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Factors Associated with Recurrent Hamstring Injuries

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Abstract

A history of muscle injury represents a predominant risk factor for future insult in that muscle group. The high frequency of re-injury and persistent complaints after a hamstring strain comprise major difficulties for the athlete on return to athletic activities. Some of the risk factors associated with the possible recurrence of the injury are, in all probability, already implicated in the initial injury. One can distinguish between those events peculiar to the sport activity modalities (extrinsic factors) and other contributing factors based on the athletes individual features (intrinsic factors). For both categories, the persistence of mistakes or abnormalities in action represent an irrefutable component contributing to the re-injury cycle. Additional factors leading to chronicity can come from the first injury *per se* through modifications in the muscle tissue and possible adaptive changes in biomechanics and motor patterns of sporting movements. We emphasise the role of questionable approaches to the diagnosis process, drug treatment or rehabilitation design.

To date, the risk factors examined in the literature have either been scientifically associated with injury and/or speculated to be associated with injury. In this context, quantifying the real role of each factor remains hypothetical, the most likely ones corresponding to inadequate warm-up, invalid structure and the content of training, muscle tightness and/or weakness, agonist/antagonist imbalances, underestimation of an extensive injury, use of inappropriate drugs, presence of an extensive scar tissue and, above all, incomplete or aggressive rehabilitation. Such a list highlights the unavoidable necessity of developing valid assessment methods, the use of specific measurement tools and more rigorous guidelines in the treatment and rehabilitation. This also implies a scientific understanding as well as specifically qualified medical doctors, physiotherapists and trainers acting in partnership.

Muscle injury can be classified into three different forms depending on the occurrence mechanism: lacerations, contusions or strains.[1] Hamstring injuries are usually strains that most commonly occur proximally near the muscle tendon junction and laterally injure the biceps femoris.[2,3] The insult is a common occurrence in specific ranges of sports based on rapid active knee extension (e.g. sprinting, track and field, jumping, soccer, football, rugby) and/or gesture imposing position of maximal muscle lengthening (e.g. martial arts, dance, water-skiing).[2,4-10] These sports share either a passive stretch or solicit a protective eccentric action (tension development while lengthening) to the hamstring muscles decelerating the lower leg.[2]

The circumstances under which injury takes place correspond to the periods at which the athlete surpasses the mechanical limits tolerated by the muscle unit. Hamstrings are characterised by a very particular behaviour toward high intensity contractions and injury. Not only do hamstrings appear particularly sensitive to delayed onset muscular soreness (DOMS) following bouts of eccentric exercises,[11] but have a pronounced tendency to relapse after an initial strain.

Preventing both the first injury and re-injury represent a seemingly normal, but complex, target. The common denominator between the two is the need for the identification of the factors leading to injury and the development of the most appropriate preventive strategy. Despite a plentiful scientific literature and the willingness of trainers and therapists, the problem remains acute. A number of possible causes for an initial hamstring injury have been postulated, including their polyarticular characteristic and high percentage of fast-twitch fibres, inadequate flexibility, muscle weakness and strength imbalance, unsatisfactory warm-up, excessive fatigue, disturbed posture and dyssynergic contraction.[1,4,10,12-21]

Orchard[22] distinguishes between extrinsic factors principally inherent to sport activity (environment-related) and intrinsic factors related to individual features and profile (player-related). He suggests that intrinsic factors are more predictive of muscle strain than the extrinsic factors. The review by Parkkari et al.[23] confirmed that strategies designed to prevent sports injuries can be effective, but some measures may have no effect or even negative effects. The analysis of the predisposing factors of muscle strain themselves show contradictions, complicating the probability for a consensus management. For instance, Bennell et al.[24] asserted that isokinetic muscle strength testing was not able to directly identify Australian Rules football players at risk for hamstring muscle injury. Other authors have found that muscle deficits do contribute to injury. Orchard et al.[25] concluded that pre-season isokinetic testing of professional football players can identify those players at risk of developing hamstring muscle strains. In a recent study dedicated to soccer players, Croisier et al.[26] reported a risk factor of hamstring injury fixed at 15% in the presence of pre-season isokinetic strength imbalance versus only 3% in the absence of strength abnormality.

The high rate of re-injury[19,25,27] and lingering discomfort after the return to athletic activities can pose complex problems for trainers as well as sports

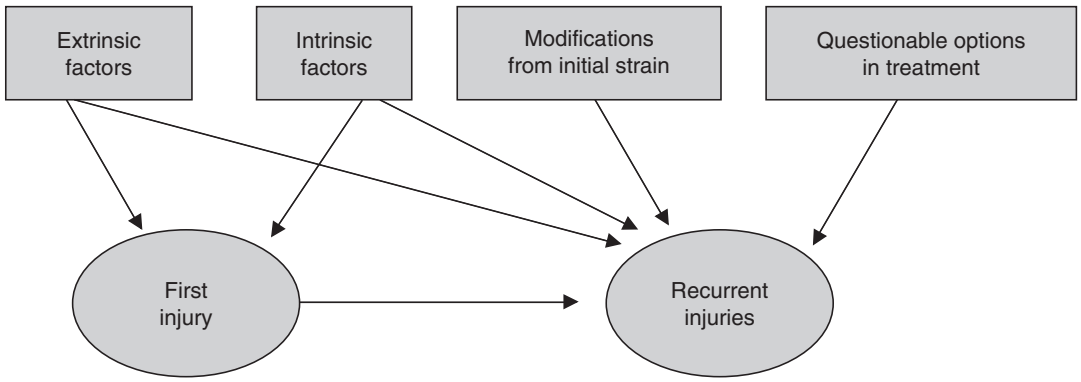

Fig. 1. The groups of factors associated with hamstring muscle strains.

medicine clinicians. This was further investigated through a prospective epidemiological study of the injuries sustained in English professional football (2376 players) over two competitive seasons; re-injuries accounted for 48% of injuries classified as strains.[28] Hamstring injuries have the highest recurrence rate of all injuries: 34% of the incidence of new hamstring strains.[29] In a study of the Australian Football League, Orchard and Best[30] showed that the persistence of a significantly increased risk of recurrence lasted for many weeks after a return to play. The cumulative risk of the recurrence of a hamstring strain for the remainder of the season was 30.6%. Interestingly, Hawkins et al.[28] demonstrated that injury and re-injury to the hamstring primarily occurred during competition compared with training (67% and 33%, respectively) in 749 strains studied.

There is a paucity of research relating to the real causes of repetitive hamstring injury. Most studies[22,31,32] have confirmed that a history of injury to a muscle group is a predominant risk factor for future injury in that group. Ordinarily, authors speculate that aetiology combines factors already implicated in the initial strain and additional specific abnormalities resulting from the insult itself. The aim of this article is to analyse the factors associated with recurrent hamstring strain in order to update the management of that relapsing injury.

1. Factor Analysis

The misinterpretation of aetiological factors, as well as the lack of a general consensus regarding both treatment and rehabilitation, give rise to muscle strain recurrence and persistence after an initial hamstring muscle strain in speed and flexibility athletes. This statement highlights that the clarification of factors leading to injury and re-injury need to represent the cornerstone of the practitioner approach. To date, only a few factors have been scientifically associated with injury, while others have been suggested as being implicated.

In this article, we label these factors (figure 1) as:
1. extrinsic factors;
2. intrinsic factors;
3. modifications from initial strain;
4. questionable options in treatment.

Such a structure could lead us to analyse the aetiological factors independently of one another; we emphasise the necessity of considering the situation as a whole as injury factors may be multiple and interact.

The theoretical model proposed by Worrell[33] suggests that the combination of abnormalities (strength, flexibility, warm-up, fatigue) increases the likelihood of hamstring strain. On the other hand, some factors may be present without re-injury occurring. There may be a threshold at which a greater number of risk factors would produce an injury, or some factors may be more predictive of injury than others.[34]

We propose a list of factors possibly associated with recurrent hamstring injuries and their respective estimated role (see table I).

1.1 Extrinsic Factors

Some factors peculiar to a sport activity contribute to a first episode of injury. Therefore, it is logical to assume that, despite possible well designed rehabilitation, persisting errors in sport training modali-

Table I. Factors associated with recurrent hamstring injuries and their respective estimated role

Factors	Degree of involvement
Extrinsic factors	
Inadequate warm-up	+++
Fatigue related to unsuitable structure and content of training	+++
Intrinsic factors	
Agonist/antagonist imbalances	+++
Lack of flexibility	+++
Age	+
Low back pain	+
Sacroiliac joint dysfunction	+
Chronic hormone imbalance	+
Modifications from initial strain	
Tightness and/or weakness	+++
Extensive scar tissue	++
Muscular calcification	+
Changes in the biomechanics and motor patterns of sporting movements	+
Neural inhibition	+
Psychological stress	?
Adverse neural tension	?
Questionable options in treatment	
Incomplete or aggressive rehabilitation	+++
Underestimation of an extensive injury	++
Prescription of glucocorticoids	++
Use of local anaesthetic	++
Inappropriate prolonged use of NSAIDs	?

+ indicates low level of involvement; **++** indicates medium level of involvement; **+++** indicates high level of involvement; **?** indicates level of involvement unknown.

ties will give rise to re-injury through the same factors.

1.1.1 Warm-Up

Absent or inadequate warm-up represents a causative event commonly reported in the literature.[2,32,33] This unavoidable stage of actively initiating the sport session facilitates increases in connective tissue extensibility (via the modifications of viscoelastic properties), which is temperature dependent.[2] In an animal model,[35] muscles were held isometrically and tetanically stimulated for 10 to 15 seconds, provoking a 1°C rise in muscle temperature. As a result, muscles could stretch more before failure and could produce increased force. Stretching before exercise seems critical because of the capability of the musculotendinous unit to absorb energy is directly proportional to both resting length and muscle temperature.[35,36]

In 1999, Hawkins and Fuller[32] hypothesised that the higher proportion of strains observed in professional football players playing for English clubs, compared with that reported for European teams, could be the result of less attention being paid to general body conditioning, in particular warm-ups.

1.1.2 Fatigue

The role of fatigue is frequently suggested, as hamstring strains regularly occur late in training as well as in the competitive settings.[2] Worrell[33] suggested that strain also occurs later or soon after a fatiguing practice. Fatigue may induce physiological changes within the muscle, as well as altered coordination, technique or concentration, predisposing the player to injury.[34] In particular, the dual innervation of biceps femoris could lead to asynchrony in the activation of separate parts of the muscle and result in inefficiencies. Abnormalities in running style may be the consequence of fatigue, increasing the workload of the stabilising biarticular muscles around the pelvis. This situation amplifies fatigue and predisposes the athlete to subsequent injury, justifying the technical education of the athlete.

Cooling down procedures may also influence residual fatigue on subsequent days. With the goal of preventing fatigue and decreasing work capacity, the correction of fluid balance[37] and ingestion of liquid carbohydrate drinks facilitating glycogen replenishment is sometimes recommended.[33] Thus, ingestion of approximately 30–60g of carbohydrates during each hour of exercise would be sufficient to maintain blood glucose oxidation late in exercise and delay fatigue.[38]

Experimental work by Mair et al.[14] indicated that fatigued rabbit muscles had a decreased ability to absorb energy before reaching the amount of stretch that causes injuries. The amount of energy absorbed by the fatigued muscle was 69.7–92% that of the energy absorbed in the control muscle. The lowest level of energy absorption corresponded to the more fatigued muscles. Furthermore, muscles were injured at the same length, regardless of the effects of fatigue.

Fatigue management (i.e. proper conditioning to avoid fatigue and exercise modification when fatigue occurs[14]) is considered an important part of the rationale for preventing muscle injury and re-injury.[2] We particularly refer to the modalities of training or competition in relation to the specific physiological demand of the activity.

1.1.3 Fitness Level and Training Modalities

Inadequate pre-season training resulting in low fitness levels may contribute to an increased injury rate.[32,39] In general, sprint training should be utilised on an adequate foundation for general fitness and implemented slowly from walking drills.[34] It is well documented that abrupt increases in training volume may further contribute to injury risk.[40] In soccer, at least 90% of the energy release must be aerobic, even if players run at an intensity close to the anaerobic threshold.[41]

Inadequate training modalities, favouring long periods above the anaerobic threshold instead of specific exercise improving maximum oxygen consumption ($\dot{V}O_{2max}$) [such as aerobic interval training], may contribute to increase the (re)injury rate. Heart rate monitoring as an indicator of actual exercise intensity during training offers the possibility of individualised surveillance. Specialised sessions aimed at developing muscular explosivity and speed (qualities required in soccer) through short-lasting exercises, must consider the need for recovery periods between exercise sequences, a precaution that is regularly neglected and leads to unsuitable training conditions. Obviously, some activities (such as 400m sprints or 400m hurdles in athletics) require exercising muscle to bear accumulating lactate by means of specific exercise modalities.

Persistent errors in training modalities and conditions inherent to sport activity represent an irrefutable factor contributing to recurrent injury and this highlights the necessity for trainers to have scientific knowledge and qualifications, most notably in the exercise physiology area. A recent study by Junge et al.[42] suggests that an injury prevention programme improved both the structure and content of training. For thigh muscles, the average number of injuries per player per year was reduced in a group benefiting from a prevention programme (0.15 ± 0.38) compared with a control group (0.26 ± 0.55). The education of coaches and players regarding training protocols as well as injury prevention strategies[37,42,43] is an important factor in injury prevention.

1.2 Intrinsic Factors

Quantifying the respective role of each parameter is complicated and illustrates this multifactorial problem. The real determination of predisposing factors to re-injury rest partially on theoretical assumptions because of the sparse scientific documentation currently available.[20] Croisier and Crielaard[7] isokinetically demonstrated frequent muscle strength performance disorders (about 70% of cases) in the context of hamstring muscle strains with persistent discomfort, suggesting that recurrent injuries may be the consequence of inadequate rehabilitation. Nevertheless, it remained unclear whether strength performance disorders were solely the consequence of the initial injury or a current causative factor for re-injury, or both.

Some authors[44,45] have concluded that isokinetic testing could identify muscle imbalances and allow correction, but the specific criteria for 'adequate' strength is largely undefined. For instance, Noonan and Garrett[10] recommended a cut-off of 80% of the strength on the contralateral side in isokinetic testing. Considering that the initial strain may already arise from a strength weakness, allowing an athlete to return to competition with a 20% deficit after injury in the bilateral comparison represents, in our opinion, a factor predisposing the player to re-injury. Other authors[44] suggest the restoration of the injured leg strength to within 10% of that of the unaffected leg.

1.2.1 Eccentric Deficits

According to Jönhagen et al.,[4] Croisier and Crielaard[7] confirmed the discriminating character of the eccentric trial by combining a preferential eccentric peak torque deficit and a significant reduction of an original mixed[46] eccentric flexors/concentric quadriceps ratio (figure 2). An ordinary concentric protocol would not have revealed exclusive eccentric deficits in 23% of patients. In that context, recommendations by Clanton and Coupe,[44] who advised clinicians against using the eccentric mode of contraction, may appear illogical and we draw

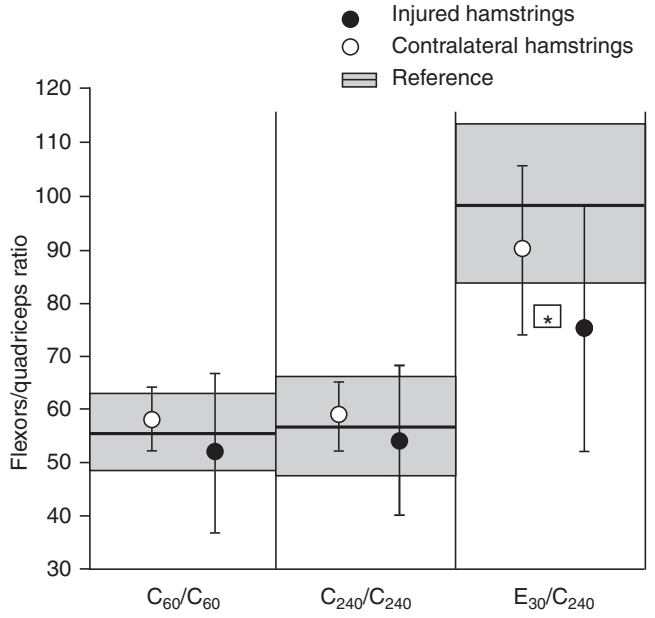

Fig. 2. Comparative study between healthy and injured hamstrings. Flexors (Fl)/quadriceps (Q) ratios (mean ± SD) calculated in the concentric mode at 60 °/sec and 240 °/sec or mixed Fl_E/Q_C. Characteristic values of a normal control group (mean ± SD) are represented by shaded areas (reprinted from Croisier and Crielaard,[7] with permission from IOS Press). **C** = concentric; **E** = eccentric; * $p < 0.01$.

attention to the risk of misinterpretation from a nonspecific isokinetic protocol. Provided that eccentric exercise is introduced cautiously, negative side effects, such as DOMS or new strain on the dynamometer,[11] would be unlikely. However, Orchard et al.[47] reported an isolated hamstring strain caused by isokinetic testing in a professional rugby player.

Croisier et al.[19] selected athletes with muscle strength deficits on the basis of statistically selected cut-offs of peak torque, bilateral differences and flexors/quadriceps ratio. These athletes followed a rehabilitative programme individually adapted from their strength profile (emphasising eccentric training at an elongated position of the muscle). This model resulted in isokinetic parameter normalisation in 17 of 18 athletes. Prospectively observed for 12 months after returning to athletics, none of the corrected athletes sustained a clinically diagnosed hamstring muscle re-injury. They all returned to their prior level of competition with significantly reduced subjective intensity of pain and discomfort. Criteria were severe, for instance, the goal in side-to-side comparison was to reach a deficit of less than 5%. This study confirmed that while multifactorial origin of muscular injuries were present in 31% of subjects, the persistent complaints and recurrent strains during sports participation were not correlated with significant bilateral asymmetries or agonist/antagonist imbalances. Alternatively, 69% of subjects were selected for this study because of an abnormal isokinetic profile, indicating the key role played by strength quality, particularly eccentric,[4,48] in the complex interaction between aetiological factors. In summary, any factor that impairs the contractile function of the muscle will lead to a reduction in its energy-absorbing capabilities, increasing the likelihood of re-injury.

On the basis of these observations, we strongly recommend that athletes recovering from injury should undergo specific isokinetic testing before resuming athletic activities to indicate the extent of a residual muscle performance dysfunction. Nevertheless, the determination of 'normative values' for agonist/antagonist ratios in isokinetic analysis must take into account the lack of compatibility between different devices.[49,50] The improper use of statistically defined limits of ratio simply extracted from the literature (e.g. fixing the inferior limit to 0.65, even though the proper value on the personally used dynamometer would be 0.55) could lead to erroneous interpretation and increase the risk of re-injury. Clinicians are, therefore, encouraged to make sure that the referential values they use come from identical machines and evaluative protocols.

1.2.2 Flexibility

For some authors, a lack of flexibility is not been conclusively linked to risk of hamstring injuries.[34] In fact, each specialist involved in sport management has their own opinion about the role of flexibility in injury and divergences may result from lack of clarity in definitions.

Gleim and McHugh[51] distinguish between static (end of the range of motion), dynamic-passive (stiffness/compliance) and dynamic-active (muscle contracted, stiffness/compliance) measurements. They consider dynamic assessment, with respect to the degree of muscle contraction, to be less dependent on patient discomfort and more objective. A study by McHugh et al.[52] provided experimental evidence of an association between flexibility and mus-

cle injury. Exercise involving predominantly eccentric actions frequently causes muscle damage, resulting in DOMS.[11,53,54] Passive muscle stiffness plays a key role in the occurrence of DOMS symptoms, possibly explained by tendon-aponeurosis mechanics:[52] the strain imposed by active lengthening of stiff muscles would be transferred from a rigid tendon-aponeurosis complex to the muscle fibres, resulting in myofibrillar strain. Conversely, the tendon-aponeurosis of compliant muscles would be able to partially absorb lengthening and limit myofibrillar strain.

A recent prospective study by Witvrouw et al.[55] indicated that soccer players with an increased tightness of the hamstring have a statistically higher risk ($p = 0.02$) of developing a lesion on that muscle group. Interestingly, players with a quadriceps muscle injury were also found to have significantly ($p = 0.047$) lower flexibility in this muscle before injury compared with the uninjured group, even though flexibility and injury occurrence were independent factors for adductor and calf muscles. Thus, for hamstring muscles, strength and elasticity should be at a normalised level (through bilateral comparison and referential values) when the athlete restarts a maximal training programme.

1.2.3 Age-Related Factors

Orchard[22] proposed age as a risk factor for hamstring muscle strains ($p < 0.001$) but not for quadriceps muscle strains pertaining to abnormalities of the lumbar spine. The sequential mechanism evoked for the increased age-related susceptibility is low lumbar degeneration leading to L5 and S1 nerve impingement, promoting muscle fibre denervation and ending in decreased hamstring muscle strength. The quadriceps are unaffected so this could create an agonist/antagonist imbalance. In that case, older athletes with a recurrent profile of injury could benefit from examinations defining spine and neurological status, as well as any agonist/antagonist torque ratio imbalances.

Orchard[22] found more hamstring and calf injuries in players with higher body mass index. However, this association may be a result of confounding as body mass index correlated highly with player age and previous injury.

1.2.4 Joint Dysfunction

One cause of low-grade hamstring symptoms might be subthreshold low back pain of a zygapophyseal origin.[34] Sustained pain input following motor and sensory neural irritation of the levels that serve the posterior thigh may provoke local muscular responses such as increased muscle tension and result in local damage. However, this phenomenon has not yet been scientifically shown to be associated with injury.

A study by Cibulka et al.[56] suggested that, by means of an anterior pelvic tilt, sacroiliac joint dysfunction may be associated with a weakness in the transversus abdominus,[34] therefore elongating the entire hamstring musculotendinous unit. Correcting sacroiliac joint dysfunction permitted a greater increase in hamstring peak torque in comparison with non-manipulated patients. These results must be analysed with great caution and further studies are required to determine whether sacroiliac joint dysfunction assessment can predict the (re)occurrence of hamstring strains.

1.2.5 Hormonal Status

It is also worth mentioning a case report by Naessens et al.[57] dedicated to recurrent muscle injury over several years in a high-level soccer player as a result of hypogonadism. In that study, laboratory findings showed abnormally low resting testosterone levels, most prominent during training periods, and an unfavourable testosterone/cortisol ratio during recuperation after exercise. An anti-oestrogenic drug (tamoxifen) normalised the hormonal status and spectacularly decreased the player's muscle injury rate. The authors assumed that a chronic anabolic/catabolic hormone imbalance could be of significant influence in sports based on eccentric and explosive work.

2. Modifications from Initial Strain

Structural modifications in the muscle can be the result of muscle strain, and some, if not treated, will promote persistent complaints and chronic injury.

The formation of haematoma and oedema must be reduced as well as the extravasation of the inflammatory cells to the injury site because of the further harmful consequences they may lead to. Starting the application of rest, ice, compression and

elevation (RICE) immediately after injury should be both essential combined therapy and regularly recommended.[44] However, Thorsson et al.[58] concluded that the application of a maximum compression bandage within 5 minutes of a muscle trauma did not significantly reduce the size of the haematoma or significantly shorten the time to complete subjective recovery compared with no immediate treatment.

Although the muscles most commonly concerned by ossification are the quadriceps and the brachialis, myositis ossificans can occur in any muscle.[59] Trauma is almost always the initiating cause and it is theorised that muscular calcification would result from ossification of a muscular haematoma.[60]

Early vigorous massage and excessive mobilisation[1] may give rise to heterotopic bone formation, inducing modification on the functional outcome of the muscle structure.

According to Arrington and Miller,[59] recovery of full unrestricted motion and return to normal activity are not dependent on the heterotopic bone exostosis. However, our practical background indicates that such a complication inevitably lessens the muscle flexibility, contributes to persistent complaints and worsens the functional prognosis promoting injury recurrence.

Two processes that both support and compete with each other are at work in the healing of skeletal muscle injury;[39,44] these processes must be balanced. One process results in muscle regeneration (to be maximised) including satellite cell migration into the injured area, forming new myoblasts, the other process results in production of connective tissue scar (to be minimised). Reduced tensile strength of the scar tissue at the site of previous disruption may contribute to the pathogenesis of recurrent muscle strain. After muscle laceration injury in animals, the scar weakness was proposed as the main factor responsible for early re-injury.[61] As scarring after muscle strain in humans is primarily composed of type III collagen during early phase,[62] unsuitable loading could then promote re-injury.

2.1 Immobilisation and Remobilisation of Injured Muscle

In the course of muscle healing, a short period of relative immobilisation, depending on the grade of injury, is needed for the wound tissue and collagen network to be established into which the satellite cells can then migrate.[63] The purpose is to promote the granulation tissue matrix formation[1] and that the scar can bear the pulling forces directed on it without re-rupture. Remobilising the muscle too quickly may disrupt the newly formed scaffold for the satellite cells.[63] Nevertheless, prolonged immobilisation is discouraged as it leads to excessive connective tissue[63] and detrimental long-term effects. Immobilised muscles have a lower load to failure and a lower total deformation to failure compared with non-immobilised muscle.[10] The only exception may be in complete rupture,[10] in which case immobilisation may allow some re-approximation of the torn muscle ends, with the muscle not to be used for between 10 and 14 days.

Mobilisation is then necessary to avoid muscle atrophy and to achieve the best results in the resorption of connective tissue scar and re-capillarisation of the damaged area.[1] Järvinen and Lehto[64] emphasise early mobilisation to promote the penetration and proper orientation of the regenerating muscle fibres through the connective tissue. These exercises will prevent or decrease adhesions within the connective tissue. Stretching has to distend the scar tissue when still plastic and prevent functionally disabling retraction.[1] Proprioception also recovers faster with early motion. Eccentric actions at a very low contraction intensity could be implemented, although the benefits of this have not yet been scientifically demonstrated. The aim is not classical strengthening, but, in our experience, it does favourably influence the scar characteristic and proprioceptive control of the motion.

Puranen and Orava[65] referred to the so-called 'hamstring syndrome' associated with the shortening and fibrosis near the ischial tuberosity origin. In some cases, surgery can eliminate the development of extensive scar tissue and facilitate the re-innervation of the myogenicallly denervated stumps.[66]

Worrell et al.[67] used a passive-knee extension test to demonstrate that hamstring-injured athletes were significantly ($p < 0.05$) less flexible on their hamstrings (for both sides) compared with a group of matched controls. Moreover, injured hamstring muscles showed less flexibility ($p < 0.05$) than the contralateral non-injured hamstrings which demon-

strates that muscle injury caused an additional loss of hamstring flexibility.[67,68] The feature of tighter hamstrings after injury has been confirmed elsewhere.[4] Inflammation in the short term and the muscle scar in the longer term may explain this distinctive profile.

Passive stretching of muscle at the end of the healing process is thought to be beneficial because it reduces muscle stiffness. Much of the decrease in stiffness is due to viscoelastic properties rather than to reflex changes.[36]

2.2 Reducing Stress

Diminishing stress within the muscle for a given length change could represent a plausible mechanism by which stretching might prevent further reinjury.[10] Acute and chronic changes in flexibility are likely to occur with stretching exercises, but it is difficult to distinguish between changes in subjective stretch tolerance as opposed to changes in muscle stiffness;[51] only this last change will protect against injury.

Psychological stress, resulting from the competitive situation or previous injury and persisting symptoms, could predispose the hamstring to injury[1] by increasing the tension on it. Harmful tension on the hamstring would decrease the ability of the muscle to absorb pull-forces[2] and lead to dyssynergic muscle contraction during high intensity activity.[1] However, it has not been proven scientifically to be associated with hamstring injury. Thus, interest in the occasional use of relaxation techniques, in addition to classical rehabilitation, remains hypothetical.

While the contribution of possible adaptive changes in the biomechanics and motor patterns of sporting movements following the original strain[30] have not been determined to date, this factor may also favour further injury.

2.3 Neural Tension

Some authors from the manual therapy field also refer to the presence of 'adverse neural tension' as a factor in repetitive hamstring strain.[69] Adverse neural tension can be described as abnormal physiological and mechanical responses produced from the nervous system structures when their normal range of movement and stretch capabilities are tested.[69] Branches of the sciatic nerve can become tethered to the scar following a hamstring injury, creating extraneural tension with or without local irritation. Peripheral sensitisation could occur following sustained input to the hamstrings and local damage would then result.[34] The slump test, used as an indicator of adverse neural tension, was positive in more than 50% of subjects with a non-repetitive grade I hamstring strain.[69] In our opinion, the relationship (cause and effect) between muscle strain and adverse neural tension remains speculative at this point. The exact relevance of incorporating special mobility techniques including 'neural tension positions' in the rehabilitative design has not yet been scientifically established.

3. Questionable Options in Treatment

3.1 Diagnosis

The initial correct diagnosis of the injury governs the application of the appropriate treatment and avoidance of prolonged hamstring pain syndromes.[1] Complementary to clinical history and examination, imaging techniques[1-3,43,70] such as x-ray, ultrasonography, CT scanning or magnetic resonance imaging (MRI), must be utilised in differential diagnostics. Occasionally, imaging studies show a more extensive injury than that initially and clinically anticipated.[30] Diagnosis of avulsion in the ischial tuberosity requires longer immobilisation, and complete hamstring muscle origin rupture necessitates operative treatment.[1,71] To avoid long-term disability, Clanton and Coupe[44] recommend that complete hamstring avulsions from the ischial tuberosity (including bone avulsion with 2cm or more of displacement) should be repaired.

As the use of MRI is expense and the equipment is not readily available, ultrasonography is usually the method of choice[66,70] for localising and differentiating haematoma and oedema. Extensive bleedings is occasionally evacuated by puncture or surgery[70] because large intramuscular haematomas can adversely influence scar formation and lead to the development of an ossification.[59]

Croisier et al.[19] reported long-term persistent complaints and rehabilitation failure in one sprinter

for whom complementary electromyogram (EMG) and MRI measurements revealed an innervation defect due to nerve compression because of ectopic calcification. Within 3 weeks of the injury, the osteoblastic activity would be identified by nuclear medicine scanning and MRI.[59]

Surgical management is not recommended in the early stage of injury, with treatment consisting initially in rest followed by active mobilisation and range of motion recovery.[1] Surgical options and excision are only indicated in mature symptomatic heterotopic bone.[59]

3.2 NSAIDs

The use of NSAIDs in the treatment of muscle injuries is a matter of controversy[1,72,73] regarding the appropriate timing and duration of administration. The normal healing process, starting with an inflammatory reaction including phagocytosis of damaged tissue and capillary ingrowth, could be blunted and the repair response delayed if anti-inflammatory medication is used.[63] However, others contend that anti-inflammatories may be used to keep inflammatory process under control and provide analgesia. Hence, NSAIDs should be started immediately after injury in order to control pain, avoid reflex inhibition and improve active muscle contraction.[66,74]

According to Almekinders,[75] NSAIDs have no adverse effects on the tensile or contractile properties of the injured muscles. Obremsky et al.[74] demonstrated that piroxicam does not adversely influence the recovery of strength in a follow-up period of 1 week, but does lead to a delay in the histological repair process. The animal model by Mishra et al.[76] showed that the effects of flurbiprofen are time dependent; the contractile properties and histological data suggest that NSAIDs cause a short-term gain, but have a middle-term detrimental effect leading to functional loss (deficit in force generation at 28 days). These findings could prompt the rethinking of the widespread prescription of NSAIDs. The inappropriate and prolonged use of anti-inflammatories is not generally considered a factor predisposing an athlete to re-injury but the potential harmful adverse effects must be considered.[2] Thus, Noonan and Garrett[10] suggest the use of NSAIDs for pain relief only in the first 2 to 3 days after injury, though some clinicians, inspired by their personal experiences, prolong therapy of 7 to 10 days.[77]

3.3 Glucocorticoids

The prescription of glucocorticoids is clearly contraindicated because they lead to a delayed elimination of haematoma and necrotic tissue, as well as the retardation of muscle regeneration.[66]

3.4 Local Anaesthetics

The use of local anaesthetics is poorly documented in the literature. In the 2002 article by Orchard,[78] he explains that the non-publication of research on their use was influenced by both the possibility of complications arising from their use and the ethical questions associated with use. Nevertheless, techniques such as local anaesthetic injection at an injury site, sometimes with corticosteroids, are increasingly being used to attempt to rapidly return the athlete to competition.[79] Such techniques for local pain relief while the muscle is still healing and remodelling represent a primary factor in further injury. The pain of the injury may be a protective mechanism to prevent tensile forces above the threshold for strain or complete rupture. The lack of inhibition from pain could result in excessive mechanical demand on the muscle. Despite the risk of worsening the initial insult, which is clearly explained to players, the procedure seems attractive because the risks are often perceived by the athlete to be less than the potential benefits.[78] The motivation for players to return to competition early is probably higher in professional teams who are competitively placed, particularly towards the end of the season.

4. Consequences of a Premature Return to Competition

The possible role of medical staff in the premature return of athletes to competition is worrisome. A study into medical staff associated with English professional football teams by Waddington et al.[80] has suggested that the methods of appointing club doctors and physiotherapists and their level of qualification is in need of careful re-examination.

Rehabilitation is often prolonged and a proper rehabilitation programme reduces the number of re-injuries.[1,19,32,44] The impatience of the athlete, reinforced by frequent pressure from the team and trainer, frequently provokes an early return to training and competition in spite of lingering symptoms.

That an early return to activity entails the risk of further major injury is a logical assumption;[37] this has been further emphasised by Taylor et al.[79] based on experimental data. In an animal experimental model, Kaariainen et al.[61] suggested that scar weakness is a limiting factor until 10 days post-injury and, thereafter, muscle atrophy is a more decisive factor. Wisely, Orchard and Best[30] underline that the healing process is probably much slower than clinical findings would indicate.

Alteration in the mechanical characteristics of the muscle resulting from a prior minor injury might precipitate a more major injury. This type of situation is frequently experienced in the arena of European professional soccer. Anecdotally, we report the case of an international level player presenting a significant hamstring strength deficit with agonist/antagonist imbalance (demonstrated by isokinetic assessment) after 5 weeks of rehabilitation. Despite our recommendations, he returned to official competition and re-injured the same muscle only 2 weeks later, missing the subsequent 8 weeks of matches.

One important task for medical staff associated with sports teams is to advise the athlete and coach on the prognosis, time and type of rehabilitation needed before a return to competition is possible.[81] The consequence of incomplete rehabilitation, particularly the neglecting of stretching and strengthening techniques adapted to the post-injury phase, has been outlined in sections 1.2.1 and 1.2.2. The injured athlete should not participate in normal sports practices when they are still experiencing feelings of tightness, weakness or pain – returning to sports activity before the injury has properly healed clearly represents a lack of understanding of the injury mechanism. As a result of pain, neural inhibition will develop, preventing maximal motor recruitment. This phenomenon, which could enlarge pre-existing muscle weaknesses, justifies not only progressive pain-free strengthening of the strained muscle but also a real proprioceptive rehabilitation.

On the other hand, the use of overly aggressive rehabilitation designed to prematurely return an athlete to competition may be too stressful for the muscle, risking further injury[2] (table I). Connective tissue remodelling may involve transient periods of mechanical weakness and structures could be damaged if insufficient time is allowed for adaptation.[82] Exercise in the presence of DOMS will limit exercise intensity and duration, but will, above all, promote the occurrence of a real anatomical strain, as mentioned by Cheung et al.[83] Consequently, and for instance, the optimal prescription of eccentric training at that moment would not exceed three times a week in athletes unaccustomed to this particular mode of contraction.[11] Physiotherapists should be knowledgeable of the sequelae that are associated with muscle damage after high-force eccentric exercise and take the appropriate precautions.[83-85] DOMS will behave like fatigue and represents an unsuitable situation giving rise to re-injury. Submaximal intensity, low repetition, low volume eccentric exercise can provide a further protective effect against DOMS.[11] We propose a model imposing contractions up to 50% of the maximal intensity in the early phase of training and rehabilitative programmes, and that this be increased progressively, reaching maximal performance around the fifth session.[11]

5. Proposal for a Management Model

Based on our current understanding of mechanisms and a review of the literature, we propose a plan for the rational management of a grade 2 muscle injury in order to prevent recurrence (see table II). The list of methods and exercises is obviously not exhaustive. Treatment chronology will be adapted according to the gravity, individual features and symptoms of the strain.

We re-emphasise the necessity of a multidisciplinary approach to muscle injury. It is legitimate to support specific and scientific education for all those supervising sport activity (medical doctor, physical therapist, trainer).

6. Conclusion

Hamstring strains are a frequent complaint for athletes on return to athletic activities and these

Table II. Proposed guidelines for the management of a grade 2 muscle injury in order to prevent recurrence

Acute phase (to 48–72 hours)
First aid with rest, ice, compression, elevation (RICE)
Proper imaging techniques, such as ultrasonography on J0 or J1 and occasionally MRI in differential diagnosis (sometimes at a later date)
NSAIDs for 2–3 days
Ambulatory aids avoiding tissue irritation and subsequent inflammation
Patient performs active knee extension and flexion exercises (pain-free progression)
Subacute phase: (day 2–4 to day 8) [decrease of inflammation signs]
Electrical stimulations facilitating pain control and preventing amyotrophy
Submaximal (symptom-free) isometric contractions in a short and comfortable muscle length position
Passive mobilisation of the knee and hip joints allowing cautious and pain-free stretching
Swimming pool activities with gait pattern correction
Remodelling phase (weeks 2–4)
Progressive jogging in swimming pool conditions
Stationary cycling maintaining cardiovascular fitness
Low-intensity and low-speed eccentric contractions on isokinetic device (avoiding the maximal muscle length) promoting healing of good quality and proprioceptive control
Isometric contractions at multiple angles, progressively maximal
Progressively intensified dynamic concentric and eccentric exercises in a prone position, close from the length tension relationship occurring during activities
Progressive intensification of stretching modalities
Proprioceptive exercises promoting hamstring control activity
Deep-tissue massage fighting against a painful fibrotic area
Recovering phase (week 4 to weeks 6–8)
Coordination exercises
More aggressive flexibility training with static exercises in an anterior pelvic tilt, static-stretch method and ballistic exercises
Progressive jogging without sprint and speed exercise
Maximal strengthening, most notably in eccentric mode in elongated muscle position
Isokinetic assessment verifying especially absence of bilateral asymmetries in eccentric, normalisation of a mixed FI_E/Q_C ratio and angle to peak torque
Correction of persistent strength and flexibility abnormalities before return to competition
Complementary mobilisation or manipulation of sacroiliac joint if needed
Transition phase (week 6 to return to competition [in partnership with physical trainer])
Individualised exercises on the field, incorporating speed exercises and specific gestures
Normal training within the group of players
Education about active (rather than passive) warm-up, stretching modalities, running style, nutrition, hydration
Encouragement to maintain an in-season hamstring strengthening programme
In a few cases, a relaxation technique may complement classical rehabilitation and reduce stress impact
Discussion (when accepted) with coach and physical trainer about main risk factors (fatigue, overtraining, etc.)

FI_E = eccentric flexors; **MRI** = magnetic resonance imaging; **Q_C** = concentric quadriceps.

injuries are characterised by a high tendency to become recurrent. Sport science and medicine need to provide therapists and trainers with rational guidelines for the prevention of muscle re-injury and education on the factors contributing to injury mechanisms.

Hamstring re-injuries appear to have a multifactorial aetiology, these factors include those inherent in sport activity, intrinsic factors, factors linked to systemic modifications from initial strain and those following questionable options in treatment. One current difficulty is the lack of valid objective measures to identify the factor or combination of factors responsible for strain recurrence. Among the most likely factors are:

- mode of training, in particular warm-up and fatigue management;
- muscle strength abnormalities, most notably in the eccentric mode and the key role played by agonist/antagonist imbalances;
- increased hamstring tightness;
- features of the scar tissue and the definition of optimal rehabilitation in the course of the muscle healing;
- approximate diagnosis and treatment options, which lead to chronic muscle weakness, tightness and long-lasting exercise-induced pain.

In sports medicine, rehabilitation includes procedures designed to restore athletes to their prior level of function within the shortest period of time possible. Nevertheless, it remains difficult to decide when an athlete can safely return to full sporting activities following a hamstring strain and the risk in to allowing the athlete to resume practice before real normalisation of all the implicated components.

A management model of hamstring strain has been proposed in order to facilitate not only the return of the athlete to full fitness but also to prevent recurrence of the injury (see table II). The prevention of re-injury is not only the priority of the athletes themselves but also the health professionals and trainers that the athletes train with.

Heavy emphasis also needs to be placed on the knowledge of therapists and trainers involved in the rehabilitation process, this includes a combination of disciplines: the medical doctor supplying the diagnosis and forecasting the athlete's period of inactivity; the physiotherapist choosing the rehabilitation techniques and speed of rehabilitation; the trainer in designing the training programmes; and club management in the safety culture it promotes and pressure exerted on players, as well as therapists, to get the athlete back to full competition as quickly as possible.

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