



Biomechanical response to hamstring muscle strain injury

Anthony G. Schache^{a,b,*}, Tim V. Wrigley^b, Richard Baker^{a,c,d}, Marcus G. Pandy^a

^aDepartment of Mechanical Engineering, University of Melbourne, Victoria 3010, Australia

^bCentre for Health, Exercise and Sports Medicine, School of Physiotherapy, University of Melbourne, Victoria 3010, Australia

^cHugh Williamson Gait Laboratory, Royal Children's Hospital, Melbourne, Victoria 3052, Australia

^dGait CCRE, Murdoch Childrens Research Institute, Royal Children's Hospital, Melbourne, Victoria 3052, Australia

ARTICLE INFO

Article history:

Received 13 May 2008

Received in revised form 9 September 2008

Accepted 3 October 2008

Keywords:

Sprinting

Kinematics

Joint torque

Joint power

Muscle-tendon length

ABSTRACT

Hamstring strains are common injuries, the majority of which occur whilst sprinting. An understanding of the biomechanical circumstances that cause the hamstrings to fail during sprinting is required to improve rehabilitation specificity. The aim of this study was to therefore investigate the biomechanics of an acute hamstring strain. Bilateral kinematic and ground reaction force data were captured from a sprinting athlete prior to and immediately following a right hamstring strain. Ten sprinting trials were collected: nine normal (pre-injury) trials and one injury trial. Joint angles, torques and powers as well as hamstring muscle-tendon unit lengths were computed using a three-dimensional biomechanical model. For the pre-injury trials, the right leg compared to the left displayed greater knee extension and hamstring muscle-tendon unit length during terminal swing, an increased vertical ground reaction force peak and loading rate, and an increased peak hip extensor torque and peak hip power generation during initial stance. For the injury trial, significant biomechanical reactions were evident in response to the right hamstring strain, most notably for the right leg during the proceeding swing phase after the onset of the injury. The earliest kinematic deviations in response to the injury were displayed by the trunk and pelvis during right mid-stance. Taking into account neuromuscular latencies and electromechanical delays, the stimulus for the injury must have occurred prior to right foot-strike during the swing phase of the sprinting cycle. It is concluded that hamstring strains during sprinting most likely occur during terminal swing as a consequence of an eccentric contraction.

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1. Introduction

Hamstring strains are common injuries [1,2], most of which occur whilst sprinting [2,3]. In order to optimise the rehabilitation and prevention of hamstring strains, exercise interventions must be specific to the mechanism of injury [4]. An understanding of the biomechanical conditions that cause the hamstrings to fail during sprinting is therefore of clinical significance.

The hamstrings are active throughout terminal swing and initial stance of the sprinting cycle [5–7]. Conjecture exists regarding the precise point when hamstring strains occur [4,8]. Some researchers have argued that the hamstrings are most biomechanically susceptible to injury during terminal swing [7,9–11]. Others have proposed initial stance to be the critical point [12,13]. As all of these studies are based on either theoretical rationale [10] or analyses of

asymptomatic subjects [7,9,11–13], they are unable to definitively establish when in the sprinting cycle the hamstrings fail.

In order to identify the biomechanical circumstances that lead to injury during sprinting, *in vivo* experimental data of an acute hamstring strain are ideally required. Unfortunately, such data are virtually impossible to generate practically. It is therefore not surprising that there exists only one published case study reporting biomechanical data of a running athlete captured at the time of an acute hamstring strain [14]. A 130 ms interval during terminal swing was identified in this study as the most likely time of injury. Whilst a unique insight into the potential timing of hamstring strains was obtained, the study was associated with several limitations. First, the injury occurred whilst the subject was running at a sub-maximal speed (5.36 m/s) on an inclined treadmill. Whether these results can be generalised to overground sprinting is difficult to determine. Second, as the subject was running on a treadmill, no ground reaction force (GRF) data were collected and thus relevant stance phase dynamics were not considered. Finally, the results are limited to the single subject evaluated. Before the conclusions can be scientifically accepted, verification by further independent experimental investigations is required.

* Corresponding author at: Department of Mechanical Engineering, The University of Melbourne, Victoria 3010, Australia. Tel.: +61 3 8344 6748; fax: +61 3 8344 4290.

E-mail address: anthony@unimelb.edu.au (A.G. Schache).

In the current study, bilateral kinematic and GRF data were captured from a sprinting athlete prior to and immediately following a right hamstring strain. These data were obtained unexpectedly during a routine quantitative gait analysis assessment conducted prior to the athlete returning to competition following previous right hamstring strains. The specific aims were to: (a) investigate whether pre-injury biomechanical asymmetries existed; (b) evaluate the biomechanical response to the injury and; (c) identify the timing and segmental location of the initial response. It was anticipated that this information would prove useful for generating hypotheses regarding the likely time of occurrence of the injury.

2. Materials and methods

2.1. Subject

The subject was an elite Australian Rules male football player (height: 186.0 cm; body mass: 91.5 kg; age: 20.3 years). Written informed consent was obtained to analyse data for research purposes and approval was obtained from the institutional

Human Research Ethics Committee. The subject was participating in a quantitative gait analysis assessment. He was suffering from recurrent right hamstring strains. The first injury occurred 67 days prior to the assessment and was re-aggravated 42 days later. Both injuries occurred whilst sprinting during competition. The subject had no other history of hamstring strains, nor was there a history of any associated medical problems. At the time of the assessment, the subject was participating fully in his usual training activities and was scheduled to return to competition.

The subject performed repeated sprints over ~30 m on an indoor running surface. Nine sprints were completed unimpeded and symptom-free. However, during the 10th sprint the subject sustained a right hamstring strain. Sudden pain was experienced that was severe enough for him to grasp his posterior thigh and quickly decelerate. Immediately following the injury, a thorough clinical examination was performed by an experienced physiotherapist (AS). Palpable tenderness as well as pain and weakness with resisted contraction were all evident. Magnetic resonance imaging performed 24 h after the injury revealed a significant and complex injury involving the proximal musculotendinous junction of the biceps femoris long head and semitendinosus muscles [15].

2.2. Instrumentation

Kinematic and GRF data were collected for all 10 sprint trials. Kinematic data were acquired using a three-dimensional (3D) motion analysis system (VICON 612,

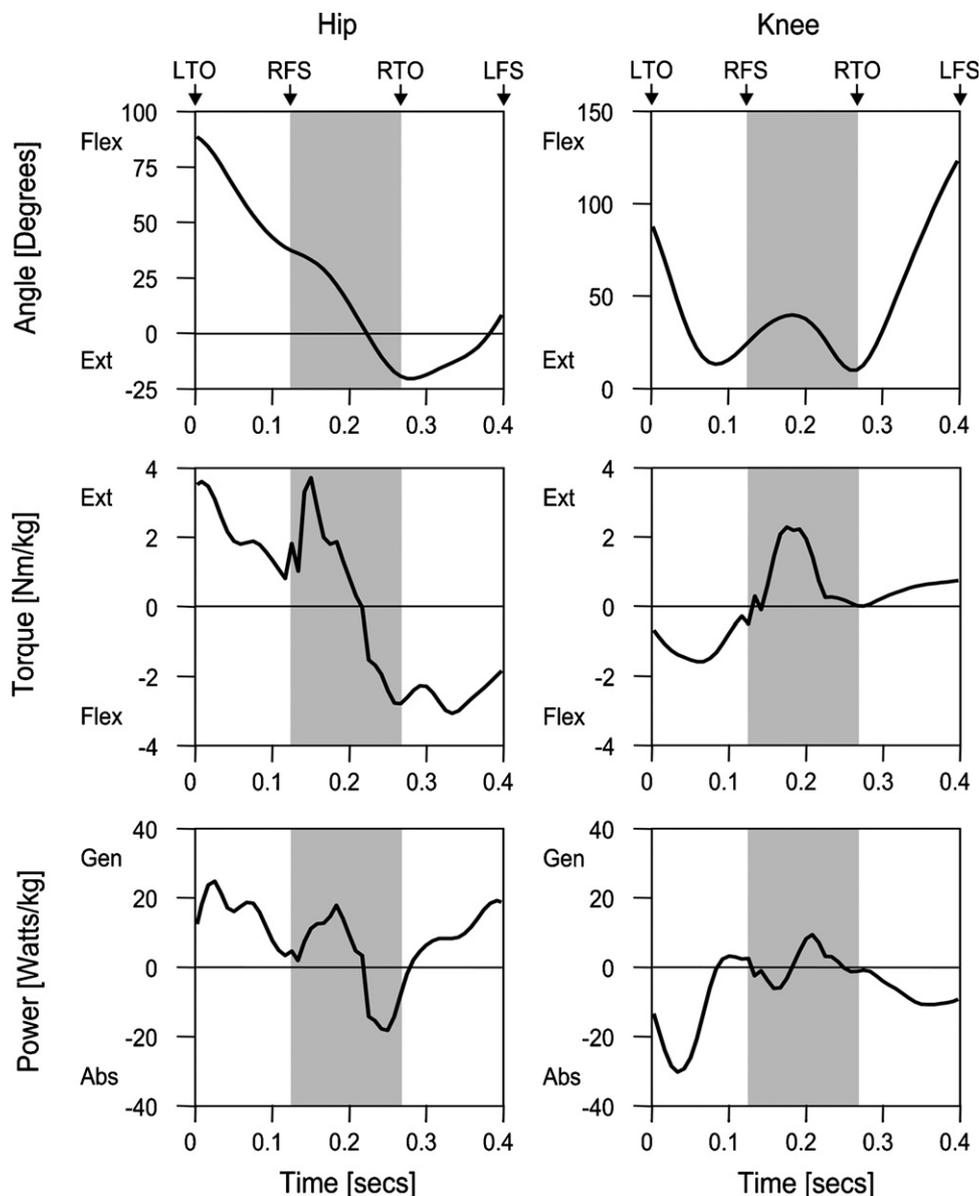


Fig. 1. Terminal swing, stance phase and initial swing hip and knee joint kinematics and kinetics during sprinting for the right leg. The grey shaded region represents stance phase. Data are for a single representative pre-injury trial from contra-lateral (left) toe-off to contra-lateral (left) foot-strike. LTO, left toe-off; RFS, right foot-strike; RTO, right toe-off; LFS, left foot-strike.

Oxford Metrics, Oxford, UK) with eight M2 cameras sampling at 120 Hz. Two AMTI force-plates (Advanced Mechanical Technology Inc., Watertown, MA, USA) were used to capture GRF data at 1080 Hz. Both force plates were centred within a calibrated measurement field of ~4 m in length.

2.3. Procedures

Reflective markers (14 mm diameter) were mounted on the subject's trunk, pelvis and lower limbs (Appendix A, Supplementary Table 1). Technical frame definitions are outlined in Appendix A, Supplementary Table 2. An initial static trial was performed to calibrate relevant anatomical landmarks and establish joint centres. The hip joint centre was defined as per Harrington et al. [16], whilst the orientation of the knee flexion-extension axis was determined using a dynamic optimisation procedure [17]. Anatomical frame definitions are outlined in Appendix A, Supplementary Table 3.

The subject wore standard athletic shorts and running sandals (NIKE Strapranner IV) that allowed adequate exposure of the foot for marker placement. After a brief warm up that consisted of walking and slow jogging, the subject performed repeated sprints through the centre of the laboratory. At least 3 min rest was provided between trials to avoid fatigue. Each trial contained a valid foot-strike on one of the force-plates for a single leg. Five trials were collected for both the left and right legs in the following order: left–left–left–right–right–right–right–left–left.

2.4. Data analysis

Coordinate data were filtered using Woltring's general cross-validatory quintic smoothing spline [18] with a predicted mean-squared error of 15 mm. Hip and knee joint angles were computed as per Grood and Suntay [19]. Trunk and pelvis angles were computed as per Baker [20]. Trunk angles were described relative to the pelvis anatomical frame, whilst pelvis angles were described relative to the laboratory frame. Internal joint torques and powers were calculated using a standard inverse dynamics approach and were expressed in a non-orthogonal reference frame or joint coordinate system. Hamstring muscle-tendon unit length was calculated as follows. The attachment coordinates for the semitendinosus, semimembranosus and biceps femoris long head muscles were obtained from the 3D lower limb musculoskeletal model of Delp et al. [21] and available via the International Society

of Biomechanics website (<http://isbweb.org/data/delp/index.html>). The pelvis and tibia anatomical frames (Appendix A, Supplementary Table 3) were first redefined to coincide with those used in the model to describe the 3D attachment coordinates for the hamstrings. Next, the attachment coordinates were scaled as a percentage of the model's segment length, i.e., hip joint centre to hip joint centre distance for the pelvic coordinates; knee joint centre to ankle joint centre distance for the tibial coordinates. These scaled attachment coordinates were then multiplied by the relevant segment length (as computed from the static calibration trial) for the subject in the current study. Finally, the attachment coordinates were transformed into the laboratory frame, and hamstring muscle-tendon unit length was estimated as the distance between origin and insertion along the straight-line path of that muscle.

3. Results

3.1. Pre-injury trials

The subject's average (± 1 S.D.) sprinting speed for the nine pre-injury trials was 7.44 ± 0.10 m/s. The critical period during sprinting for understanding hamstring muscle function is from mid-swing until mid-stance. From mid-swing onwards, the hip and knee joints were both extending, with knee extension occurring at a faster rate than hip extension. During terminal swing, the hip continued to extend, whilst the knee reached peak extension and began flexing just prior to foot-strike (Fig. 1, top panels). The hamstrings contributed to the generation of a large hip extensor torque during terminal swing and initial stance (Fig. 1, left middle panel). Immediately following foot-strike, the GRF caused a rapid peak to develop in the hip extensor torque, which averaged $4.16 (\pm 0.84)$ Nm/kg for the right leg during the pre-injury trials. The hamstrings also contributed to the generation of a knee flexor torque during terminal swing (Fig. 1, right middle panel). The hip

Table 1
Biomechanical parameters for the pre-injury trials and the injury trial.

Parameter	Pre-injury trials		Injury trial	
	Right	Left	Right	Left
Spatio-temporal				
Stance time (s)	0.14 (0.00)	0.14 (0.00)	0.14	0.16
Velocity (m/s)	7.47 (0.06)	7.41 (0.14)	6.93	6.93
Joint angle				
Trunk flexion angle at foot-strike (°)	18.17 (2.87)	14.91 (3.86)	-11.85	7.09
Peak swing hip flexion angle (°)	92.60 (2.04)	92.24 (2.60) ^a	35.53	81.06
Hip flexion angle at foot-strike (°)	38.85 (2.30)	39.22 (1.44)	17.12	42.72
Peak swing knee extension angle (°)	17.75 (4.47)	23.45 (1.15)	5.55	17.50
Knee extension angle at foot-strike (°)	27.58 (2.63)	26.15 (0.71)	7.11	15.49
Normalised muscle-tendon unit length				
SM peak length (%)	11.60 (1.14)	9.70 (0.83)	7.18	10.23
Time SM peak length pre-foot-strike (ms)	56.67 (3.73)	39.58 (7.98)	16.68	8.33
ST peak length (%)	11.10 (1.14)	8.69 (0.77)	6.74	9.28
Time ST peak length pre-foot-strike (ms)	56.67 (3.73)	43.75 (4.17)	16.67	8.33
BF peak length (%)	12.90 (0.73)	10.63 (0.67)	6.52	10.25
Time BF peak length pre-foot-strike (ms)	61.67 (4.56)	50.00 (6.80)	66.67	16.67
Ground reaction force (GRF)				
Peak vertical GRF (BW)	3.34 (0.23)	3.13 (0.14)	-	>3.14 ^c
Peak vertical GRF loading rate (BW/s)	170.03 (32.39)	98.43 (5.82)	-	>365.38 ^c
Vertical GRF impulse (BW × s)	0.27 (0.01)	0.25 (0.01)	-	>0.28 ^c
Peak posterior (braking) GRF (BW)	0.41 (0.13)	0.37 (0.05)	-	>0.79 ^c
Peak anterior (propulsive) GRF (BW)	-0.72 (0.04)	-0.75 (0.03)	-	-0.29
Joint torques and powers				
Peak swing hip extension torque (Nm/kg)	3.41 (0.20)	3.38 (0.14) ^b	1.69	3.62
Peak swing hip power absorption (W/kg)	-13.13 (3.45) ^a	-19.97 (6.19) ^b	-8.20	-
Peak swing hip power generation (W/kg)	23.40 (1.56)	25.48 (3.60)	2.68	25.37
Peak swing knee flexion torque (Nm/kg)	-1.50 (0.07)	-1.47 (0.11)	-0.21	-1.57
Peak swing knee power absorption (W/kg)	-26.53 (2.46)	-25.21 (2.98)	-3.46	-31.35
Peak stance hip extension torque (Nm/kg)	4.16 (0.84)	3.66 (0.52)	-	>3.47 ^c
Peak stance hip power generation (W/kg)	20.56 (3.44)	15.85 (1.14)	-	>17.99 ^c

Dashed line (-) indicates data not available. SM, semimembranosus; ST, semitendinosus; BF, biceps femoris long head. All data for the pre-injury trials represent the average (± 1 S.D.) of five and four trials for the right and left legs, respectively, except: ^a data available for three pre-injury trials only and; ^b data available for two pre-injury trials only. ^c Exact magnitudes could not be determined due to saturation of the force-plate in the injury trial.

extensor torque during terminal swing and initial stance was associated with positive work/concentric muscle function, whilst the knee flexor torque during terminal swing was associated with negative work/eccentric muscle function (Fig. 1, bottom panels).

The hamstring muscle-tendon unit lengthened throughout the second half of swing (Appendix A, Supplementary Fig. 1). Lengthening commenced at the end of initial swing (~49% of the sprint cycle) and peaked during terminal swing (~90% of the sprint cycle). The average peak percentage increase in muscle-tendon unit length (calculated with reference to an upright stance position) ranged from 8.69% to 12.90% for the three hamstrings (Table 1). The percentage increase in length was greatest for biceps femoris long head when compared to semitendinosus and semimembranosus (Table 1).

Biomechanical asymmetries were evident in the data for the pre-injury trials. Of the kinematic parameters, trunk flexion at right foot-strike was increased by 3.3° compared to that at left foot-strike and peak knee extension during terminal swing was 5.7° greater for the right leg compared to the left (Table 1). Peak hamstring muscle-tendon unit length was also greater and it occurred between 11.7 ms (biceps femoris long head) and 17.1 ms (semimembranosus) earlier in swing for the right leg compared to the left (Table 1). Of the kinetic parameters, the vertical GRF peak

and loading rate were increased by 7% and 73%, respectively, for the right leg compared to the left (Table 1 and Appendix A, Supplementary Fig. 2). Furthermore, peak hip power absorption was decreased by 34% during swing, whilst peak hip extension torque and peak hip power generation were increased by 14% and 30%, respectively, during initial stance, for the right leg compared to the left (Table 1).

3.2. Injury trial

Significant biomechanical reactions occurred in response to the right hamstring strain. For example, the kinematic patterns of the trunk, pelvis and right hip for the injury trial displayed appreciable deviations away from their typical patterns for the pre-injury trials (Table 1 and Fig. 2). Peak swing hip flexion for the right leg averaged 92.6° for the pre-injury trials, whereas it was 35.5° for the injury trial. Substantial reductions were also evident in the right hip and knee joint torques and powers during swing for the injury trial in comparison to the pre-injury trials (Table 1 and Fig. 3). Most notably, peak knee power absorption for the right leg during the injury trial (-3.46 W/kg) was decreased by 87% compared to the pre-injury trials (-26.53 W/kg), demonstrating the intolerance of the right hamstrings to perform negative work about the knee joint following

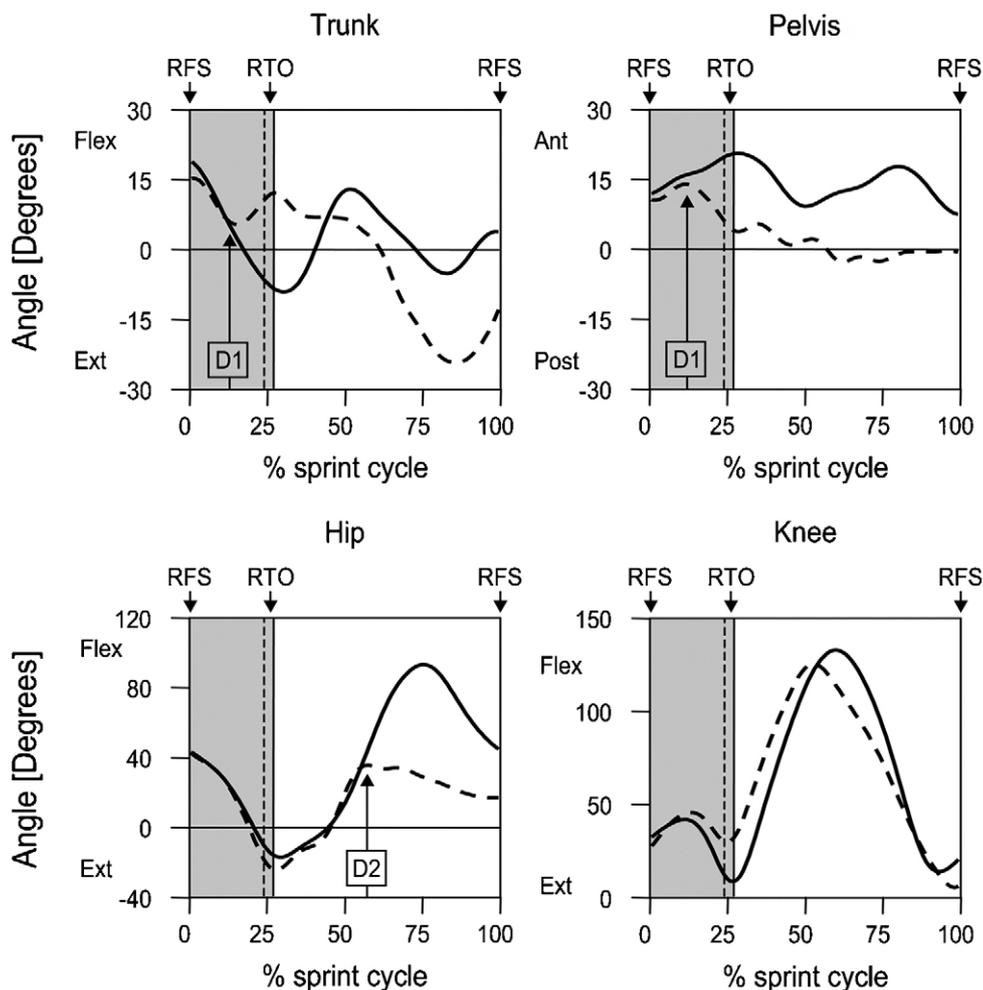


Fig. 2. Sagittal plane kinematic data, time normalised as a percentage of the full stride cycle during sprinting, for the right leg. Data are for a single representative pre-injury trial (thick solid line) compared to the injury trial (dashed line). The grey shaded region represents stance phase. The solid and dashed vertical lines indicate toe-off for the pre-injury trial and injury trial data, respectively. D1 denotes the time of the first major kinematic deviation evident in the injury trial data, which occurred for the trunk and pelvis at 15% and 11% of the sprint cycle, respectively. D2 denotes the time of the second major kinematic deviation evident in the injury trial data, which occurred for the hip at 57% of the sprint cycle. RFS, right foot-strike; RTO, right toe-off.

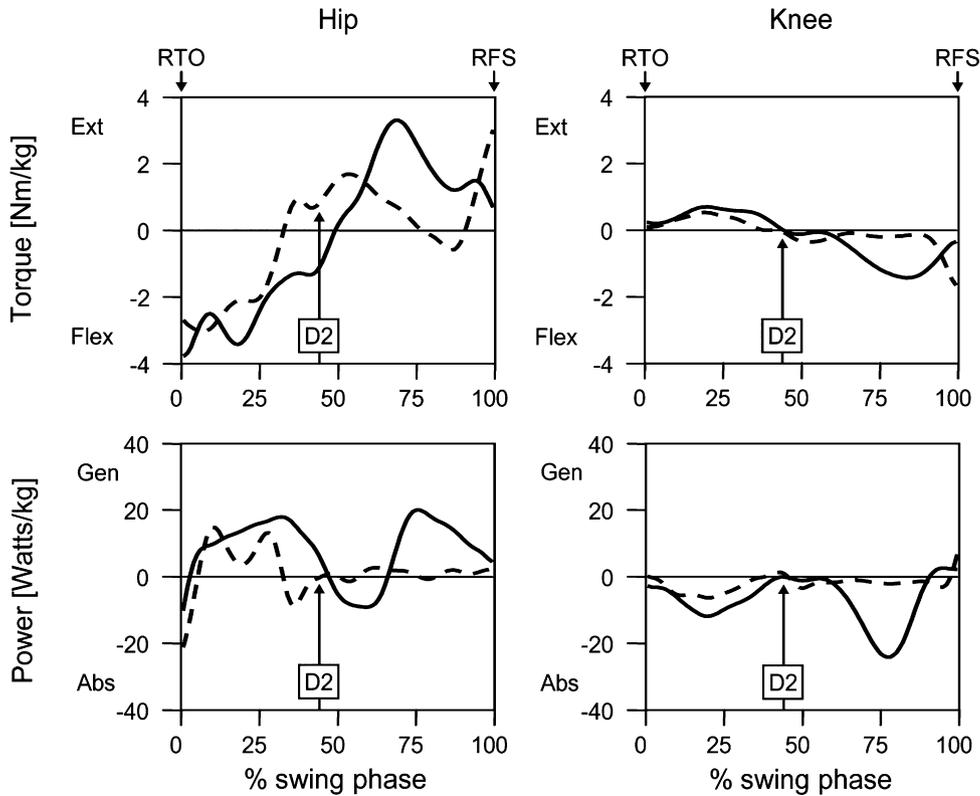


Fig. 3. Swing phase hip and knee joint torques and powers during sprinting, time normalised as a percentage of the swing phase, for the right leg. Data are for a single representative pre-injury trial (thick solid line) compared to the injury trial (dashed line). The time of D2 (the second major kinematic deviation evident in the injury trial data from Fig. 2) is displayed. Note the dramatically reduced knee flexor torque and knee power absorption after D2 in the injury trial data compared to the pre-injury trial data (see text for further explanation). RFS, right foot-strike; RTO, right toe-off.

the injury. Finally, foot-strike for the left leg during the injury trial was associated with dramatically increased GRFs, so much so that the maximum amplifier range of the force-plate was saturated for a brief period during initial stance in the vertical and posterior

(braking) directions (Table 1). This was a consequence of the subject attempting to quickly decelerate following the onset of the injury.

The various critical events and their timing during the injury trial are depicted in Fig. 4. The trunk and pelvis kinematic

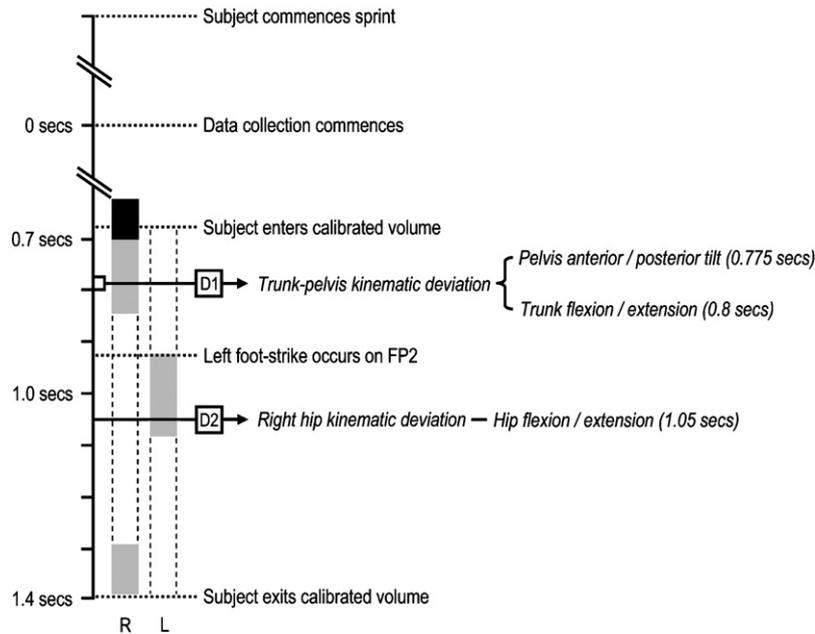


Fig. 4. Timeline depicting the series of events for the injury trial during which the right hamstring strain occurred. 'R' denotes right leg; 'L' denotes left leg. The grey shaded columns indicate the stance phases and the open columns with dashed lines indicate the swing phases. The most likely time when the stimulus for the injury took place was during the swing phase preceding the first right foot-strike, the terminal region of which is indicated by the solid black column. The time of the first (D1) and second (D2) major kinematic deviations evident in the injury trial data (from Fig. 2) are also indicated.

deviations (D1) occurred 75 ms and 100 ms, respectively, after right foot-strike. The right hip kinematic deviation (D2) occurred during mid-swing, 350 ms after right foot-strike. Assuming that D1 represented the earliest measurable biomechanical reaction, the stimulus responsible for the right hamstring strain must have occurred prior to this point (i.e., prior to right mid-stance).

4. Discussion

Given that the subject had recently suffered two right hamstring strains but had never injured his left hamstring, the quantitative gait analysis assessment was conducted to determine if biomechanical asymmetries were present in his sprinting gait. It was thought that such knowledge might prove useful clinically to identify potential contributing factors and develop subject-specific therapeutic interventions. Interestingly, asymmetries were found in the data for the pre-injury trials (Table 1 and Appendix A, Supplementary Fig. 2). Due to increased knee extension, hamstring muscle-tendon unit length was greater and occurred earlier during terminal swing for the right leg compared to the left. This may have resulted in larger right hamstring muscle fibre strains, the magnitude of which has been shown to relate to muscle damage [22,23]. The increased vertical GRF peak and loading rate, as well as the increased peak hip extensor torque and peak hip power generation, during initial stance for the right leg compared to the left is consistent with previous studies that have found increased stance phase hip extensor torques for sprinters with a past history of hamstring strains [12,13]. However, the cause-effect relationship between the evident biomechanical asymmetries and the recurrent right hamstring strains cannot be determined.

The precise time between the onset of the injury and the measured response (i.e., the neuromuscular latency) is difficult to predict with any certainty. Experiments measuring human neuromuscular latencies in response to cutaneous stimulation have found the elicited reflex strategies and thus latency times to be location-, intensity- and task-dependent. Perhaps the most relevant estimate in the context of this study can be retrieved from Tax et al. [24]. These researchers recorded the reflex responses of both ipsi- and contra-lateral lower limb biarticular muscles following non-nociceptive sural nerve stimulation of varying intensity for 11 normal adults running on a treadmill. Electromyographic responses across all muscles and subjects had a latency of ~80 ms and duration of ~30 ms. As D1 and D2 in the current study were not based on muscle activity, the time required for a change in muscle activity to result in a kinematic deviation (i.e., electromechanical delay) must also be taken into account. Thus, based on the findings of Tax et al. [24] and the need to consider an electromechanical delay, it was deemed conservative to assume that the time between the onset of the right hamstring strain and the measured response was ≥ 110 ms. Given that D1 occurred 75 ms after right foot-strike (Fig. 4), it is concluded that the stimulus for the injury must have taken place during the preceding swing phase.

Only one previous study has published *in vivo* experimental data of an acute hamstring strain [14]. In this instance, whole body kinematic data were captured from a 31-year-old male professional skier who sustained a mild right hamstring strain whilst running on an inclined treadmill. Heiderscheit et al. [14] found the right greater trochanter marker trajectory to display the earliest indication of injury, with a large deviation in its cyclic behaviour apparent during right mid-stance, 100 ms after foot-strike. Remarkably, these results from Heiderscheit et al. [14] are virtually identical to those from the current study in terms of the segmental location and the timing of the initial reaction to the injury. Therefore, despite distinctly different injury mechanisms, data

from Heiderscheit et al. [14] and this study both implicate swing rather than stance as the most likely time of injury.

It is hypothesised that the hamstrings are susceptible to injury during terminal swing. This is for several reasons. First, the hamstrings appear to be most biomechanically exposed during terminal swing. Most of the inertial force acting about the knee joint at this time is potentially imparted onto the hamstrings as they attempt to decelerate the swinging shank. The gastrocnemius is the only other significant muscle capable of providing some assistance in this capacity [25]. In contrast, a number of large muscles are likely to contribute to the generation of the hip extensor torque during initial stance. It has been demonstrated that <50% of the total hip extensor torque for a variety of different functional tasks is actually generated by the hamstrings [26–28]. Second, peak hamstring electromyographic activity during sprinting has been shown to occur during terminal swing [5–7]. Third, the hamstring muscle-tendon unit undergoes an active lengthening contraction during terminal swing [7,11]. Whilst it is not known whether this lengthening is attributable to the tendon or muscle (or both), eccentric contractions, unlike concentric contractions, have been shown to be capable of producing muscle fibre damage [22,23]. Furthermore, it has been suggested that eccentric contraction-induced damage represents the starting point for major muscle strain injuries [29].

It is acknowledged that the biomechanical model utilised in the current study was associated with certain errors (e.g., soft tissue artefact) and assumptions (e.g., location of relevant anatomical landmarks and a straight line approach for computing hamstring muscle-tendon unit length). However, the model did generate satisfactory outcomes. Joint kinematics, torques and powers were consistent with previous studies investigating sprinting [5,7,13,30] as were hamstring muscle-tendon unit length estimates [7,11]. The within-subject study design and the analysis of sagittal plane dynamics only also strengthened the validity of our conclusions. Whilst limited to a single subject, the data are highly novel, and thus of significance. If our results are interpreted in conjunction with those from Heiderscheit et al. [14], valuable insights are obtained into the biomechanics of hamstring strains.

Conflict of interest statement

There are no conflicts of interest associated with this research.

Acknowledgements

There were no sources of funding associated with this research. We wish to thank Professor David L. Morgan for reviewing an earlier version of this manuscript.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.gaitpost.2008.10.054.

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