvic musculature during functional activities. Returning such athletes, without this valuable information may be the reason for such a high recurrence of hamstring re-injury. Conversely, the NHE, which is commonly used for the prevention of hamstring strains⁵⁴ has been shown here to not be fully effective in the rehabilitation of an injured BF. The neuromuscular inhibition seen in the hamstring and pelvic musculature post HSI appears to dampen the effectiveness of NHE by reducing activation of the muscles, which results in smaller strength gains.^{42,43} Furthermore, if the hamstrings are not contracting eccentrically, the NHE may not be replicating the forces that cause HSI. Instead, Van Hooren and Bosch suggest that isometric exercises should be prescribed

in a way that the hamstrings contract isometrically to resist/reduce knee extension and to assist hip extension.⁵⁵

Electromyography (EMG) analysis may appear to have a role in HSI screening programs. Increased gluteus medius activity during running is a risk factor for HSI and is likely reflective of the weakness of the hamstrings and other pelvic stabilizing muscles. Additionally, increased gluteus maximus and medial hamstrings activity during a prone hip extension, is likely a compensatory strategy for a weak BF. Furthermore, the recruitment sequence of the lumbopelvic muscles in a prone hip extension exercise was also able to significantly predict injury occurrence and may be useful as a pre-season screening tool.⁴⁰ The incorporation of EMG and the NHE after HSI may have the utility to also screen for gluteus maximus weakness during rehabilitation.¹⁹ Finally, the advent of novel techniques such as TMS has provided further avenues to screen for intracortical inhibition following HSI and could also be an area for future research into rehabilitation strategies that could potentially reverse inhibition at a supraspinal level.²⁷

This narrative review has demonstrated that neuromuscular inhibition exists in the hamstring and lumbopelvic musculature after HSI, however, it was not without limitations. Firstly, this was a narrative rather than a systematic review because there was a lack of homogenous data for comparative analysis. Furthermore, there was a lack of consistency in EMG data acquisition and statistical analysis used by various authors. Indeed, even a recently published systematic review⁵² could not perform a meta-analysis and instead performed a best evidence synthesis due to the lack of consistent methodology across the studies. The majority of the studies were retrospective in design and therefore it could not be determined whether activation deficits were the cause or result of previous HSI. Finally, due to the intervention and population groups being investigated, the studies did have small sample sizes, which was a common theme acknowledged by most authors.

In conclusion, neuromuscular inhibition in the hamstring and lumbopelvic musculature following HSI is a phenomenon that should be addressed when rehabilitating athletes. For this to occur, EMG equipment and analysis needs to become more accessible and easier to use by clinicians in primary healthcare, in order to detect this maladaptive response to injury. Further research needs to utilize consistent EMG analysis with larger sample sizes to conduct longitudinal research that investigates interventions that are successful in restoring normal myoelectric activity following HSI.

Key Points

- Despite evidence-based rehabilitation protocols following HSI, neuromuscular inhibition is present in the hamstring and lumbopelvic musculature.
- Electromyography (EMG) may be a useful tool to detect neuromuscular changes following HSI and may be used to determine new exercises that could reverse neuromuscular inhibition.
- Electromyography (EMG) may be a useful final objective assessment tool prior to return to play following a HSI.

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