Published in final edited form as:

Med Sci Sports Exerc. 2013 March ; 45(3): 448–454. doi:10.1249/MSS.0b013e3182776eff.

Hamstring Strength and Morphology Progression after Return to Sport from Injury

Jennifer Sanfilippo¹, Amy Silder², Marc A Sherry³, Michael J Tuite⁴, and Bryan C Heiderscheit^{1,3,5}

¹Departments of Biomedical Engineering and Athletics, University of Wisconsin-Madison, Madison, WI

²Departments of Bioengineering and Orthopaedic Surgery, Stanford University, Stanford, CA

³Sports Rehabilitation, University of Wisconsin Health Sports Medicine, Madison, WI

⁴Department of Radiology, University of Wisconsin-Madison, Madison, WI

⁵Departments of Orthopedics and Rehabilitation, University of Wisconsin-Madison, Madison, WI

Abstract

Hamstring strain re-injury rates can reach 30% within the initial two weeks following return to sport (RTS). Incomplete recovery of strength may be a contributing factor. However, relative strength of the injured and unaffected limbs at RTS is currently unknown. **PURPOSE:** Characterize hamstring strength and morphology at the time of RTS and six months later. **METHODS:** Twenty-five athletes that experienced an acute hamstring strain injury participated, following completion of a controlled rehabilitation program. Bilateral isokinetic strength testing and magnetic resonance imaging (MRI) were performed at RTS and 6-months later. Strength (knee flexion peak torque, work, angle of peak torque) and MRI (muscle and tendon volumes) measures were compared between limbs and over time using repeated measures ANOVA. **RESULTS:** The injured limb showed a peak torque deficit of 9.6% compared to the uninjured limb at RTS (60°/s, $p < 0.001$), but not 6-months following. The knee flexion angle of peak torque decreased over time for both limbs (60°/s, $p < 0.001$). MRI revealed that 20.4% of the muscle cross-sectional area showed signs of edema at RTS with full resolution by the 6-month follow-up. Tendon volume of the injured limb tended to increase over time ($p = 0.108$), while muscle volume decreased 4–5% in both limbs ($p < 0.001$). **CONCLUSION:** Residual edema and deficits in isokinetic knee flexion strength were present at RTS, but resolved during the subsequent six months. This occurred despite MRI evidence of scar tissue formation (increased tendon volume) and muscle atrophy, suggesting that neuromuscular factors may contribute to the return of strength.

Keywords

magnetic resonance imaging; knee flexion torque; rehabilitation; muscle volume

Introduction

Hamstring re-injury rates have been reported as high as 30% within the same season for Australian Footballers (19). Incomplete recovery and inadequate rehabilitation have been suggested as explanations for this high re-injury rate (7). In particular, a residual strength deficit in the injured limb is considered one of the primary causes (8, 9, 23, 24, 30); however, the extent of strength loss at the time of return to sport (RTS) has not yet been investigated.

Persistent strength deficits have been observed in individuals with recurrent hamstring injuries, despite returning to athletic competition (9). For example, in comparison to the unaffected limb, a 10% reduction in concentric peak torque was noted in subjects having experienced injury two to 12 months prior, with a 22% strength deficit during eccentric testing (9). In addition to reduced peak torque production, the knee flexion angle at which peak concentric torque occurs has been found to increase in previously injured limbs (4). This finding suggests that torque production at longer muscle lengths may be compromised. Considering the susceptibility for sustaining a muscle strain injury is greatest during eccentric loading in a lengthened position (10, 11, 17, 18, 22), these observed strength deficits, particularly at longer muscle lengths likely increase re-injury risk.

Atrophy of the previously injured muscle may also contribute to the persistent strength loss (9). A substantial reduction in biceps femoris long head volume has been found in over 50% of individuals with a prior injury despite having returned to athletic competition (25). While the rehabilitative process plays an important role in the return of muscle size following the acute injury (25), changes in the relative amount of connective tissue may also impede recovery (15). Scar tissue adjacent to the site of original injury has been observed as early as 6 weeks (7) and as late as 23 months post-injury (25). The presence of scarring has been shown to alter the in-vivo muscle contraction mechanics, generating localized regions of high tissue strains near the site of prior injury of the biceps femoris (26). The presence of scar tissue may increase the overall stiffness of the musculotendon unit (5, 14), though this idea has not been scientifically tested. It is likely that, together, these changes in muscle morphology following a hamstring strain injury may compromise the return of normal muscle function.

The purpose of this study was to characterize isokinetic hamstring strength and morphology at the time of RTS following a controlled rehabilitation program for an acute strain injury. Tests were then repeated 6 months following RTS to provide additional insights into the recovery process over time. This study provides a unique understanding of the extent of healing at the time of RTS and how this healing progresses over the following 6 months. We hypothesized that, at RTS, the injured muscle would display weakness and associated changes in hamstring muscle morphology based on magnetic resonance imaging (MRI).

Methods

Participants

Twenty five recreational athletes (20 male, 5 female, 24 ± 9 y, 1.7 ± 0.5 m, 73.8 ± 25.8 kg) participated in the study. All subjects sustained an acute hamstring strain injury and completed a controlled rehabilitation program. Subjects were recruited from the University of Wisconsin Health clinics and University of Wisconsin recreation facilities. To qualify, subjects needed to be 16–50 years of age and involved in athletics a minimum of two days per week. The subjects must have sustained an acute, sudden onset, hamstring injury within the prior 10 days and display two or more of the following symptoms: palpable pain along any of the hamstring muscles, posterior thigh pain without radicular symptoms during a

straight leg raise, weakness with resisted knee flexion, or pain with resisted knee flexion. Exclusion criteria included: complete hamstring muscle disruption (grade III) or avulsion, posterior thigh pain originating from another source (e.g., inguinal or femoral hernia, nerve entrapment, lumbosacral pathology), or any comorbidity that might prevent participation in a rehabilitation program. Each subject or parent/guardian provided written informed consent prior to testing, in accordance with the University of Wisconsin's Health Sciences Institutional Review Board.

Protocol

Following enrollment, subjects immediately began a controlled rehabilitation program under the supervision of the same physical therapist (MAS). The exercises included in the rehabilitation program were the same as those previously published (3, 12). Each subject continued the rehabilitation program until established RTS criteria were met, including no significant pain with straight leg raise, full isometric hamstring strength against manual resistance in prone at 90° and 15° of knee flexion, no tenderness to palpation, and no apprehension during full effort, sport-specific movements. Once cleared to RTS by the treating physical therapist, each subject underwent an MRI exam and isokinetic strength assessment; these measures were repeated 6 months later. Subjects were encouraged to continue the rehabilitation exercises (3 times/week) on an independent basis; rehabilitation compliance after RTS was not monitored.

Strength Testing

Subjects were positioned on an isokinetic dynamometer (Biodex Multi-Joint System 2, Biodex Medical Systems, Inc., Shirley, NY, USA) such that the hip was flexed to 90° and the dynamometer and knee joint axes were aligned. Strapping was used over the shank, thigh, and waist to minimize secondary joint movement. Each subject performed maximum effort knee flexion/extension testing through full range of motion during two conditions: concentric at 60°/s (5 repetitions) and concentric at 240°/s (15 repetitions). Eccentric knee flexion testing at 30°/s (3 repetitions) was also performed with the final 13 subjects, as this test was added after the study had begun. Prior to each test, subjects received four sub-maximal practice trials. Joint angle and torque were recorded after being corrected for gravity. Full knee extension was defined as 0°.

Peak torque and angle to peak torque for each testing condition was calculated consistent with Brockett et al (4). All analyses were limited to the repetitions at each speed containing the highest peak torque values (3 repetitions at 60°/s; 12 repetitions at 240°/s; 2 repetitions at 30°/s). Torque-angle curves from the select repetitions at each speed were compiled and sorted in relation to movement direction (i.e., flexion/extension) and knee flexion angle. This resulted in one torque-angle curve for each subject, speed, and direction. Next, every successive block of nine data points from the compiled torque angle curve, was replaced with an average value. Finally, for each subject, speed, and direction, a 2nd order polynomial curve was fit to the torque data that were within 10% of the peak torque measurement for that particular condition. Peak torque and angle to peak torque were determined from the resulting polynomials. Work was calculated from 0° to 90° of knee flexion by integrating knee flexion torque with respect to time. In addition, the mixed hamstring to quadriceps ratio (H:Q) was calculated as hamstring eccentric peak torque at 30°/s relative to quadriceps concentric peak torque at 240°/s (19).

Magnetic Resonance Imaging

Images were obtained for each subject on a 1.5 Tesla Twin Speed magnetic resonance scanner (General Electric Healthcare, Milwaukee, WI, USA) using a phased array torso coil. Each MRI exam included three scans: Iterative Decomposition of water and fat with Echo

Asymmetry and Least-Squares estimation (IDEAL) combined with three-dimensional (3D) spoiled gradient echo (SPGR) imaging (20), T2-weighted fat-suppressed fast spin-echo coronal scan, and T2-weighted fat-suppressed fast spin-echo axial scan. T2 Imaging details were: IDEAL coronal 3D slab, TR=12.5ms, 3 echoes (1 echo/TR) with TE=4.4, 5.0, 6.6ms, 15° flip angle; matrix, ± 41.7 kHz bandwidth, 384×256 matrix with 46×46cm field-of-view with 84 slices, and 1.4mm slice thickness for a true spatial resolution of $1.2 \times 1.8 \times 1.4 \text{ mm}^3$ (interpolated to $0.9 \times 0.9 \times 7 \text{ mm}^3$). Water and fat images were created using homodyne reconstruction performed on-line (21, 31). Coronal T2-weighted scan - 4mm slice thickness, 4.4mm slice interval, 512×512matrix, 90° flip angle, and 2200/9.7 TR/TE; Axial T2-weighted scan - 5mm slice thickness, 5mm slice interval, 256×256matrix, 90° flip angle, and 3200/89.0 TR/TE.

The two MRI exams were analyzed by the same investigator (MJT) at different time points to avoid biased measurements. The total injured area over all muscles was determined at the level where the injury had the largest absolute axial cross sectional area. Specifically, the cross sectional area of the injury was calculated from the medio-lateral width (ML) and antero-posterior depth (AP) using the formula, $0.25 \times \pi \times \text{ML} \times \text{AP}$ (2, 7, 24, 27, 30). In addition, muscle and tendon-scar volumes of the biceps femoris long head (BFLH), biceps femoris short head (BFSH), semitendinosus (ST), and proximal conjoint biceps femoris and semitendinosus tendon (BFT) were determined at both time points and for both limbs by the same investigator (JS) using manual segmentation (Mimics Software; Materialize Corp.; Ann Arbor, MI, USA) (Figure 1). The structure boundaries were manually outlined on each coronal slice for muscles and axial slice for tendons in which the structure of interest was present. Intra-observer variability for this technique has been reported at < 5% for both muscle (13, 29) and tendon (25). Volume was then calculated by summing the cross sectional area of each slice and multiplying by the interslice distance. Absolute muscle volumes were analyzed for the BFLH, BFSH and ST, while the percent difference between limbs at each time point was assessed for the BFT due to its smaller comparative size and potential for error.

Statistical analysis

Three subjects did not undergo testing at the 6-month follow-up as they experienced an injury after RTS during sports participation (two hamstring strains, one anterior cruciate ligament tear). Only the complete data sets from the 22 subjects were included in the analysis, with the exception of the H:Q peak torque ratio which was based on only 13 subjects. The outcome measures analyzed included: peak torque, angle to peak torque, work (60 and 240°/s); H:Q peak torque ratio; muscle volumes (BFLH, BFSH, ST); and tendon volumes (BFT). Two-factor repeated measures ANOVAs (limb-by-time) were performed to compare all outcome measures ($p < 0.05$), except BFT volumes were compared over time using a dependent t-test. Post hoc testing was performed as needed using Tukey's HSD (Statistica 6.0, StatSoft Inc., Tulsa, OK, USA).

Results

MRI examination at the time of injury revealed 16 subjects sustained an injury primarily to the biceps femoris, four to the semimembranosus, and two to the ST. The average time away from sport was 26 days (range, 17–49 days).

Strength

Isokinetic strength testing at 60°/s revealed significant limb-by-time interactions for peak torque ($p < 0.001$) and work ($p = 0.002$) (Table 1). On average, the injured limb had a 9.6% deficit in peak torque ($p < 0.001$) and a 6.4% deficit in work ($p = 0.003$) at RTS compared to

the uninjured limb. These differences resolved by the 6-month follow-up (Figure 2). The knee flexion angle of peak torque decreased ($p<0.001$) for both limbs from RTS to the 6-month follow-up, reflecting a shift in peak torque development to a longer hamstring length (Table 1, Figure 2).

Testing at $240^{\circ}/s$ showed a main effect for limb, with less peak torque ($p=0.021$) and less work ($p=0.006$) produced by the injured limb compared to the uninjured limb. No significant difference in angle of peak torque was present between limbs or over time.

The H:Q ratio ($30^{\circ}/s$ eccentric: $240^{\circ}/s$ concentric) revealed a main effect for limb ($p=0.023$), with the injured limb having a smaller ratio compared to the uninjured limb (RTS: injured 1.30 ± 0.26 , uninjured 1.62 ± 0.31 ; 6-months: injured 1.39 ± 0.26 , uninjured 1.46 ± 0.15).

Morphology

At the time of RTS, the percent of muscle area showing signs of injury (i.e., T2 hyperintensity) when considering all involved muscles was $20.4 \pm 19.4\%$. By the 6-month follow-up, no evidence of injury was visible on MRI (Figure 3). The muscle volumes of the ST (4.1% , $p=0.024$) and BFSH (6.1% , $p=0.010$) for both limbs decreased from RTS to 6-month follow-up, while the BFLH volume (3.1% , $p=0.078$) showed a similar trend. To obtain a representation of the overall biceps femoris muscle volume, we summed the BFLH and BFSH volumes and found a significant decrease (5.2% , $p=0.010$) for both limbs from RTS to 6-months following RTS (Table 2). Only BFSH showed a significant difference between limbs ($p=0.036$), with the muscle volume of the injured limb being larger than the uninjured limb over both time points.

At RTS, the BFT volume of the injured limb was $4.4 \pm 0.3\%$ smaller than the uninjured limb. At the 6-month follow-up, the injured limb's BFT volume was $29.9 \pm 82.9\%$ larger; however, this change over time was not significant ($p=0.108$).

Discussion

The purpose of this study was to examine the effects of an acute hamstring strain injury on strength and morphology at RTS and after the subsequent 6 months. The injured limb showed a strength deficit at RTS ($60^{\circ}/s$: peak torque, 9.6% ; work, 6.4%) compared to the uninjured limb, with 20% of the cross-sectional area still showing signs of injury on MRI. Six months following RTS, this strength deficit resolved, despite the ST and biceps femoris muscles of both limbs experiencing 4–5% atrophy.

This study is the first to assess hamstring strength at the time of RTS following a strain injury. The observed strength deficit of the injured limb at RTS is consistent with the 10% deficit reported in individuals having already returned to sport for periods ranging from two to 12 months (9). Unlike Croisier et al (9), our results showed full strength recovery by 6 months after RTS. This difference may be reflective of the greater number of subjects with prior hamstring strain injuries: 46% of the subjects in Croisier et al (9) compared to only 13% (3 subjects) of the subjects in the current study.

The strength deficit present at the time of RTS is likely related to the remaining muscle injury observed on MRI (T2-hyperintensity). On average, 20% of the muscles' cross-sectional area showed signs of injury at RTS with the MRI performed an average of 26 days post-injury (range, 13–49 days). These findings are consistent with Askling et al (2); at 21 days following injury, 26% of the muscle area showed signs of injury, with a reduction to 17% by 42 days post-injury. In the current study, there was no evidence of remaining injury

on MRI performed at the 6-month follow-up. Thus, it appears that healing progressively continues following RTS and is completed within the subsequent 6-month period.

Given the relationship between muscle size and strength, we anticipated muscle hypertrophy would accompany the strength gains. However, our results showed an average atrophy of 4–5% in the hamstring muscles of the injured limb from RTS to the 6-month follow-up. This atrophy was not limited to the most involved muscle but was observed in the BFLH, BFSH, and ST; despite the ST being the primary muscle injured in only two subjects and no subjects having primary involvement of the BFSH. Prior work has observed an apparent compensation among agonist muscles following a strain injury. Specifically, a 10% hypertrophy in the BFSH appeared to offset the corresponding 13% atrophy in the BFLH in individuals with a history of strain injury to the BFLH (25). This potential compensation was not observed over the 6-month period following RTS in the current study, as both the BFLH and BFSH showed a similar degree of atrophy. Consequently, another explanation for the observed strength gains must exist, such as neuromuscular influences. Considering the degree of injury still evident on MRI at RTS, a protective neuromuscular inhibition may exist at that time to limit peak torque and minimize re-injury risk. At the six month follow-up, all indication of injury observed on MRI had resolved, and we re-emphasize that muscle volume had decreased. Therefore, we propose that part of the strength gains over time can be explained by removal of the neuromuscular inhibition during the course of complete muscle healing, regardless of muscle volume changes. Indeed, reduced activation of the hamstring muscles with a corresponding reduction in peak torque has been observed in individuals following a hamstring strain injury (28). However, as we did not collect electromyography data in the current study, we are unable to confirm this relationship.

It has been suggested that the time of RTS following injury is, in part, influenced by psychosocial factors such as fear and apprehension (1, 6). For example, increased fear of movement and re-injury has been associated with decreased perceived function in individuals nearing completion of rehabilitation following anterior cruciate ligament reconstruction (6). Specific to athletes with a recent hamstring strain injury, insecurity when performing a ballistic hip motion has been observed at the time of RTS testing, despite having passed common clinical strength and flexibility tests (1). As such, fear or apprehension within our subjects at the time of RTS may partially explain the corresponding reduction in strength. However, we do not believe these psychosocial factors played a primary role, as all subjects in our study were required to complete a variety of sport-specific movements at full effort without apprehension prior to being cleared for RTS and the isokinetic strength testing.

One likely explanation for the observed hamstring atrophy may be that the subjects reported a decrease in athletic participation from RTS to the 6 months follow-up. On average, our subjects reported participating in sports 5.6 ± 1.2 days/week prior to injury and only 3.7 ± 2.1 days/week at the 6-month follow-up. A reduction in activity could also explain the decreased muscle volumes observed in the uninjured limb. Why there was a reduction in activity level is not fully understood, as it could suggest residual symptoms or be related to a change in athletic season. Many of the subjects in this study were injured during their in-season athletic play. When the 6-month follow-up testing was performed, the subjects may have been out-of-season and therefore less active.

Earlier research has shown that individuals with a history of multiple hamstring strain injuries display a greater knee flexion angle of peak torque (41°) compared to their uninjured limb (30°) (9). Surprisingly, we did not observe a side-to-side difference in the angle of peak torque at either RTS or the 6-month follow-up. However, two interesting observations were present. First, both limbs displayed an average knee flexion angle of peak

torque of approximately 40° at RTS, 10° more flexed than at the 6-month follow-up. Second, the angle of peak torque for both limbs at the 6-month follow-up (~30°), was consistent with values previously reported among individuals that never incurred a hamstring strain injury (4).

Hamstring-to-quadriceps (H:Q) ratio (30°/s eccentric: 240°/s concentric) values less than 1.05 have been proposed as a predictor of re-injury (8). We observed H:Q values above this criterion in both limbs at RTS and the 6 month follow-up. While we did not observe a significant limb-by-time interaction ($p=0.120$), we did find the H:Q of the injured limb to be consistently smaller than the uninjured limb. It is important to note that eccentric isokinetic testing at 30°/s was added at the mid-point of the study, and only the final 13 subjects completed this testing; thus, further investigation is warranted.

It is widely recognized that scarring accompanies healing following a strain injury. Scar tissue has been observed as early as six weeks after an initial injury (9) and found to persist on a long-term basis (25). Considering the biceps femoris or ST were the primary muscles injured in 83% of our subjects (19/23), we anticipated the BFT volume would be larger in the injured limb and increase across time between measurement sessions. However, the 35% increase in BFT volume between RTS and the 6 month follow-up was not significant ($p=0.108$), primarily due to the large variability in volumes changes observed between subjects.

It has been proposed that the increased knee flexion angle of peak torque in injured muscles is a result of scar tissue formation and the effective shortening of the adjacent muscle fibers (4). Interestingly, we only observed changes in BFT volume in the injured limb, while the shift in angle of peak torque was bilateral. Thus, injury-induced scar tissue formation is likely not the sole cause of changes in angle of peak torque. Again, we suggest that neuromuscular influences may be present. Reduced or delayed muscle activation could produce a similar effect, causing peak torque to occur at a greater knee flexion angle as we observed here.

Despite being cleared to RTS, the presence of continued edema and strength deficits suggest that an extended convalescent period may be necessary to achieve full recovery. The required amount of time needed for this to occur is currently unknown. Our findings indicate that recovery occurs by 6 months; however, it is unlikely that this full period of time is necessary. Future work is needed to more closely identify the time point of recovery. Further, it appears that the battery of clinical tests used in the current study for determining clearance of RTS was inadequate, as all subjects in this study were cleared for RTS despite the presence of these residual deficits. Inclusion of objective measures such as isokinetic strength seems warranted in determining readiness of RTS. Although a limited number of re-injuries were observed during the 6 month period of this study, we performed a preliminary comparison of the measures at RTS between those that did and did not sustain re-injury. No significant differences between these subject groups were observed for any of our outcome measures. Thus, the influence that the strength deficits and edema present at RTS have on re-injury risk remains an area of future study.

Certain limitations within the study should be considered when interpreting its findings. We opted to report absolute muscle volumes rather than normalizing the volume of the injured limb to the uninjured limb. While normalizing would likely reduce the potential error associated with the segmentation process, muscle volume can quickly change with associated changes in physical activity; thus, the uninjured limb would not serve as a proper reference over time. However, because tendon properties are more difficult to change as a result of physical activity (16), we did report normalized tendon volumes.

Our findings indicate that, at the time of RTS following a hamstring strain injury, the injured limb displays a strength deficit compared to the uninjured limb and shows MRI evidence of muscle injury. By six months after RTS, the strength deficit and signs of injury are fully resolved. Bilateral changes in angle of peak torque appear to occur independent of scar tissue formation, suggesting a neuromuscular influence. The influence of these combined factors on re-injury risk is uncertain at this time.

Acknowledgments

Funding: This study was funded by the National Football League Medical Charities, the National Institutes of Health (RR 025011) and the University of Wisconsin Sports Medicine Classic Fund

The authors would like to acknowledge Scott Hetzel for his assistance with the statistical analyses. This work was funded by the National Football League Medical Charities, the National Institutes of Health (1UL2RR025012) and the University of Wisconsin Sports Medicine Classic Fund. The results of the present study do not constitute endorsement by ACSM.

References

1. Askling CM, Nilsson J, Thorstensson A. A new hamstring test to complement the common clinical examination before return to sport after injury. *Knee Surg Sports Traumatol Arthrosc.* 2010; 18(12): 1798–803. [PubMed: 20852842]
2. Askling CM, Tengvar M, Saartok T, Thorstensson A. Acute first-time hamstring strains during high-speed running: a longitudinal study including clinical and magnetic resonance imaging findings. *Am J Sports Med.* 2007; 35(2):197–206. [PubMed: 17170160]
3. Baquie P, Reid G. Management of hamstring pain. *Aust Fam Physician.* 1999; 28(12):1269–70. [PubMed: 10650604]
4. Brockett CL, Morgan DL, Porske U. Predicting hamstring strain injury in elite athletes. *Med Sci Sports Exerc.* 2004; 36(3):379–87. [PubMed: 15076778]
5. Butler DL, Juncosa N. Functional efficacy of tendon repair processes. *Ann Rev Biomed Eng.* 2004; 6:303–29. [PubMed: 15255772]
6. Chmielewski TL, Jones D, Day T, Tillman SM, Lentz TA, George SZ. The association of pain and fear of movement/reinjury with function during anterior cruciate ligament reconstruction rehabilitation. *J Orthop Sports Phys Ther.* 2008; 38(12):746–53. [PubMed: 19047767]
7. Connell DA, Schneider-Kolsky ME, Hoving JL, Malara F, Buchbinder R, Koulouris G, Burke F, Bass C. Longitudinal study comparing sonographic and MRI assessments of acute and healing hamstring injuries. *AJR Am J Roentgenol.* 2004; 183(4):975–84. [PubMed: 15385289]
8. Croisier JL, Ganteaume S, Binet J, Genty M, Ferret JM. Strength imbalances and prevention of hamstring injury in professional soccer players: a prospective study. *Am J Sports Med.* 2008; 36(8): 1469–75. [PubMed: 18448578]
9. Croisier J, Forthomme B, Namurois M, Vanderthommen M, Crielaard J. Hamstring muscle strain recurrence and strength performance disorders. *Am J Sports Med.* 2002; 30(2):199–203. [PubMed: 11912088]
10. Garrett WE, Safran MR, Seaber AV, Glisson RR, Ribbeck BM. Biomechanical comparison of simulated and nonstimulated skeletal muscle pulled to failure. *Am J Sports Med.* 1987; 15(6):448–54. [PubMed: 3674268]
11. Heiderscheit BC, Hoerth DM, Chumanov ES, Swanson SC, Thelen BJ, Thelen DG. Identifying the time of occurrence of a hamstring strain injury during treadmill running: a case study. *Clin Biomech (Bristol, Avon).* 2005; 20(10):1072–8.
12. Heiderscheit BC, Sherry MA, Silder A, Chumanov ES, Thelen DG. Hamstring strain injuries: recommendations for diagnosis, rehabilitation, and injury prevention. *J Orthop Sports Phys Ther.* 2010; 40(2):67–81. [PubMed: 20118524]
13. Holzbaur KR, Murray WM, Gold GE, Delp SL. Upper limb muscle volumes in adult subjects. *J Biomech.* 2007; 40(4):742–9. [PubMed: 17241636]

14. Huijing P, Baan G. Myofascial force transmission: muscle relative position and length determine agonist and synergist muscle force. *J Appl Physiol.* 2003; 94(3):1092–107. [PubMed: 12571138]
15. Jarvinen T, Jarvinen T, Kaariainen M, Kalimo H, Jarvinen M. Muscle injuries: biology and treatment. *Am J Sports Med.* 2005; 33:745–64. [PubMed: 15851777]
16. Kubo K, Kanehisa H, Ito M, Fukunaga T. Effects of isometric training on the elasticity of human tendon structures in vivo. *J Appl Physiol.* 2001; 91(1):26–32. [PubMed: 11408409]
17. Lieber RL, Friden J. Mechanisms of muscle injury gleaned from animal models. *Am J Phys Med Rehabil.* 2002; 81(11):S70–9. [PubMed: 12409812]
18. Lieber R, Friden J. Muscle damage is not a function of muscle force but active muscle strain. *J Appl Physiol.* 1993; 74(2):520–6. [PubMed: 8458765]
19. Orchard J, Best T. The management of muscle strain injuries: an early return versus the risk of recurrence. *Clin J Sports Med.* 2002; 21(1):3–5.
20. Reeder SB, McKenzie CA, Pineda AR, Yu H, Schimakawa A, Brau AC, Hargreaves BA, Gold GE, Brittain JH. Water-fat separation with IDEAL gradient-echo imaging. *J Magn Reson Imaging.* 2007; 25:644–52. [PubMed: 17326087]
21. Reeder SB, Pineda AR, Wen Z, Shimakawa A, Yu H, Brittain JH, Gold GE, Beaulieu CH, Pelc NJ. Iterative decomposition of water and fat with echo asymmetry and least-squares estimation (IDEAL): application with fast spin-echo imaging. *Magnet Reson Med.* 2005; 54:636–44.
22. Schache AG, Koulouris G, Kofoed W, Morris HG, Pandy MG. Rupture of the conjoint tendon at the proximal musculotendinous junction of the biceps femoris long head: a case report. *Knee Surg Sports Traumatol Arthrosc.* 2008; 16(8):797–802. [PubMed: 18360748]
23. Schiltz M, Lehance C, Maquet D, Bury T, Crielaard J, Croisier J. Explosive strength imbalances in professional basketball players. *J Athl Training.* 2009; 44(1):39–47.
24. Schneider-Kolsky ME, Hoving JL, Warren P, Connell DA. A comparison between clinical assessment and magnetic resonance imaging of acute hamstring injuries. *Am J Sports Med.* 2006; 34(6):1008–15. [PubMed: 16476919]
25. Silder A, Heiderscheit B, Thelen D. MR observations of long-term musculotendon remodeling following a hamstring strain injury. *Skeletal Radiol.* 2008; 37(1):1101–9. [PubMed: 18649077]
26. Silder A, Thelen DG, Heiderscheit BC. Effects of prior hamstring strain injury on strength, flexibility, and running mechanics. *Clin Biomech.* 2010; 25:681–6.
27. Slavotinek JP, Verrall GM, Fon GT. Hamstring injury in athletes: using MR imaging measurements to compare extent of muscle injury with amount of time lost from competition. *Am J Roentgenol.* 2002; 179:1621–8. [PubMed: 12438066]
28. Sole G, Milosavljevic S, Nicholson H, Sullivan SJ. Selective strength loss and decreased muscle activity in hamstring injury. *J Orthop Sports Phys Ther.* 2011; 41(5):354–63. [PubMed: 21289455]
29. Tingart MJ, Apreleva M, Lehtinen JT, Capell B, Palmer WE, Warner JJ. Magnetic resonance imaging in quantitative analysis of rotator cuff muscle volume. *Clin Orthop Relat Res.* 2003; (415):104–10. [PubMed: 14612636]
30. Verrall GM, Kalairajah Y, Slavotinek JP, Spriggins AJ. Assessment of player performance following return to sport after hamstring muscle strain injury. *J Sci Med Sport.* 2006; 9:87–90. [PubMed: 16621702]
31. Yu H, Reeder SB, Shimakawa A, Brittain JH, Pelc NJ. Field map estimation with a region growing scheme for iterative 3-point water-fat decomposition. *Magnet Reson Med.* 2005; 54:1032–9.

Figure 1.

Manual segmentation was used to determine A) the bilateral volumes of the biceps femoris long head (BFLH), short head (BFSH), and semitendinosus (ST), as well as B) the tendon-scar tissue volumes of the proximal conjoint biceps femoris-semitendinosus tendon (BFT).

Figure 2.

Representative torque versus angle data for an injured limb, overlaid with a polynomial curve fitted to the top 10% of the torque measures. The increase in torque and decrease in angle of peak torque from time of return to sport (RTS) to the 6 month follow-up is shown. Error bars represent the nine averaged data points when the repeated cycles were compiled.

Figure 3. T2 weighted magnetic resonance image shows A) residual edema in the injured limb (left side of image) at return to sport, with B) no remaining edema present at the 6-month follow-up.

Table 1

Isokinetic strength testing measures (mean (SD)) at 60°/s and 240°/s for each limb performed at return to sport (RTS) and 6-months following RTS.

60°/s						
Limb	Peak Torque (Nm/kg)		Angle of Peak Torque (°) [†]		Work (J/kg)	
	RTS*	6-month	RTS	6-month	RTS*	6-month
Injured	1.15 (0.29)	1.30 (0.26)	39.9 (14.6)	28.8 (11.6)	107.0 (32.0)	126.8 (31.0)
Uninjured	1.28 (0.29)	1.28 (0.28)	40.1 (14.1)	30.0 (15.0)	116.6 (33.4)	124.1 (36.2)
240°/s						
Limb	Peak Torque (Nm/kg) [§]		Angle of Peak Torque (°)		Work (J/kg) [§]	
	RTS	6-month	RTS	6-month	RTS	6-month
Injured	0.74 (0.24)	0.80 (0.22)	43.2 (11.1)	40.3 (8.5)	16.7 (5.5)	18.8 (4.8)
Uninjured	0.81 (0.23)	0.85 (0.21)	45.1 (10.2)	41.8 (8.4)	19.0 (5.2)	20.1 (5.2)

* Peak torque (p<0.001) and work (p=0.003) at 60°/s were less in the injured limb than the uninjured limb at RTS.

[†] Angle of peak torque at 60°/s for both limbs decreased over time (p<0.001), reflecting a longer hamstring length.

[§] Peak torque (p=0.021) and work (p=0.006) at 240°/s were less in the injured limb than the uninjured limb.

Table 2

Volumes (mean (SD)) determined from MRI for the biceps femoris long head (BFLH), short head (BFSH) and semitendinosus (ST) muscles. Muscle and tendon volumes were estimated at return to sport (RTS) and 6-months following RTS.

	Limb	Volume		Change over Time (%)
		RTS	6 month follow-up	
BFLH	injured	237.2 (46.3)	229.3 (45.0)	-3.0 % (9.2 %)
	uninjured	244.9 (49.6)	236.9 (47.6)	-3.2 % (6.7 %)
BFSH ^{*†}	injured	113.1 (35.3)	103.5 (30.7)	-7.5 % (12.3 %)
	uninjured	103.0 (28.0)	97.7 (25.0)	-4.7 % (10.4 %)
ST [*]	injured	260.2 (61.6)	250.5 (63.1)	-3.3 % (11.2 %)
	uninjured	265.5 (66.9)	250.3 (59.2)	-4.9 % (8.2 %)

^{*} Main effect over time (BFSH, $p=0.010$; ST, $p=0.024$) indicating the muscle volume of both limbs decreased over time

[†] Main effect between limbs ($p=0.036$) indicating muscle volume of the injured limb was larger than the uninjured limb.