Hamstring Strains in Athletes: Diagnosis and Treatment

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Abstract

Hamstring strains are among the most common injuries (and reinjuries) in athletes. Studies combining electromyography with gait analysis have elucidated the timing of activity of the three muscles of the hamstring group; they function during the early-stance phase for knee support, during the late-stance phase for propulsion, and during midswing to control the momentum of the leg. Muscle injury, whether partial or complete, occurs at the myotendinous junction, where force is concentrated. The healing response begins with inflammation, associated edema, and localized hemorrhage. After an initial period of reduced tension, the healing muscle regains strength rapidly as long as reinjury does not occur. Although the use of anti-inflammatory medication is a keystone of treatment, a certain degree of inflammation is necessary for removing necrotic muscle fibers and rescaffolding to allow optimal recovery. The protocol of rest, ice, compression, and elevation is still the preferred first-aid approach. After a brief period of immobilization (usually less than 1 week for even the most severe strain), mobilization is begun to properly align the regenerating muscle fibers and limit the extent of connective tissue fibrosis. Concurrent pain-free stretching and strengthening exercises (beginning with isometrics and progressing to isotonics and isokinetics) are essential to regain flexibility and prevent further injury and inflammation. Readiness for return to competition can be assessed by isokinetic testing to confirm that muscle-strength imbalances have been corrected, the hamstring-quadriceps ratio is 50% to 60%, and the strength of the injured leg has been restored to within 10% of that of the unaffected leg. The only indication for surgery is a complete rupture at or near the origin from the ischial tuberosity or distally at its insertion (either soft-tissue avulsion with a large defect or bone avulsion with displacement by 2 cm).

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Citius—Altius—Fortius ("Faster—Higher—Stronger") is the Olympic motto. The intense training and phenomenal accomplishments of Olympic-caliber athletes pursuing the goals reflected in this motto remind us of the awe-inspiring nature of the human body. Yet these intensive efforts often generate injuries—some acute and others chronic, some annoying and others career ending. Few injuries are as dramatic as the acute hamstring strain of a sprinter who pulls up lame or falls to the track.

The hamstring strain is a well-recognized entity that results from excessive stretching of the musculotendinous unit. Applying the knowledge gained from basic science research and clinical experience can allow the athlete to recover from the injury, rehabilitate the extremity, and resume competition as quickly as possible while reducing the chance of reinjury. This same knowledge and experience can be incorporated into an effective preventive program. Most hamstring strains respond to conservative treatment with rest, ice, gentle stretching, and gradual return to activities, but reinjury is common if not treated properly. The injury occurs at the musculotendinous junction in the vast majority of cases. Because of the difficulty of reestablishing structural integrity with the use of current surgical techniques, surgery is rarely indicated. It is only in cases of proximal or distal avulsion of a hamstring tendon that serious consideration should be given to surgical treatment.

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Basic Science

Microscopic Anatomy and Physiology

The musculotendinous unit consists of muscle and its surrounding connective tissue along with the tendon and junctional attachments to bone. Skeletal muscle is composed of aggregates of muscle bundles containing multiple muscle fibers[1] (Fig. 1). Each muscle fiber, or myofiber, is a cell containing protoplasm, mitochondria, nuclei, glycogen, adenosine triphosphate, lysosomes, sarcoplasmic reticulum, and the distinguishing contractile element called the myofibril. Repeating units of actin and myosin protein filaments within the myofibril form the sarcomere that runs from one Z line to the next within the myofibril.[2] Each myofiber is surrounded by a plasma membrane (sarcolemma) and is enclosed by a basement membrane that merges with extracellular matrix and collagen fibrils to collectively form the endomysium. The endomysium is contiguous with the perimysium, which surrounds the muscle bundles, and the epimysium, which ensheathes the muscle as a whole.[3]

The contractile nature of the motor unit can be classified on the basis of whether it is composed of fast-twitch or slow-twitch fibers. This is determined by the length of time for the motor unit to reach peak tension.[4] From a biochemical standpoint, the twitch property of a muscle is related to the rate at which myosin splits adenosine triphosphate, the myofibrillar adenosine triphosphatase reaction.[4] Type I (slow-twitch) fibers utilize aerobic metabolism, which, at the cellular level, means greater numbers of mitochondria and increased storage of triglyceride. Type II (fast-twitch) fibers are generally better for anaerobic metabolism and provide greater muscle contraction.[4] Type II fibers are also more easily fatigued. They can be subdivided into type IIA and type IIB on the basis of their different properties[2,4] (Table 1).

Muscle fiber composition is one of the intrinsic factors that helps determine an individual's adaptability for certain sports activities. For example, elite sprinters have a high proportion of type II fibers, and endurance athletes have a preponderance of type I fibers.[2] Garrett et al[4] showed that hamstring muscles contain a relatively high proportion of type II fibers, and a higher percentage of type II fibers than the quadriceps. Little difference has been found between muscle fiber types in each specific hamstring muscle.[4] The high percentage of fast-twitch fibers in the hamstring muscles indicates the strength and speed that are requisite for the function of this muscle group in locomotor activities.[4]

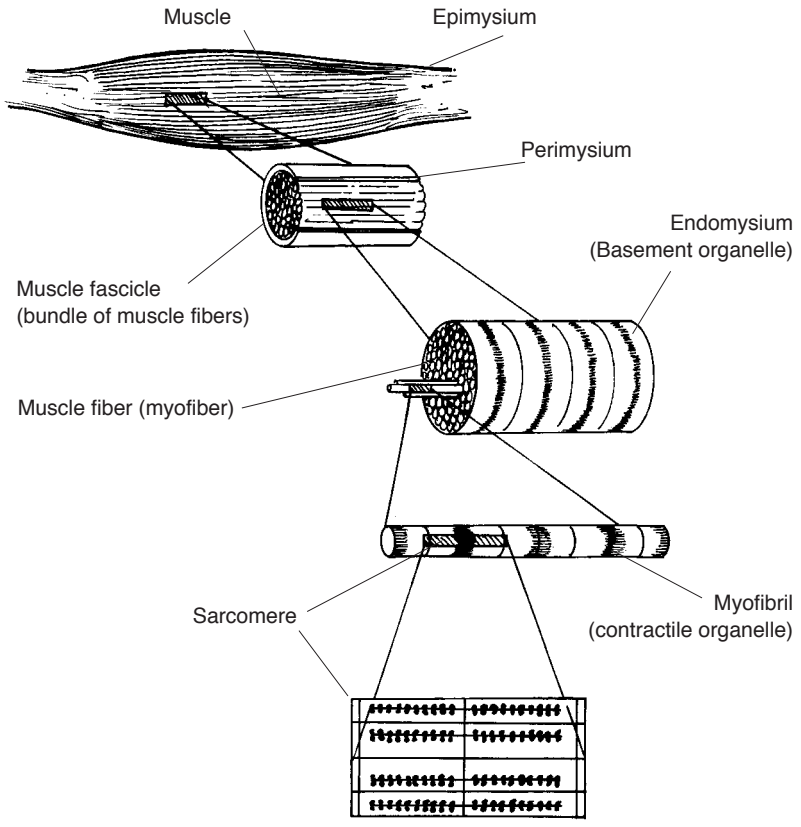

Fig. 1 The structure of skeletal muscle and the sarcomere.

Gross Anatomy

The hamstring muscle group consists of three muscles: the biceps femoris (long and short heads), the semitendinosus, and the semimembranosus. All three muscles, except for the short head of the biceps femoris, originate as an incompletely separated tendinous mass from the ischial tuberosity of the pelvis, a common point of avulsion fractures[5] (Fig. 2). The separate muscles become distinguishable 5 to 10 cm from the tuberosity, with the semimembranosus splitting off first.[6] The mus-

Table 1
Characteristics of Human Skeletal Muscle Fiber Types

Characteristic	Type I	Type IIA	Type IIB
Other names	Red, slow-twitch	White, fast-twitch	. . .
Metabolic description	Slow oxidative	Fast oxidative glycolytic	Fast glycolytic
Speed of contraction	Slow	Fast	Fast
Strength of contraction	Low	High	High
Fatigability	Fatigue-resistant	Fatigable	Most fatigable
Aerobic capacity	High	Medium	Low
Anaerobic capacity	Low	Medium	High
Motor unit size	Small	Larger	Largest
Capillary density	High	High	Low

cle fibers of the biceps femoris are visible 6 cm distal to the tuberosity, and the proximal myotendinous junction encompasses approximately 60% of the total length of the muscle.[6] The semimembranosus muscle fibers appear within the proximal 30% of this muscle, but the semitendinosus has a different arrangement, with muscle fibers coming directly off the tuberosity and the proximal musculotendinous junction.[6]

The short head of the biceps femoris group originates just medial to the linea aspera in the distal femur and is the only component of the hamstring muscle group with dual innervation. The short head of the biceps femoris is innervated by the peroneal portion of the sciatic nerve. The semimembranosus, the semitendinosus, and the long head of the biceps femoris are all innervated by the tibial branch of the sciatic nerve.

The long head of the biceps femoris attaches through a complex tendinous and fascial insertion to the fibular head and the lateral condyle of the tibia[7] (Fig. 3). The short head of the biceps femoris descends from its origin at an angle to the femur and has attachments into the tendon of the long head of the biceps femoris as well as fascial and tendinous insertions to the posterolateral capsule, the capsuloosseous layer of the iliotibial tract, the fibular head, and the proximal lateral tibia.[7] The semimembranosus and semitendinosus muscles course along the medial side of the femur to their separate attachment sites. The semimembranosus has multiple insertions at the postero-

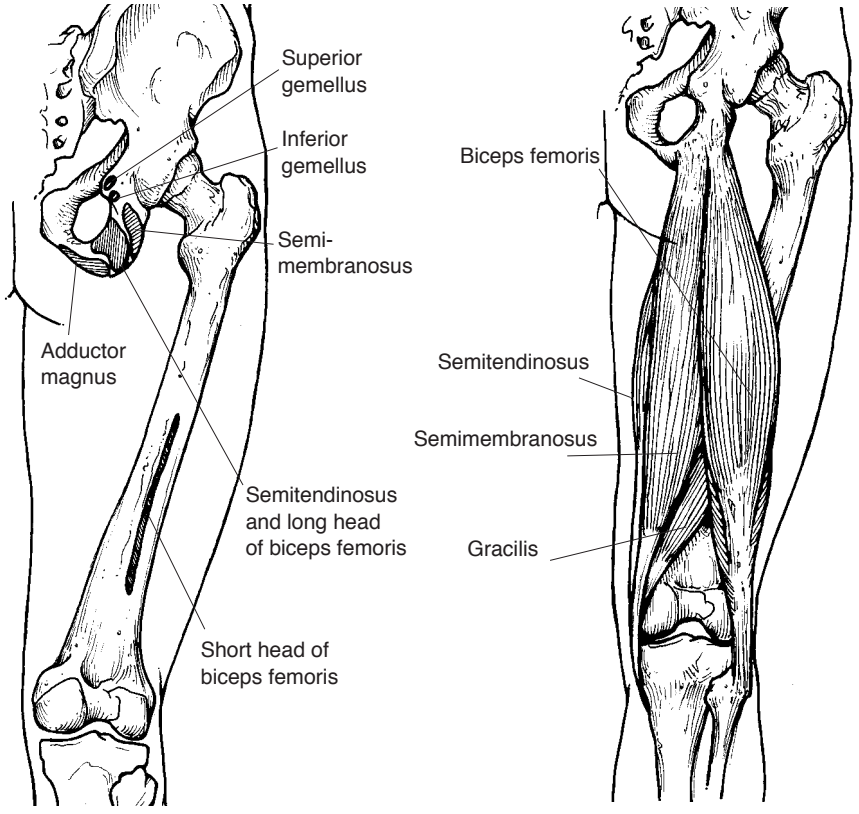

Fig. 2 Origins of the hamstring tendons **(left)** and muscles of the hamstring group **(right)**.

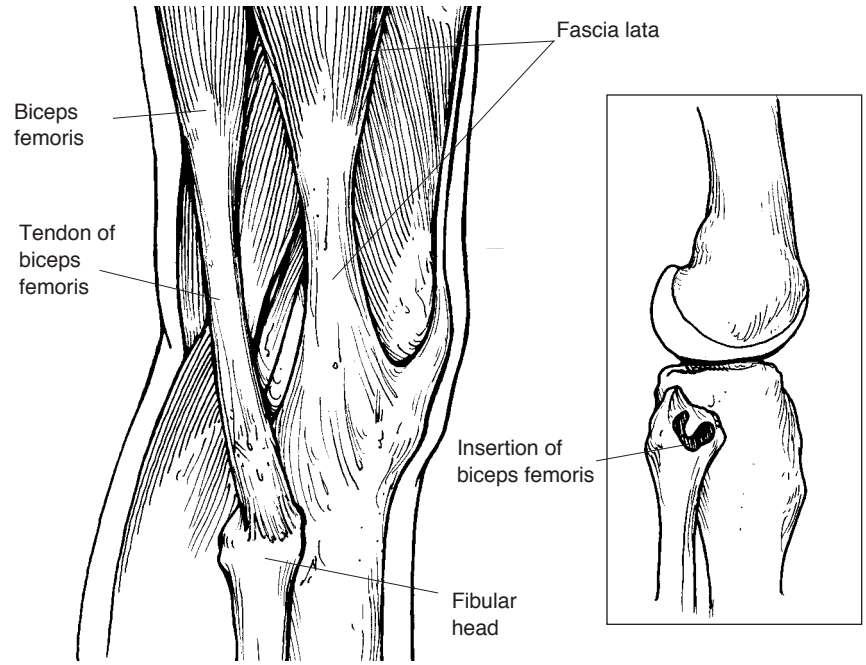

Fig. 3 Insertions of the long and short heads of the biceps femoris at the lateral aspect of the knee.

medial corner of the knee, where it is a significant contributor to the stability of the knee[8] (Fig. 4). The semitendinosus joins the sartorius and gracilis tendons to form the pes anserinus attachment on the proximal medial tibial metaphysis overlying the distal insertion of the medial collateral ligament of the knee[8] (Fig. 5).

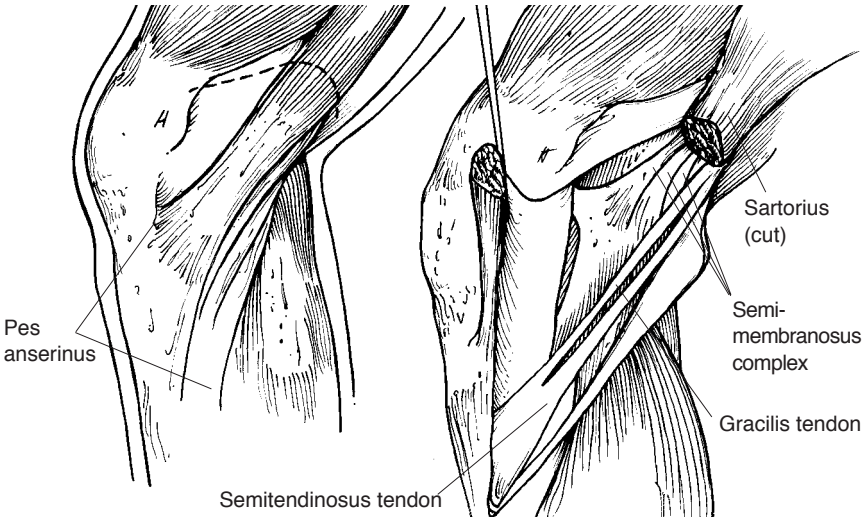

Fig. 4 Insertions of the semimembranosus at the posteromedial aspect of the knee.

The distal musculotendinous complex covers approximately 66% of the length of the biceps femoris and slightly more than 50% of the length of the semimembranosus and semitendinosus muscles. Anatomic study has revealed that the musculotendinous junction extends over the entire length of the biceps femoris, which is why "mid-muscle" tears are nevertheless musculotendinous junction injuries.[6]

Pathology

Experimental and clinical studies of musculotendinous injuries have indicated that the most common site for injury is the myotendinous junction.[3] This was originally proposed in 1933 in the work of McMasters, who produced muscle ruptures by passive extension. Ruptures generally do not occur in the tendon unless there is a preexisting abnormality within the tendon itself. Garrett et al[9] have demonstrated experimentally that failure occurs at the musculotendinous junction regardless of muscle type (fusiform, unipennate, bipennate, or multipennate) or strain rate.

In the simplest injury, when only the myofibrils are damaged, leakage of the cytoplasmic enzyme creatine kinase occurs along with an increase in calcium influx caused by the breach in the plasma membrane. As the injury progresses, the extracellular matrix and fascia become damaged. As the collagen that makes up the fascia is degraded, hydroxyproline is released. This, in turn, leads to the release of muscle enzymes, collagen and proteoglycan degradation, and inflammation. If there is a more extensive muscle injury, blood vessel damage results in bleeding and clotting. A local ischemic environment can be created, causing further muscle damage and edema that leads to a localized

compartment syndrome. This will progress until normal blood flow can be reestablished.[10]

Pivotal considerations in the regenerative process of the injured muscle are the quantity and quality of fibrous scar formation. The interaction between these two processes of muscle regeneration and fibrosis characterizes the effectiveness of the remodeling phase of muscle injury.

With an intact or repaired basal lamina acting as a scaffold, myofibrils can regenerate, but it is imperative to have a properly aligned extracellular matrix to maintain correct myofibril orientation. This is the fundamental principle behind early range of motion of strained muscles to prevent disorganized scar formation and reinjury.[10] The biochemical and cellular responses to injury are important to understanding both the etiology of injury and the diverse treatment modalities that have been utilized for muscle strains.

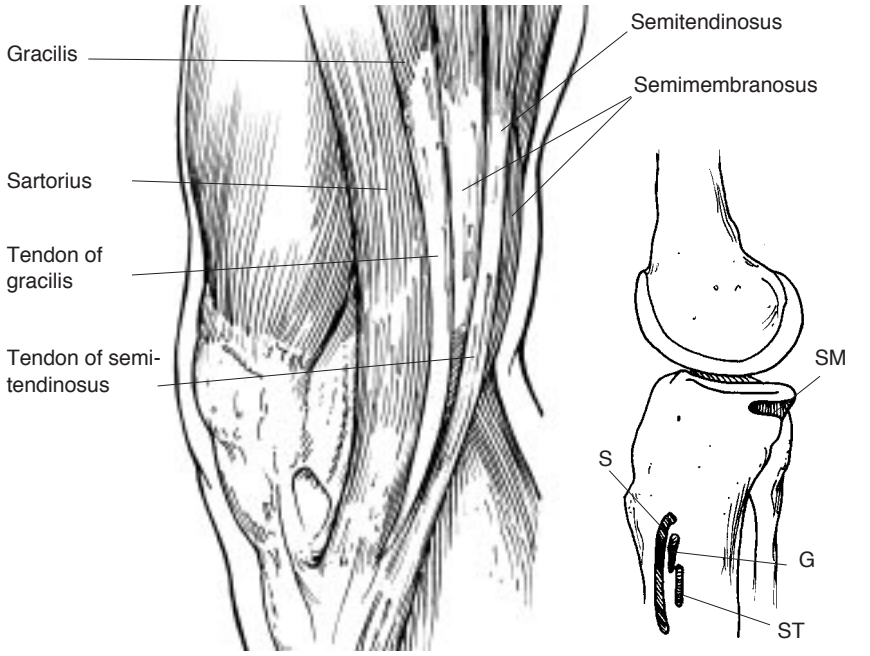

Fig. 5 **Left,** Attachment of the semitendinosus with the pes anserinus at the proximal aspect of the tibia. **Right,** Insertions of the gracilis (G), sartorius (S), semimembranosus (SM), and semitendinosus (ST).

Mechanism of Injury

The two most commonly cited factors in hamstring strain are strength imbalances in the hamstrings and a lack of adequate flexibility.[11] Strength imbalance can refer to a difference in the muscle strength of the hamstrings between limbs or to an alteration in the ratio between flexor and extensor groups. A strength imbalance of 10% or more between the right and left hamstrings or a flexor-extensor strength ratio of less than 0.6 has been proposed as a causative factor in hamstring injury.[11-14] The testing of muscle strength employed cable tension strength tests until 1967, when isokinetic exercise was first introduced by Wyatt and Edwards.[15] Since that time, numerous studies incorporating isokinetic dynamometry have suggested appropriate flexor-extensor ratios, extension torque ratios, and flexion torque ratios.[13,15,16] The flexion-extension ratio of 0.5 to 0.6, which was considered a standard for many years, has been severely scrutinized. It is now evident that these ratios vary between male and female athletes, between athletes in different sports, and between athletes playing different positions on the same team.[14-16]

Evidence suggests that right-left strength imbalances increase the likelihood of strains and sprains in the lower extremity, but this is far from conclusive.[13] Furthermore, the contribution of factors such as flexibility, prior injury, joint laxity, and overall conditioning of the athlete must be evaluated. As a general rule, a ratio of at least 50% to 65% for hamstring strength compared with quadriceps strength is recommended to decrease the chance of sustaining a hamstring strain. More sophisticated testing capabilities have confirmed that this ratio decreases with increased speeds of isokinetic testing.[16] The leg velocity through a normal gait cycle is 233 degrees per second.[16] Isokinetic testing machines should test athletes at their functional speed of 200 to 300 degrees per second. It appears that muscles respond to training in a very specific way, and strength development may be speed-specific.[16]

Identifying weakness in the hamstring muscles may allow prediction of risk for muscle strain and allow a rehabilitation program to correct the problem before injury occurs.[12,17] Nevertheless, other controllable factors, such as muscle fatigue and lack of adequate warm-up, should not be overlooked in the etiology of hamstring strain.[17-19] Worrell[18] has proposed a theoretical model for

hamstring injury that encompasses four factors: flexibility, strength, warm-up, and fatigue.

The fact that the hamstring is a two-joint muscle group is an etiologic factor in its susceptibility to strain—a phenomenon also observed in the biceps brachii, rectus femoris, and gastrocnemius muscles. This is logical because most indirect muscle injuries occur when the muscle is loaded eccentrically during exercise. The mechanism is related to the increased force that is generated during eccentric action of the muscle as opposed to concentric action.

Because the biceps femoris has two heads with different origins and dual innervation, it is considered a "hybrid" muscle.[20] This may also be a predisposing factor in hamstring strains. In the running cycle, the hamstring becomes vulnerable to injury when the constituent muscles decelerate the extending knee during forward swing and also at takeoff, due to the sudden change in function of the muscles from stabilizing the knee in flexion to assisting in paradoxical extension of the knee.

An additional indicator of potential injury is a history of previous hamstring injury. The high percentage of reinjuries suggests that inadequate rehabilitation and premature return to competition may be important factors in recurrent strains.

Clinical Findings

History

Hamstring strains are among the most frequent injuries seen in athletes who participate in the running, jumping, and kicking sports. If avulsions are also considered, other activities, such as water skiing, dancing, weight lifting, and ice skating, become significant. The ubiquitous nature of hamstring strains is evident from their occurrence in both young and old, in the "weekend warrior" and the elite athlete, and in various sports settings. The relative infrequency of hamstring strains in youth may be attributable to the greater flexibility in this age group and the susceptibility of the apophyseal attachment to stretch injury.[21]

The majority of hamstring strains are seen in the acute setting when the athlete experiences sudden onset of pain in the posterior portion of the thigh during strenuous exercise, most commonly sprinting. The injury occurs in the early or late stage of athletic participation, and a history of inadequate warm-up or fatigue may be elicited from the patient. An audible pop is frequently described, and immediate pain limits continuation of activity. Depending on the extent of injury, a fall to the ground or turf may ensue. The athlete may be unable to continue with activity, but can ambulate with only mild discomfort after recovering from the acute event. Another common scenario is for the athlete to feel tightness or an impending "pull" in the muscle during exercise and subsequently limit participation. Not uncommonly, this sensation develops into a chronic situation of recurring tightness in the posterior thigh that restricts the athlete's level of performance. Avulsion injuries usually result from severe hip flexion while the knee is maintained in a position of full extension.[22]

Physical Examination

Because of the relatively homogeneous topography of the posterior portion of the thigh, the deep location of the hamstring muscle group, and the variable degree of injury, it is often difficult to observe or palpate a clear abnormality in many athletes. Acutely, the patient may be lying on the ground grabbing the thigh—a fairly diagnostic sign. There may be palpable induration and tenderness immediately or even a palpable defect in cases of complete muscle tear. Later, the posterior portion of the thigh may be diffusely swollen if there is a severe strain. A minor strain may present no physical findings, whereas a severe tear will be characterized by extensive ecchymosis, swelling, tenderness, and a palpable defect.

It is important to palpate the entire length of the muscle suspected of injury, from origin to insertion. This is best done with the patient prone and the knee flexed to 90 degrees. Extending the knee may stimulate cramping or increased pain, which will limit the examination. The physician should palpate with the muscle fully relaxed and then with mild tension. Even with severe strains, it is often difficult to feel any significant defect.[19] The position of maximum tolerance for a straight leg raise should be recorded, as this has been a useful guide to the severity of the injury and the response to rehabilitation. Another useful guideline is the restriction of passive extension of the knee with the hip flexed to 90 degrees. In this position, active knee flexion will indicate in a relative way the amount of tension that can be generated before pain compared with the normal contralateral leg. On rare occasions, the injury is dramatic, and there is a large defect with an impressive wad of muscle appearing in the posterior thigh during muscle contraction (Fig. 6).

Classification

Muscle injuries can be classified into direct and indirect forms, including lacerations, contusions, strains, delayed-onset muscle sore-

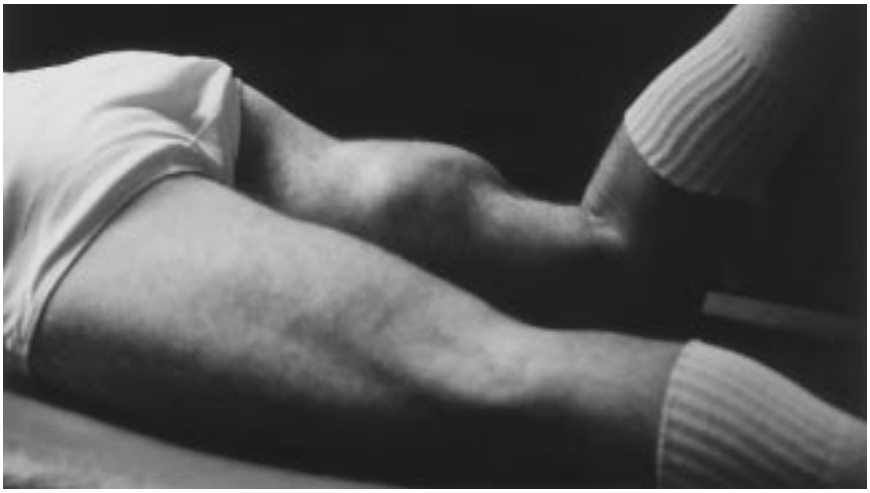

Fig. 6 Clinical photograph of an athlete with a large tear in the hamstring muscle group.

ness, and cramps.[2] The indirect muscle injury categorized as a strain can be either incomplete or complete. The former is known as a muscle pull; the latter, a tear or rupture in lay parlance. Clearly, there is a continuum of injury ranging from minor damage of a few myofibers without loss of structural integrity to a complete muscle tear with fiber disruption. With progression, there is more serious damage to the structure of the muscle and its connective tissue component, with loss of continuity.

For the purpose of clinical assessment and treatment, muscle strains are classified into three groups: mild (grade I), moderate (grade II), and severe (grade III).[23] Grade I, a "pulled muscle," is an overstretching of the muscle that results in disruption of the structural integrity of less than 5% of the musculotendinous unit. Grade II is a partial tear in which the injury is more significant but still constitutes an incomplete rupture of the musculotendinous unit. Grade III is a complete rupture of the muscle that results in a "ragmop" appearance. Severe, grade III strains are rarely seen in hamstring injuries. Avulsion fractures of the ischial tuberosity or the distal insertion are included in this category.[23]

Kujala and Orava[24] have further classified injuries to the ischial apophysis. These include apophysitis, adult tug lesion, painful unfused apophysis, and acute and chronic avulsions. The importance of these lesions is their consideration in the differential diagnosis of ischial pain and the possible need for surgical intervention in some situations. The more serious nature of avulsion injury to the hamstrings and the potential for long-term disability have been documented by more than one author.[21,22,24]

Diagnostic Studies

Plain radiographs are of very little help in the diagnosis of hamstring strains unless an avulsion fracture from the ischial tuberosity is suspected (Fig. 7). Soft-tissue swelling is difficult to appreciate radiographically except in the case of an extensive strain with clinically obvious swelling. Chronic myositis ossificans may be discovered on plain radiographs, but this is quite uncommon.[23] The discovery of ossification or calcification in the soft tissues of the thigh on plain films in association with pain would generally warrant more extensive investigation.

Until the advent of magnetic resonance (MR) imaging, the studies available for evaluation of muscle injury included ultrasonography, scintigraphy, and computed tomography.[25] Computed tomography was used by Garrett et al[6] to visualize the hamstring group in ten collegiate athletes with acute strains in order to localize and characterize their injuries. The injury appears as an area of low density on computed tomographic scans, suggesting the presence of inflammation and edema or bleeding as the major components of the injury. Other techniques for investigation of muscle injury have largely been supplanted by MR imaging.[25] However, because of the expense and inconvenience of the study and the ability to diagnose a hamstring strain from the history and physical examination, MR imaging should be utilized infrequently.

Acute injuries are depicted as areas of high signal intensity on

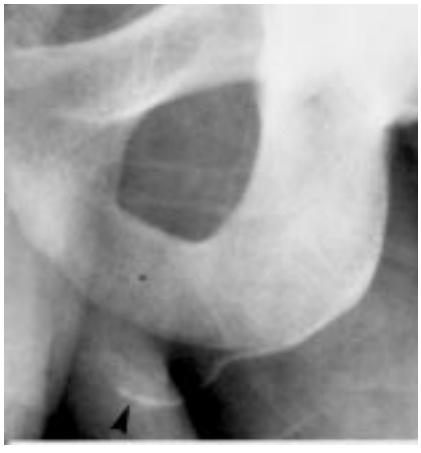

Fig. 7 Radiograph demonstrates an avulsion injury (arrowhead) of the common hamstring tendon.

T2-weighted MR images as a result of the edema and/or hemorrhage within or surrounding the muscle belly. They are usually depicted as areas of intermediate intensity on T1-weighted images. Chronic muscle injuries are somewhat less predictable in appearance. Axial images through the zone of injury provide the most valuable information, but coronal or sagittal images are also helpful in differentiating complete and partial tears when some fibers remain in continuity. If the cost for these studies diminishes, MR imaging may assume greater clinical importance in the evaluation of hamstring strain, but at present there is little indication that the information provided changes the course of treatment.

Treatment

The most useful treatment regimen for all acute strains consists of rest, ice, compression, and elevation, known by the familiar mnemonic device "RICE" (Table 2). The main goal of treatment in the first 3 to 5 days after injury is control of hemorrhage, edema, and pain. A gradual increase in range of motion and

Table 2
Treatment Protocol for Hamstring Strain*

Phase		Goals	Treatment
I (acute)	3 to 5 days	Control pain and edema	Rest, ice, compression, elevation
	1 to 5 days	Limit hemorrhage and inflammation	Immobilization in extension, NSAIDs
	After 1 to 5 days	Prevent muscle fiber adhesions	Pain-free PROM (gentle stretching), AAROM
	Up to 1 week	Normal gait	(Crutches)
II (subacute)	Day 3 to >3 weeks	Control pain and edema	Ice, compression, and electrical stimulation
		Full AROM	Pain-free pool activities
		Alignment of collagen	Pain-free PROM, AAROM
		Increase collagen strength	Pain-free submaximal isometrics, stationary bike
		Maintain cardiovascular conditioning	Well-leg stationary bike, swimming with pull buoys, upper body exercise
III (remodeling)	1 to 6 weeks	Achieve phase II goals	Ice and compression
		Control pain and edema	Ice and electrical stimulation
		Increase collagen strength	Prone concentric isotonic exercises, isokinetic exercise
		Increase hamstring flexibility	Moist heat or exercise prior to pelvic-tilt hamstring stretching
		Increase eccentric loading	Prone eccentric exercises, jump rope
IV (functional)	2 weeks to 6 months	Return to sport without reinjury	Walk/jog, jog/sprint, sport-specific skills and drills
		Increase hamstring flexibility	Pelvic-tilt hamstring stretching
		Increase hamstring strength	Prone concentric and eccentric exercises
		Control pain	Heat, ice, and modalities; NSAIDs as needed
V (return to competition)	3 weeks to 6 months	Avoid reinjury	Maintenance stretching and strengthening

* Abbreviations: AAROM = active-assistive range of motion; AROM = active range of motion; NSAIDs = nonsteroidal anti-inflammatory drugs; PROM = passive range of motion.
† Concentric high speeds at first, proceeding to eccentric low speeds.

use of strengthening exercises is followed by a gradual resumption of activities. This may take several days to weeks, depending on the extent of injury, the activities anticipated, and the level of competition. Since there are two competing processes at work in the healing of skeletal muscle injury, one resulting in muscle regeneration and the other resulting in production of connective tissue scar, it is imperative that treatment maximizes the former and minimizes the latter.

Rest

The influence of rest on the healing of skeletal muscle injury can be related to the effects of immobilization and mobilization. Complete immobilization has fallen into disfavor because it results in muscle atrophy and loss of flexibility and strength. Nevertheless, Järvinen and Lehto[26] have shown that a short period of immobilization is advantageous in limiting the extent of connective tissue proliferation at the site of injury. The optimal length of time for immobilization has not been well defined for human skeletal muscle strains, but less than 1 week has been recommended.[26] During the immobilization period, the muscle should be kept under tension to maximize the healing response and limit contracture. Aggressive mobilization immediately after muscle injury results in dense scar formation that can impair muscle regeneration.

Early controlled mobilization guided by pain tolerance and initiated after 1 to 5 days of immobilization can allow better regeneration, orientation, and alignment for the injured muscle fiber. The weakened muscle is more susceptible to further injury until it regains normal capacity for energy absorption. This occurs within 7 days in the laboratory setting. Early mobilization is also critical to the early return of mechanical properties of the muscle and reduction in muscle tissue necrosis.[27]

In the acute setting, crutches or even bed rest may be warranted for the more severe grade 2 and grade 3 hamstring strains, but complete immobilization of the knee or hip is rarely necessary. Crutches are discontinued when the patient can ambulate without an alteration in gait. Early motion is important, but must proceed in a controlled fashion.

Ice

Cold therapy is well accepted in athletic circles as the primary first-aid intervention for acute soft-tissue injuries. Cold can be applied in a variety of forms, such as ice, gel packs, chemical mixtures, and coolant sprays.[28] The most efficacious and cost-effective method is ice application.

Physiologic effects on inflammation, metabolism, circulation, and nerve conduction are produced by cold therapy, and most of these effects are beneficial to the healing process. Inflammation and edema are delayed but not prevented by cold application; however, cold alters enzymatic activity to slow metabolism, which may limit the spread of the zone of injury in muscle strains. Vasoconstriction also results from cold application, but the 5- to 10-minute delay in the vascular response probably prevents any major beneficial effect related to reduction in hemorrhage. One of the most neglected aspects of cryotherapy is the analgesia it can provide through the slowing of nerve conduction. Further pain reduction happens as a consequence of the decrease in muscle spasm produced by cold.

Deleterious results of cold treatment occur in individuals with cold allergy, Raynaud's phenomenon, or cryoglobulinemia. Other relative contraindications include rheumatologic disease, cardiovascular disease, pheochromocytoma, and anesthetic skin.[28] In the absence of these conditions, ice should be applied after an acute hamstring strain using crushed or cubed ice in a plastic bag and wrapped directly over the posterior thigh with an elastic bandage. It should remain in place for approximately 20 to 30 minutes and be reapplied at least two to four times a day or as frequently as every 2 hours for the first 48 to 72 hours.[18,28]

Compression

Compression is considered efficacious in reducing hemorrhage and thereby limiting the inflammatory response and subsequent soft-tissue scarring. Edema control may be a secondary effect, although it seems that the fluid is simply displaced from the subcutaneous region temporarily. A further beneficial effect may be the proprioceptive feedback stimulated through a firm compressive bandage on the skin. No studies are available to document the efficacy of compression alone as a treatment modality for muscle injury.

Elevation

The final component of the treatment regimen is elevation of the injured body part above the level of the heart. For hamstring strains, this essentially requires bed rest in the immediate postinjury period. A reduction in edema is one goal of elevation, but immobilization may be the primary therapeutic effect. Unfortunately, there are as yet no scientific studies documenting the beneficial nature of elevation in the treatment of soft-tissue injury.

Medication

Inasmuch as inflammation is a key feature in the body's response to stretch-induced injury to muscle, nonsteroidal anti-inflammatory drugs (NSAIDs) are an almost uni-

versally accepted therapy. They work through the inhibition of prostaglandin production. It is prostaglandin that serves as one of the mediators in the inflammatory process, but reduction in prostaglandin levels does not always correlate with beneficial results in muscle-injury models.[27] Nonsteroidal anti-inflammatory drugs have little effect on the other mediators of the inflammatory process that play a role in perpetuating the response, including histamine, serotonin, and oxygen free radicals. The anti-inflammatory outcome of NSAID usage may be surpassed by the analgesic effect, which allows the patient to resume activity more quickly and participate more effectively in the rehabilitation program.

The only controversial aspect to the use of NSAIDs is the appropriate timing of administration. Almekinders[27] recommends starting NSAIDs immediately after injury and discontinuing them after 3 to 5 days, as further use is known to have potential side effects, such as gastric irritation and interference with the repair and remodeling of regenerating muscle. Other research indicates that NSAIDs interfere with the chemotaxis necessary to bring in the cells essential to the laying down of new muscle fibers, thereby inhibiting the healing response. This would suggest that delaying their administration for 2 to 4 days might be more beneficial.

Rarely will a patient require narcotic pain medication, except in the instance of avulsion fractures from the ischial tuberosity. Other medications, such as corticosteroids, muscle relaxants, hyaluronidase, and other proteolytic enzymes, have been advocated by some; however, there is little support in the literature for their use. Recent attention has focused on the role of oxygen free radicals in the initiation and/or perpetuation of the injury process and whether vitamin E or other antioxidant vitamins can ameliorate this effect.[29,30] To date, results on the usefulness of these vitamins are conflicting. There is little evidence to support routine dietary supplementation with antioxidant vitamins.

Therapeutic Exercise

As the injured hamstring muscle progresses through the phases of healing, specific treatment is directed toward restoring strength and flexibility to the muscle. This is critical not only from the standpoint of the competing processes of muscle regeneration and scar formation but also for the preventive effects of avoiding reinjury. The beneficial results from an exercise program have been demonstrated in collegiate athletes by Heiser et al.[17] In their retrospective and prospective study, they concluded that isokinetic testing and rehabilitation could identify muscle imbalances and allow correction, thus preventing hamstring strain or recurrence of a strain.

In the early period after hamstring strain, it is important to initiate muscle action to prevent atrophy and promote healing. Since motion may initially be limited and painful, isometric exercise is started first so that the limb can be positioned comfortably. Commencing the rehabilitation program with multiple-joint-angle, submaximal isometric contraction (e.g., two to three sets of five repetitions, 5-second contraction, varying by 15- to 20-degree increments)[18] limits tension in the injured muscle, avoiding the risk of reinjury.

As motion improves and pain resolves, the isometric exercise can be replaced by isotonic exercise with light weights, which can be increased daily in 1-lb increments. This program involves the muscle in concentric contractions and should produce no pain. Eccentric muscle activity is avoided because of the increased tension it produces within the muscle. When the athlete is progressing pain-free through the prone hamstring exercise program, a high-speed, low-resistance isokinetic exercise program is incorporated. Machines that create only concentric contractions are utilized because eccentric contractions cause greater forces per contraction than concentric work. Isokinetic exercises are advanced as tolerated to include higher resistance and slower speeds.

The swimming pool and the stationary bike can also be useful in the early stage of rehabilitation because they allow pain-free motion and controlled resistance exercise. In the first few days after injury, the athlete can often walk in the pool when walking on land is still difficult. Generalized conditioning can be maintained in the pool by using pull buoys to support the legs while swimming laps with an arm stroke. Hamstring curls in the water are also beneficial during the early postinjury period. As the athlete improves, running in a supportive vest and swimming with a kick board increase the workload.

Other general conditioning activities can include an upper-body program and well-leg exercises. When the patient finally reaches the point where there is a normal gait, little tenderness, and improving muscle strength, a walking program is begun on the track with gradual inclusion of a walk/jog. This is progressed as tolerated by adding more jogging, longer duration, and increased speed.

As the patient gains strength, endurance, and flexibility, a return to customary activities is possible.

Isokinetic testing may provide some useful information on the strength balance and degree of persistent deficit in strength, but the final decision on return to play must be a clinical one based on the athlete's progress in functional activities.[18] Treatment protocols for hamstring strains have been divided into phases that provide useful guidelines for the treating physician, patient, parent, coach, and trainer (Table 2).[18]

Stretching

Loss of flexibility is a characteristic feature of hamstring strains and is probably related to both the pain and inflammation of the injury and the proliferation of connective-tissue scar formation at the injury site, as confirmed both histologically and radiographically.[6] Stretching is therefore critical in the treatment regimen and should be started early. Pain during exercise may be an indication that the capacity of the soft tissue to absorb energy has been exceeded, resulting in inflammation. Accordingly, stretching exercises should begin with gentle active stretching and progress to passive static stretching as pain allows. Worrell[18] has emphasized the advantage of hamstring stretching in an anterior pelvic tilt and minimized the advantage of proprioceptive neuromuscular facilitation stretching over static stretching. Proprioceptive neuromuscular facilitation stretching generally requires an assistant who is trained in the technique, such as a physical therapist or athletic trainer, but it is advocated by some as preferable for gaining and maintaining flexibility.

Operative Considerations

Operative treatment is rarely considered for the treatment of muscle strains, and this holds true for hamstring strains. Only with a complete rupture of the proximal or distal attachment of the musculotendinous complex into bone (with or without bone avulsion) is surgery a realistic consideration.[21,22,24] To avoid long-term disability, complete hamstring avulsions from the ischial tuberosity (including bone avulsions with 2 cm or more of displacement) should be repaired. Distal musculotendinous injuries are usually present in serious injuries to the knee joint, as in biceps femoris tendon ruptures associated with posterolateral corner injury. Distal avulsions should be treated like proximal ones when these rare injuries occur in isolation.

Summary

Hamstring strains are common problems for athletes. The diagnosis and treatment of these injuries has been largely based on trial and error and hearsay. The past two decades has seen a dramatic increase in the basic science related to the musculotendinous system, providing a scientific basis for treatment. With incorporation of this knowledge into orthopaedic practice, the severity of hamstring strains will be recognized, and their treatment can follow an established protocol designed to restore function as quickly and completely as possible. Education of the patient (and often his or her parent and/or coach as well) is crucial to this process, as inappropriate stretching or exercise can create further injury. This is seen most often when athletes return to competition prematurely without adequate rehabilitation. Surgery is rarely indicated except in cases of proximal avulsion.

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