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Guest Editorial

The Management of Muscle Strain Injuries: An Early Return Versus the Risk of Recurrence

For the sport medicine clinician, muscle strains can be one of the most frustrating injuries to treat. Despite apparently thorough management plans, objective testing, and clinical evaluation before return to play, these injuries often recur, with the impression that the clinician has failed by allowing the athlete to return to sport too early. The inexperienced or lay person will offer a simple solution: that these recurrences could have been prevented by allowing athletes a little extra time for healing before return to play. Analysis of recurrence data from the Australian Football League (AFL) injury surveillance system (Table 1)[1,2] shows that the situation may not be all that simple. While a fair proportion of muscle strains recurred during the first week after return, there was a persistent significantly increased risk of recurrence for *many weeks* after return to play. This pattern contrasts with a muscle contusion injury, for which after a single week of increased risk for recurrence, there is a quick return-to-baseline level of risk (Table 1). The cumulative risk of recurrence for a hamstring strain for the remainder of the season was 30.6%. By comparison, in the AFL over the same period, the cumulative recurrence risk for thigh contusion was 12%, for concussion was 5%, for knee medial collateral ligament strain was 11%, and for ankle sprain was 15%.[2] Although the pessimist will cite that an unacceptable 12.6% of hamstring strains recur during the first week after return to play, the optimist, who is often the coach, will be reassured that 87.4% of players successfully complete their next game. The realist will understand that even the majority who make it through that first game are still at high risk in the ensuing weeks. Therefore, although most athletes do not suffer recurrences of a muscle strain injury, those who are successful must negotiate a lengthy period of increased susceptibility. These results for AFL football have not been reproduced for other sports to the best of our knowledge, but clinical experience suggests similar trends.

This lengthy period of increased susceptibility for recurrent injury is not surprising when the pathophysiology of muscle stretch injury and repair is examined in laboratory studies.[3,4] Despite our clinical impressions that an average muscle strain will resolve with an appropriate rehabilitation program in 2–3 weeks, recent evidence shows that there is still ongoing muscle regeneration in the presence of mature scar tissue formation.[3] The early postinjury period is marked by upregulation of Type III collagen mRNA expression with relatively little myosin mRNA expression.[3] This observation extends to the protein level where Type III collagen is present prior to any histological evidence of myofiber regeneration. Clinicians and therapists face an ongoing dilemma of requiring some new collagen formation for the muscle–tendon unit to carry load and generate torque about a joint while at the same time seeking minimal scar formation in order to minimize stiffness. Basic science studies are needed to delineate factors that regulate and control new collagen synthesis.

Imaging studies of athletes diagnosed with muscle strain injury have shown that the injury can be much more extensive than initially appreciated by clinical examination.[5–7] These findings, together with unpublished data from one of the authors (T.M.B.) that repeat magnetic resonance imaging of athletes who have been cleared for return to sport often show persistent high signal changes and muscle edema on T2 images, should prompt us to consider the clinical strategies used to determine recovery from injury. Fluid collections may be a cause of swelling and weakness in the absence of a fascial tear that may predispose to recurrent injury.[5] There is no consensus as to when an athlete can safely return following a muscle strain. No single test or clinical observation is regarded as the gold standard. Perhaps the most popular and safe approach has been that an individual can return once full range of motion, strength, and functional activities can be performed, yet there is limited scientific data to support this strategy.[8] The situation is somewhat analogous to return following anterior cruciate ligament (ACL) reconstruction. Many practitioners will allow a player to return approximately 6 months after an ACL reconstruction, as long as clinical examination and functional assessment are normal. Basic science studies, however, suggest that the ACL graft is still maturing between 12 and 24 months after reconstruction.[9,10] When the pathophysiology of muscle stretch injury and repair is taken into account, the difficult question becomes: How do the majority of muscle strains *not* recur, given that the athlete will generally return to play well before healing is complete?

Although a muscle strain can be created in the laboratory, the forces that produce this injury in vivo are not well understood, hence it is equally difficult to explain the forces involved in the recurrence of a muscle strain. Factors that may contribute to the pathogenesis of recurrent muscle strain include:

1. Reduced tensile strength of the scar tissue at the site of previous disruption
2. Reduced strength of the muscle at other sites due to disuse atrophy and/or pain limitation and/or reflex inhibition

TABLE 1. *Chance of recurrence after return from injury (1992–1998 Australian Football League)*[18]

Weeks after return from initial injury	Weekly percentage risk of injury recurrence (%)			
	Hamstring strain (n = 858)	Quadriceps strain (n = 251)	Calf strain (n = 217)	Thigh contusion (n = 123)
1	12.6[a]	9.0[a]	7.8[a]	5.6[a]
2	8.1[a]	4.7[a]	5.7[a]	1.2
3	6.8[a]	3.3[a]	3.3[a]	1.3
4–5	4.7[a]	3.7[a]	0.0[b]	0.0[b]
6–8	3.1[a]	3.3[a]	2.8	1.3
9–14	2.7[a]	0.5	1.1	1.6
15–22	1.4	2.2	2.1	0.0[b]
Cumulative risk of recurrence for remainder of season (%)	30.6	22.9	23.8	12.2

[a] Significantly greater than weekly risk of reinjury during following season ($p < 0.05$).
[b] No recurrence reported during this time period.

3. Reduced flexibility of the muscle-tendon unit secondary to inhibition and/or scar formation
4. Possible adaptive changes in the biomechanics and motor patterns of sporting movements following the original injury.

The relative contribution of these four factors has been difficult to ascertain in studies to date, making some aspects of clinical management controversial. An animal study of muscle laceration injury that induced reinjury at various times following the initial injury has suggested that scar weakness is the limiting factor until 10 days postinjury, and thereafter muscle atrophy is a more important factor.[11] It is likely that a similar situation is true following muscle stretch injury, given that early scar formation is composed predominantly of Type III collagen.[3] Although there are no published data that report location of tear with recurrent injury at other sites, we have seen several cases of where the new tear is in a different location from the original site of injury (T. M. Best, unpublished results). Whether there is true muscle atrophy or relative changes induced by the scar that predispose to this pattern of injury is not known.

It has been difficult to determine from epidemiological studies the role of factors such as strength and flexibility, both in predicting injury and recovery from injury. Reduced strength has been found to be a risk factor for primary muscle strain injury by some authors[8,12–14] but not others.[15,16] Although stretching can alter the short-term viscoelastic properties of the muscle–tendon unit,[17,18] the extrapolation of these findings from laboratory studies to humans has not provided conclusive evidence that stretching can prevent or even reduce the risk of injury.[19,20]

Previous injury is the one risk factor for recurrent muscle injury for which there is universal agreement.[15,21,22] A recent study has confirmed the common clinical impression that a recent history of strain of one muscle group confers an increased risk of injury to surrounding muscle groups.[21] For example, on return to play from a hamstring strain, a player is not only more likely to reinjure the hamstring, but is also more likely to strain a quadriceps muscle.[21] This finding suggests that biomechanical and perhaps neurological alteration in muscle and joint function subsequent to injury are important to appreciate in the rehabilitation phase.

Perhaps the greatest advances in appreciating the risk for recurrence of muscle strain injury will arrive with a greater understanding of sprinting biomechanics. After a muscle strain occurs, some athletes may make subtle alterations to their normal running mechanics. Are these alterations protective or detrimental? Perhaps they may be protective specifically to the injured (weakened) muscle but detrimental to other nearby muscles. If the adaptive changes are generally protective, are they counterproductive with respect to performance (e.g., maximal velocity)? Are athletes who are unable to make adaptive changes and/or the ones under most pressure to return to maximal (normal) sprinting performance the ones who are most likely to suffer injury recurrences?

Following a muscle strain injury, nonsteroidal antiinflammatory drugs (NSAIDs) and cortisone injections induce changes in the healing response that have yielded conflicting effects.[23–27] Not too surprisingly, therefore, use of antiinflammatory agents is currently a decision based on clinical experience rather than basic and clinical science. A recent review highlights the uncertainty about the role of NSAIDs for the treatment of soft tissue injuries.[28] Their administration may delay the healing response and slow the rate of recovery of tensile strength of the muscle–tendon unit.[23,25,26] It might also be argued that by limiting pain and inflammation, other changes predisposing to injury at new sites such as inflammation-induced atrophy and/or pain inhibition are reduced. Early return-to-high-loading-activities is a more routinely accepted part of management programs, but a similar dilemma applies. Early loading will help to minimize secondary atrophy, but can itself increase the risk of reinjury while the scar is still weak and composed primarily of Type III collagen.

Timing of return to play following muscle strain injury is often a most challenging management decision for the clinician and the therapist. The most important information that the clinician must convey to the athlete and the coach is that return to play following a muscle strain

does not usually coincide with full recovery and healing from the injury. The concept of a player "carrying" an injury is an accurate one, and it helps if the player and the coach can appreciate this. It is understandable that muscle strains have a high rate of recurrence clinically, because basic science studies show that healing is much slower than our clinical findings would indicate. There appears to be a trade-off between quick return to play, rate of recurrence, and perhaps athletic performance. While we do not fully understand why muscle strains recur, and even in the future when we do, the issue of return to play will most likely be one of risk management, not risk elimination.

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REFERENCES

1. Orchard J. Recurrent hamstring injury in Australian Football [Abstract]. *Med Sci Sports Exer* 1998;30:S52.
2. Orchard J. *Epidemiology of Lower Limb Injuries in the Australian Football League* [Ph.D. Thesis]. Sydney: University of New South Wales, 1999.
3. Best T, Shehadeh S, Leverson G, et al. Analysis of changes in mRNA levels of myoblast- and fibroblast-derived gene products in healing skeletal muscle using quantitative reverse transcription-polymerase chain reaction. *J Orthop Res* 2001;19:565–572.
4. Nikolaou PK, MacDonald BL, Glisson RR, et al. Biomechanical and histological evaluation of muscle after controlled strain injury. *Am J Sports Med* 1987;15:9–14.
5. DeSmet A, Best T. MR imaging of the distribution and location of acute hamstring injuries in athletes. *Am J Roent* 2000;17:393–399.
6. Garrett WE Jr, Rich FR, Nikolaou PK, et al. Computed tomography of hamstring muscle strains. *Med Sci Sports Exer* 1989;21: 506–514.
7. Speer K, Lohnes J, Garrett W. Radiographic imaging of muscle strain injury. *Am J Sports Med* 1993;21:89–96.
8. Heiser TM, Weber J, Sullivan G, et al. Prophylaxis and management of hamstring muscle injuries in intercollegiate football players. *Am J Sports Med* 1984;12:368–370.
9. Ng G, Oakes B, Deacon O, et al. Long-term study of the biochemistry and biomechanics of anterior cruciate ligament-patellar tendon autografts in goats. *J Orthop Res* 1997;14:851–856.
10. Ng G, Oakes B, Deacon O, et al. Biomechanics of patellar tendon autograft for reconstruction of the anterior cruciate ligament in the goat: three-year study. *J Orthop Res* 1995;13:602–608.
11. Kaariainen M, Kaariainen J, Jarvinen T, et al. Correlation between biochemical and structural changes during the regeneration of skeletal muscle after laceration injury. *J Orthop Res* 1998;16:197–206.
12. Burkett LN. Causative factors in hamstring strains. *Med Sci Sports Exer* 1970;2:39–42.
13. Orchard J, Marsden J, Lord S, et al. Preseason hamstring muscle weakness associated with hamstring muscle injury in Australian footballers. *Am J Sports Med* 1997;25:81–85.
14. Yamamoto T. Relationship between hamstring strains and leg muscle strength. A follow-up study of collegiate track and field athletes. *J Sports Med Phys Fitness* 1993;33:194–199.
15. Bennell K, Wajswelner H, Lew P, et al. Isokinetic strength testing does not predict hamstring injury in Australian Rules footballers. *Br J Sports Med* 1998;32:309–314.
16. Worrell TW. Factors associated with hamstring injuries. *Sports Med* 1994;17:338–345.
17. Best T, McElhaney J, Garrett WJ, et al. Characterization of the passive responses of live skeletal muscle using the quasi-linear theory of viscoelasticity. *J Biomech* 1994;27:413–419.
18. Taylor D, Dalton J, Seaber A, et al. Viscoelastic properties of muscle-tendon units. The biomechanical effects of stretching. *Am J Sports Med* 1990;18:300–309.
19. Pope R, Herbert R, Kirwan J, et al. A randomized trial of preexercise stretching for prevention of lower-limb injury. *Med Sci Sports Exerc* 2000;32:271–277.
20. Shrier I. Stretching before exercise does not reduce the risk of local muscle injury: a critical review of the clinical and basic science literature. *Clin J Sport Med* 1999;9:221–227.
21. Orchard J. Intrinsic and extrinsic risk factors for muscle strains in Australian football. *Am J Sports Med* 2001;29:300–303.
22. Upton PAH, Noakes TD, Juritz JM. Thermal pants may reduce the risk of recurrent hamstring injuries in rugby players. *Br J Sports Med* 1996;30:57–60.
23. Almekinders LC, Gilbert JA. Healing of experimental muscle strains and the effects of nonsteroidal antiinflammatory medication. *Am J Sports Med* 1986;14:303–308.
24. Levine W, Bergfeld J, Tessendorf W, et al. Intramuscular corticosteroid injection for hamstring injuries. *Am J Sports Med* 2000;28: 297–300.
25. Misra D, Friden J, Schmitz M, et al. Anti-inflammatory medication after muscle injury. *J Bone Joint Surg* 1995;77:1510–1519.
26. Obremsky W, Seaber A, Ribbeck B, et al. Biomechanical and histologic assessment of a controlled muscle strain injury treated with piroxicam. *Am J Sports Med* 1994;22:558–561.
27. Thorsson O, Rantanen J, Hurme T, et al. Effects of Nonsteroidal Anti-Inflammatory medications on satellite cell proliferation during muscle regeneration. *Am J Sports Med* 1998;26:172–176.
28. Buckwalter J. Current concepts review pharmacological treatment for soft-tissue injuries. *J Bone Joint Surg* 1995;77A:1902–1914.