Muscle Strain Injuries: Research Findings and Clinical Applicability

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Abstract and Introduction

Abstract

Muscle strain injuries occur when muscle is elongated passively or activated during stretch. Eccentric contraction of the muscle contributes to injury by generating high muscle forces during lengthening, adding to the forces already produced by the passive connective tissue element. The most common hamstring strain seen in the doctor's office typically involves only one muscle, usually the biceps femoris. Extensive injuries involve more than one muscle, typically at the common tendon of origin of the hamstrings. Cyclic stretching appears to be beneficial, but stretching that leads to forces in excess of 70% may make the muscle more, rather than less, susceptible to injury. Viscoelasticity is temperature-dependent, and adequate warm-up is therefore thought to be protective against muscle strains. Previously strained muscle may carry an increased risk of injury. Injection for local pain relief while the muscle is still injured may not be appropriate because the lack of inhibition from pain could result in excessive stress on the muscle, increasing the risk of additional injury. Clinical observation and the medical literature suggest that muscle strain injuries occur late in either training sessions or competitive settings. Nonsteroidal anti-inflammatory agents may be of some benefit for the early treatment of pain control and functional improvement. However, the delay in the repair process seen histologically raises concern regarding long-term treatment.

Introduction

Typical skeletal muscle injuries, such as contusions, lacerations, strains, ischemia, and complete ruptures usually occur at workor during athletic endeavors. Any of these musculoskeletal ailments can lead to serious pain and disability, causing lost time to both occupational and leisure activity participation. The preponderance of stretch-induced injuries, or strains, is evident to the clinician involved in occupational or sports medicine practices; they can account for up to 30% of all injuries seen.[1,2]

Noncontact or indirect injuries can affect muscle function. Some examples include delayed onset muscle soreness (DOMS), partial strain injury, or complete rupture of the muscle. These injuries make up a continuum that has one thing in common: eccentric exercise-tension development during muscle lengthening.[2,3] In comparison with shortening, or concentric contractions, eccentric contractions generate high forces but activate few motor units.[4]

During unaccustomed exercise, eccentric loading leads to microscopic damage to the contractile portion of muscle in what appears to be random disruptions of the Z-lines.[5] Reversible pain, weakness, and reduced range of motion are the hallmarks of DOMS. Localized pain usually peaks in the 24 to 72 hours following exercise,[6] while weakness and limited range of motion can last for as long as a week.[7,8] There is, however, rapid adaptation of muscle. Successive bouts of the unaccustomed exercise produces progressively less tissue damage and soreness.[6]

Muscle strain injury is characterized by a disruption of the muscle-tendon unit.[9] Local pain and muscle weakness occur during activity. With DOMS, further exercise is protective leading to less pain and damage. In contrast, exercise too soon after the injury, improper rest and rehabilitation, and a minor strain of skeletal muscle frequently proceed to a far more disabling injury that further lengthens the time lost to work and athletics.

Despite the prevalence of muscle strain injuries, our understanding of the pathophysiology, treatment and recovery are limited. Perhaps the natural history, self-limiting nature, and minimal surgical requirements have made stretch-induced injuries of minimal clinical interest. This article reviews stretch-induced injuries and their mechanism, location, and treatment, as well as some relevant clinical observations.

Injury Mechanisms

A basic understanding of how muscles are injured in athletic or occupational settings needs to be appreciated before we can reproduce the injury in the laboratory. Most clinicians and researchers agree that muscle strain injuries occur when the muscle is elongated passively or activated during stretch.[1,3,10] Eccentric contraction of the muscle contributes to injury by generating high muscle forces during lengthening,[4] adding to the forces already produced by the passive, connective tissue element.[11] Eccentric contractions are frequently involved in muscle injuries.[2,3,12]

In the laboratory, standard techniques in the study of muscle mechanics and electrophysiology are used on rabbit hind limb muscles, usually the tibialis anterior (TA) and the extensor digitorum longus (EDL). To research injury patterns, it is necessary to develop a model that allows reproducibility of a strain injury. Activation alone does not result in either a partial or complete strain injury.[13] To produce an injury, stretch is required. The forces needed to cause muscle failure are several times higher than the force normally produced during a maximal isometric contraction,[14] suggesting that passive forces play a role in muscle injury. With that information, a model of muscle injury can be defined. Only an intact muscle of the rabbit hindlimb, with neural and vascular supply intact, can be stretched to failure or activated during stretch.

Injury Resulting From Passive Stretch

Muscles can be stretched from the proximal or distal tendon to failure. Variables of interest include the rate of strain (1cm/sec, 10cm/sec, and 100cm/sec) and muscle architecture (pennation) or mechanical properties of the muscle. Regardless of strain rate or architecture, we found that muscle failed at the most frequently distal muscle-tendon junction, (Fig. 1) leaving a small, inconsistent amount of muscle tissue attached to the tendon.[13] Thus, the location of a stretch-induced injury was predictably near the muscle-tendon junction, but most often was not an avulsion since a small and variable amount of muscle remained attached to the tendon.

Figure 1. Appearance of the tibialis anterior muscle of the rabbit following controlled strain injury. Note the small hemorrhage at the distal tip of the injured (left) muscle at 24 hours.

Another variable of interest in passive strain is the influence of muscle length. While muscle length affects a number of biomechanical properties, there is a wide variability of strain based on the length of the muscle. In any event, there is a passive length, beyond which muscle strain injury occurs. In a previously strained muscle, this length is reduced effectively making the muscle more susceptible to a future strain injury.

Injury Resulting From Active Stretch

Most clinicians would agree that strain injuries occur during powerful eccentric contractions. Nikolaou and colleagues[15] attempted to mimic these contractions in rabbits by isolating and stretching to failure the rabbits' hindlimb muscles. During stretch one of three activation conditions was applied: tetanic stimulation, submaximal stimulation, and no stimulation. The location of failure was, as usual, the muscle-tendon junction and the total strain at failure was similar among the three conditions. Of note, the force generated at failure was only 15% greater in the activated muscles. The energy absorbed (the difference in strain energy between passive and active conditions), however, was about 100% greater in the activated condition (Fig. 2). This indicates that passive elements of muscle can absorb energy, but the ability to absorb energy is greatly enhanced when the muscle is activated.

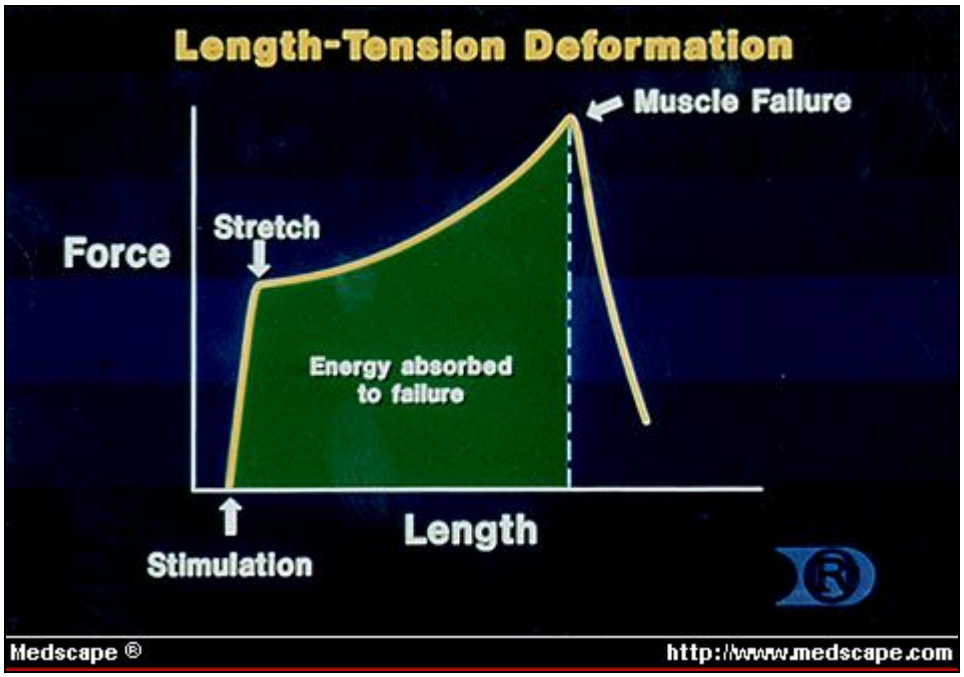

Figure 2. Energy absorbed is the area under each length-tension deformation curve. The figure shows the relative differences in energy absorbed to failure in stimulated vs passive muscle preparations.

Muscles protect themselves and joint structures from injury. The more energy muscle can absorb, the more resistant the muscle is to injury. Both the passive and contractile elements of muscle contribute to the muscle's ability to absorb energy. These passive elements, which are not dependent on activation, include connective tissue and the fibers themselves. The contractile element of the muscle also participates because activation of the muscle increases the ability to absorb energy (Fig. 2). The increase in energy absorbed due to contraction was found to be around 100%. Any setting that diminishes the ability of the muscle to contract also diminishes the ability of the muscle to absorb energy, leaving the muscle more susceptible to injury. Two variables that may contribute to strain injury susceptibility are fatigue and weakness.

Injuries That Are Nondisruptive

Complete disruption of the muscle-tendon unit is one type of muscle injury. Another is the change in linearity of the force-displacement curve of a stretched, inactivated muscle, suggesting a "plastic" deformation that results in an alteration to the material structure. Using this model of the rabbit hind limb, physiological, mechanical, and histological muscle characteristics can be observed.

While the model may result in a nondisruptive injury, ultrastructural damage still occurs. Histological sections of muscle demonstrate the expected damage near the muscle-tendon junction with a variable amount of muscle tissue still attached to the tendon. Some hemorrhaging occurs. An obvious inflammatory response is seen in the first 1 to 2 days after the injury. By the seventh day, fibrous tissue has begun to replace the inflammatory reaction leading to the formation of scar tissue.[15]

This type of tissue damage alters the ability of the muscle to develop tension. Immediately after injury the muscle is able to manufacture only 70% of the normal amount of tension. Within 24 hours, the muscle's ability to create tension further declines to 50% of the contralateral control muscle. Thereafter, tension production improves, and by the day 7, the muscle is able to develop 90% of the tension produced by its contralateral control muscle.

In contrast, if muscle with a 7-day-old nondisruptive injury is stretched, the tensile strength recorded is only 77% of the control muscle.[16] This is well below the level of 90% tension the muscle can attain. As strains are, in part, caused by stretch, this loss of tensile strength may make the muscle more susceptible to a second injury, a scenario frequently seen by clinicians.

Viscoelasticity of Skeletal Muscle

Important factors in preventing muscle strain injuries include flexibility, warm-up, and pre-exercise stretching. The beneficial adaptation to stretching has been most frequently credited to stretch reflex mechanisms and viscoelastic properties of the muscle. Viscoelasticity can be visualized by imagining hanging a weight on a muscle and observing its new length, then watching the muscle slowly continue to increase in length over time. For tendons and ligaments, stretching the tissue to a constant length causes the tension to gradually decrease with time. This is referred to as stress-relaxation. Perform this cyclically and a gradual decrease in tension occurs with each successive stretch.[17]

To determine if similar features are present in the muscle-tendon unit, rabbit hindlimb muscle was stretched from an initial force of 1.96N to 78.4N, held for 30 seconds, then returned to the initial force. This was repeated 10 times (Fig. 3). The length necessary to reach the predetermined tension increased 3.45% over the 10 cycles with 80% of this change in length occurring in the first 4 cycles.[18]

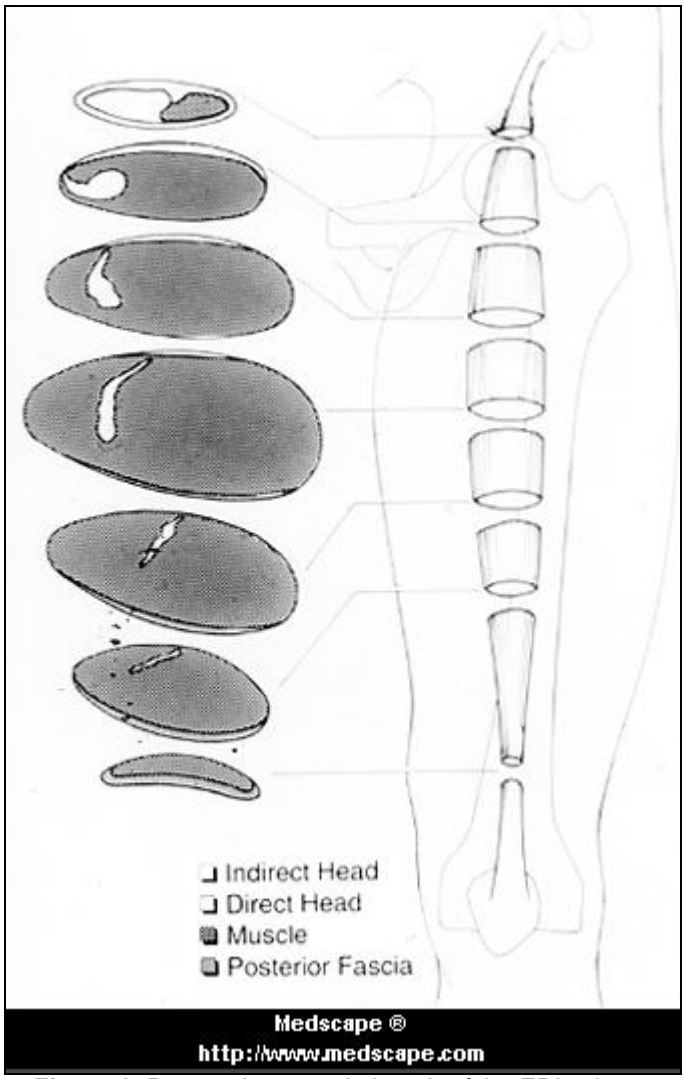

Figure 3. Percent increase in length of the EDL when repeatedly lengthened to a constant tension.

Another way to look at the same feature is to stretch the muscle to 10% above its resting length and return it to its resting length. This cycle also was repeated 10 times (Fig. 4). Tension was reduced by nearly 17% over the 10 cycles with the bulk of the reduction occurring, again, in the first 4 cycles.[19]

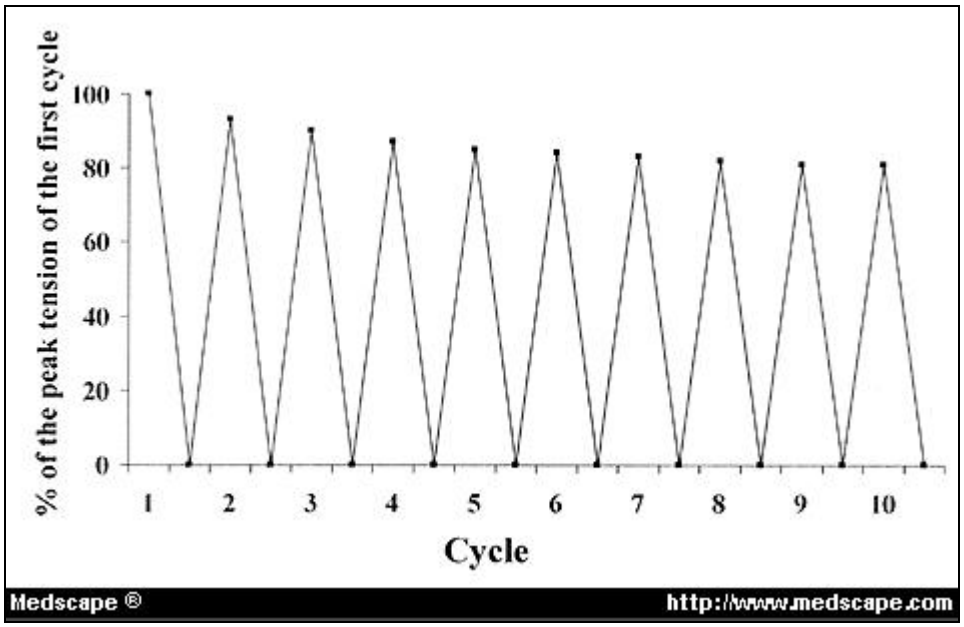

Figure 4. Muscle tension of the EDL when repeatedly stretched to the same length (10% beyond resting length).

These experiments show that repetitive stretching reduces the load on the muscle-tendon unit at any given length. Of interest is the absence of reflex effects or other mediation by the central nervous system. These experiments were repeated for innervated and denervated muscle with no differences noted. Clearly, a large component of the changes seen in muscle due to stretching are a result of inherent muscle-tendon viscoelasticity with neural influence. Certainly there are additional reflex and central nervous system effects on the muscle during stretch, especially during physiological movements.

Clinical Applications

It is important to take the findings of laboratory-based projects and apply them clinically. In the rabbit, it was shown that muscle strain injuries occurred at the muscle-tendon junction. Would this finding also be seen in the injured athlete? To answer that question, acute hamstring strain injuries were evaluated within 48 hours of the injury in 10 college-aged athletes.[20] All were examined clinically and imaged with computed tomography (CT) to determine the location of their injury. All injuries occurred while either sprinting or kicking a soccer ball and were mostly proximal and lateral, typically in the biceps femoris. The common mechanism involved ballistic hip flexion and knee extension. The injured area appeared as a region of hypodensity by CT scanning, suggesting inflammation and edema, but not localized bleeding.The long head of the biceps femoris was most frequently injured in our sample (9 of 10). The injury was localized to the muscle-tendon junction of the common tendon of the hamstrings. The tenth patient (soccer player) injured his semimembranosis while kicking overhead, suggesting a different mechanism from that seen in the sprinters.

Further imaging studies were carried out on a larger sample of athletes with a variety of injuries.[21] Fifty patients underwent CT (n=27) or magnetic resonance imaging (MRI, n=23) to identify their muscle strain injuries. Damage was localized to the quadriceps, hamstrings, adductors, and triceps surae groups. T2-weighted images were found to be better than T1-weighted images for visualizing edema, inflammation, and possible hemorrhage of muscle strain injuries. CT scanning demonstrated the expected areas of low density. Quadriceps strains were isolated to the rectus femoris. Adductor strains were confined to the adductor longus. Of the 17 hamstring strains, 11 were to the biceps femoris, 4 to the semimembranosis, and 2 to the semitendinosis. For the triceps surae group, all injuries were at the distal muscle-tendon junction of the medial head of the gastrocnemius. The effectiveness of both CT and MRI to image strain injuries was demonstrated. More important, the particular muscles susceptible to strain injuries were identified. The muscles were, predictably, two-joint muscles (biceps femoris, rectus femoris, gastrocnemius) or of a complex architecture (adductor longus) and occurred, as can best be determined by CT and MRI imaging, at the muscle-tendon junction.

Additional Types of Injuries

Unexplained muscle injuries, such as persistent strain of the rectus femoris, also exist in the clinic setting. Therefore, we deemed it necessary to determine if our understanding of the nature of the strain injury was consistent with our understanding of the anatomy of the rectus femoris.[22] Cadaveric dissections of the rectus femoris muscle showed the expected direct head that originated at the anterior inferior iliac spine, plus an indirect head that originated from the superior acetabular ridge. The tendon of the indirect head extended well into the mass of the rectus femoris. While prior laboratory work demonstrated that most strain injuries occur superficially at the muscle-tendon junction, clinical evidence pointed to a

strain at the muscle-tendon junction of the deep, indirect head of the rectus femoris. These are quite different from the classic injury near the distal tendon, because asymmetry, chronic pain, and anterior thigh masses are evident. Ten patients with an incomplete intrasubstance strain of the proximal, deep tendon of the rectus femoris were evaluated with physical examination and imaging studies. Patients presented anywhere from 4 to 156 weeks post-injury. Eight of the 10 injuries involved sprinting or kicking (2 could not recall the mechanism) and all but one had pain when running. Imaging studies detected the strain to be in the area of the tendon of the indirect head of the rectus femoris. Surgical exploration was performed on two patients with removal of the muscle in one and the excision of a fibrotic mass in the other. Subsequently, both were asymptomatic and able to return to full activity.[21] The reason for chronic pain in these subjects was not determined, but may be due to differential activation of the superficial and deep portions of the muscle.

Many strain injuries appear to be dependent on the design of the muscle, and our earlier experiences[22] warranted a detailed study of the rectus femoris to determine if these persistent strains were related to some curious architectural feature.[23] The rectus femoris of fresh or embalmed cadavers was dissected. The superficial and deep tendons were confirmed. The tendon of the deep component penetrated nearly the entire length of the muscle. It arose from the superior acetabular ridge and was somewhat medial throughout its course through the muscle. It began as a rounded structure and then flattened out and migrated laterally, and was nearly vertical in the distal third of the muscle (Fig. 5). The pennation of the rectus femoris was more complex than the simple bipennate arrangement normally attributed to the muscle. The proximal third appeared to be unipennate while the distal two-thirds was bipennate. The deep tendon and the bipennate arrangement of the distal portion of the muscle created what appeared to be a "muscle within a muscle." Exploration of three chronic strain injuries showed a pseudocyst consisting of vascular, fibrotic loose connective tissue that surrounded the deep tendon. Serous fluid collected between the connective tissue and the tendon. This anatomic finding is consistent with CT or MRI images of vascular fibrotic processes of the deep tendon of the indirect head.

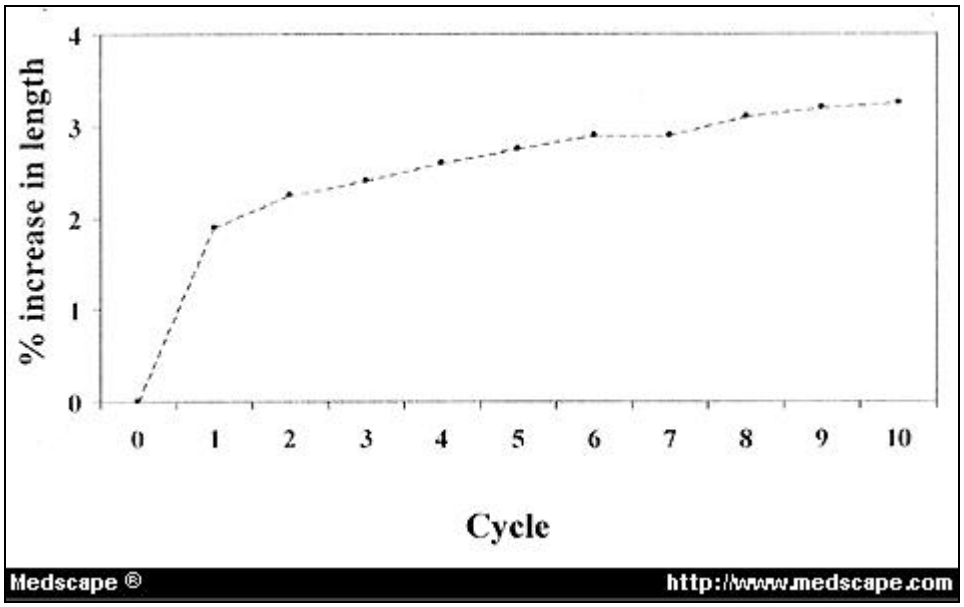

Figure 5. Architecture of the indirect head of the rectus femoris muscle.

The most common hamstring strain seen in the doctor's office typically involves only one muscle, usually the biceps femoris. Extensive injuries involve more than one muscle, typically at the common tendon of origin of the hamstrings. A unique mechanism of severe hamstring strain injury occurs in water skiers.[24] The novice skier assumes a crouched position prior to being pulled by the boat into a standing position. If the skier extends the knees too soon, the ski is forced down into the water. Forward momentum of the boat pulls the skier forward, leading to excessive hip flexion while the knees are extended. This powerful stretch leads to either a muscle-tendon junction injury or to a more disabling injury involving avulsion of the tendinous origin from the ischial tuberosity. Hamstring strains can also occur in experienced skiers secondary to a separate mechanism -- falling forward on a single slalom ski. Twelve water skiers with a history of skiing-induced hamstring injuries were followed for 0.5 to 18 years post-injury. All patients realized they had a significant injury at the time of the accident. Complete or partial avulsion had occurred at the proximal tendon. The extent of the injury was obvious on physical examination, revealing distal tendon retraction of the hamstring muscles and visible asymmetry. Conservative management of this injury leads to a poor prognosis; surgical repair is an alternative. Three of the 12 patients were treated surgically. Acute surgical repair of these injuries can lead to acceptable results. If surgery is delayed, the repair is difficult and only partial restoration of function is likely. If surgery is not elected, the patient must strengthen the remaining hamstrings and modify his activities of choice. Seven of the 12 patients returned to prior athletic activity at a lower level, and the rest, all with complete disruptions, were hampered in sports involving running or requiring agility.

Acute groin injuries are also common in sports, especially in the game of soccer.[25] The adductor longus can be injured during hip abduction. Direct and indirect hernias may also occur. In addition, there is an abnormality in the lower abdominal

wall musculature causing a vague and poorly localized pain in the groin. This pain is noted during high-intensity, ballistic movements such as kicking a soccer ball or sprinting, and is most common in high-caliber athletes during intense training and competition. This "athletic pubalgia" is associated with pain and muscle-tendon injury in the inguinal area near the attachment of the rectus abdominis to the pubis and in the adjacent internal oblique muscles near the area of abdominal weakness where direct inguinal hernias present themselves. However, this pain may exist without any evidence of herniation. When conservative measures fail, a herniorrhaphy can provide excellent relief.

The clinical investigations referred to above should be interpreted in light of the basic science. It was demonstrated that imaging studies and direct observation identified muscle disruption near the muscle-tendon junction in common muscle injuries. Disruption did not occur in the midsubstance of the muscle fibers. The muscle-tendon unit could also be injured within the tendon or at the tendon-bone junction. Eccentric activation was the common mechanism of injury as the basic studies suggested.

Prevention Strategies

Repetitive Stretch and Failure Properties

Prior work suggests that viscoelastic properties of muscle contribute greatly to changes in muscle length, and increased length can be seen to decrease strain in a muscle. A more practical question is the use of stretching to prevent muscle strains. To study stretching, repeated stretch-release cycles were studied using the rabbit model.[26] First, the force to failure of the hindlimb muscle was determined. Then the contralateral muscles were cyclically stretched to 50% or 70% of that force to failure. Ten cycles to 50% of failure force resulted in an increase in muscle stretch at failure with no change in the force at failure or energy absorbed. When muscles were cyclically stretched to 70% of failure force, macroscopic evidence of failure was seen even before the 10 cycles were completed. Thus, cyclic stretching appears to be beneficial and stretching that leads to forces in excess of 70% may make the muscle more, rather than less, susceptible to injury.

Warm-Up

Viscoelasticity is temperature-dependent and warm-up is therefore thought to be protective against muscle strains. An attempt was made to mimic warm-up that was due to prior activity[27] instead of external heating. Rabbit hindlimb muscle was held isometrically and tetanically stimulated for 10 to 15 seconds, resulting in a 1°C rise in muscle temperature. The muscle was able to stretch more prior to failure and with more force. While the changes might be due to temperature elevation, the effects of stretch cannot be discounted in spite of the muscle being held isometrically. A constant length still must allow for some stretch of the muscle-tendon unit as the fibers contract and elastic components become stretched.

Prior Injury

Previously strained muscle appears to carry an increased risk of injury. Patients with major muscle strains often describe prior minor injuries. This would suggest that after a minor injury, mechanical characteristics of the muscle are somehow altered. Such alterations might precede a more major injury. To determine the mechanical characteristics of a muscle with a minor strain, the EDL of rabbits underwent a nondisruptive strain by stretching the muscle just short of tissue rupture.[18] Isometric and isotonic contractile properties of the control muscle were used for comparison. Finally, the muscle was passively stretched to failure at a rate of 10cm/min. The peak tensile load and length at that load were derived for use on the experimental contralateral limb. The length change to peak load (of the control limb) was duplicated in the experimental muscle, just short of a disruptive injury. The injured muscle was then subjected to passive stretch to failure. Histology was performed on the muscles with minor injuries in a subset of rabbits. In the experimental muscles, the peak load to rupture was 63% of control and the length at rupture was 79% of control. Isotonic shortening was reduced by 51% and 6% for 100-gm and 1000-gm weights, respectively. The minor strain injury caused incomplete disruptions along the muscle-tendon junction. Thus, a prior minor injury makes the muscle more susceptible to another injury. This suggests that early return to activity prior to complete healing increases the risk for further, more major, injury. In addition, aggressive rehabilitation designed to return an athlete to competition can be too stressful for the muscle, risking further injury. Injection for local pain relief while the muscle is still injured may not be appropriate because the lack of inhibition from pain could result in excessive stress on the muscle, increasing the risk of additional injury.

Fatigue

Clinical observation and the medical literature suggest that muscle strain injuries occur late in either training sessions or competitive settings. This leads one to conclude that fatigue must play some role in the risk of muscle injury. Mair and colleagues[28] fatigued the EDL of rabbits to 25% or 50% of the force of the contralateral control by cycles of 5-second isometric tetanic contractions, followed by 1-second rest. Muscle was activated while being pulled (at 1cm/sec, 10cm/sec, or 50cm/sec) to failure. Similar data were collected on the unfatigued contralateral control. The force and length at failure were determined as well as the energy absorbed prior to failure. There was a trend toward a reduction in force for all groups (strain rates) tested. Strain rate did not influence force at failure, and there was no change in muscle length at any of the strain rates. Significantly less energy was absorbed in both fatigue conditions, with the greatest loss occurring in the

most fatigued muscle. The reduction in absorbed energy was the greatest when the muscle was pulled at 1cm/sec. The slower the muscle stretch-rate, the greater the energy that was absorbed. Muscles absorb energy while controlling and regulating limb movement. These data indicate that muscles become damaged at the same length, regardless of fatigue. In contrast, fatigued muscle is unable to absorb energy prior to reaching the amount of stretch that causes injuries. Proper conditioning to delay fatigue is seen as a part of a rationale for the prevention of muscle strain injury.

Treatment of Muscle Strain Injuries

The pain of a muscle strain may prompt physicians to prescribe anti-inflammatory drugs in response to the inflammatory responses known to occur following an injury -- a treatment that is largely empirical. Before wide use of anti-inflammatory drugs can be accepted, the effects of such medication on muscle recovering from an injury need to be evaluated. Obremsky[16] caused a strain injury of the tibialis anterior in 50 rabbits (strain rate of 10cm/min) that were subsequently administered piroxicam (16mg/kg) within 6 hours, plus 13mg/kg every 6 hours thereafter. Forty served as control and received no medications. Contractile properties and histology were determined at 1, 2, 4, or 7 days after the injury. On day 1, there was a significantly greater force in the treated animals. There was no difference between the treated and untreated animals on days 2, 4, or 7. The treated animals' muscles showed a delay in the histological repair process, as well we delayed inflammatory cell infiltration, necrosis, myotube regeneration, and collagen deposition. Based on these results, nonsteroidal, anti-inflammatory agents may be of some benefit for the early treatment of pain control and functional improvement. However, the delay in the repair process seen histologically raises concern regarding long-term treatment.

Routine treatment of muscle strain injury emphasizes the restoration of flexibility and muscle strength. During this period of rehabilitation, exercise and stretching must be controlled to protect the injured tissue. As rehabilitation continues, the patient must be cautioned against rapid return to activity until near normal flexibility and strength are achieved. This reduces the risk of a more major injury from too rapid a return to sports.

Conclusions

One of the most common injuries seen in the office of the practicing physician is muscle strain. Until recently, there were little data available on the basic science and its clinical application for the treatment and prevention of muscle strains. Studies in the last 10 years represent follow-up to investigations of muscle strain injuries from the laboratory and clinical settings.

Studies from the laboratory indicate that certain muscles (muscles that cross multiple joints or have complex architecture) are most susceptible to strain injury and have a strain threshold for both passive and active injury. Strain injury is not the result of muscle contraction alone; they are produced by excessive stretch or stretch while the muscle is being activated. When the muscle tears, the damage is very close to the muscle-tendon junction. Following injury, the muscle is weaker and is at risk for further injury. The force output of the muscle eventually returns while the muscle undertakes a predictable progression toward tissue healing.

Current imaging studies have been used to document the muscle-tendon junction as the site of injury. The most commonly injured muscles include the hamstrings, the rectus femoris, the gastrocnemius, and the adductor longus. Injuries inconsistent with the involvement of a single muscle-tendon junction proved to be located at tendinous origins rather than within the muscle belly. Injuries associated with a poor prognosis have been identified as potential candidates for surgery. These include injuries to the rectus femoris, the hamstring origin, and the abdominal wall.

Management techniques of common muscle injuries are available from sports organizations and have been described in the literature, and the risks of re-injury have been documented. Early efficacy and potential for long-term risks of nonsteroidal anti-inflammatory agents have been described as well.

New data demonstrate the beneficial effects of warm-up, temperature, and stretching on the mechanical properties of muscle, potentially reducing the risk of strain injury to the muscle. Fortunately, many of the factors protecting muscle, such as strength, endurance, and flexibility are also essential to the muscle's maximum performance. Future studies are intended to delineate the repair and recovery process, emphasizing not only the recovery of function, but also the susceptibility to re-injury during the recovery phase.

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This research agenda could not have been conducted without the able assistance of the following colleagues: Louie Almekinders, Frank Bassett, Tom Best, James Dalton, Rich Glisson, Carl Hasselman, Chad Hughes, John Lohnes, Scott Mair, Pantelis Nickolaou, Tom Noonan, William Obremsky, Ross Rich, Marc Safron, Peter Sallay, Tony Seaber, Kevin Speer, and Dean Taylor.