Muscle-Tendon Junction Injury

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5.1 Introduction

Muscle strain injury is one of the most frequently occurring and debilitating injuries in sport and recreation (Glick 1980; Krejci and Koch 1979; Nikolaou et al. 1987; O'Donoghue 1984; Ryan 1969). Both partial and full muscle strain injuries result in significant short-term and long-term consequences (Garrett et al. 1988). These consequences include time lost from work at significant compensation costs and inability to regain pre-injury levels of performance (Garrett et al. 1988). Despite having knowledge of the consequences, the understanding of muscle and tendon injuries is still vastly unknown. The exact anatomical location of damage within the muscle-tendon junction and the predisposing factors for damage in normal, healthy muscle are still unanswered questions surrounding muscle strain injury (Garrett et al. 1988). Our understanding of muscle and tendon injuries has expanded considerably in the last 30 years. Much more is known about the pathophysiology of injury in animal model and in clinical practice. However, much remains to be determined about prevention and treatment.

5.2 Structure

The structural link between the myofilaments and the connective tissues is the muscle-tendon junction (Garrett et al. 1988). The muscle-tendon

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junction is responsible for aiding in the transmission of tension within the muscle (Garrett et al. 1988). The tendon fibrils insert on the muscle fibers through an elaborate system of folds which increases their surface area (Schwarzacher 1960). The muscle-tendon junction, otherwise referred to as the connecting domain, has a vast number of fine filaments which are oriented approximately perpendicular to the major force vector (Trotter et al. 1981, 1983). These fine filaments connect the terminal myofibrils of the lamina densa to the collagen fibrils of the tendon (Trotter et al. 1981, 1983). These filaments have a diameter of two to seven nanometers (Ajiri et al. 1978; Hanak and Bock 1971; Korneliussen 1973). New sarcomeres are developed at the muscle-tendon junction with growth and development and/or hypertrophy as a result of stretch (Williams and Goldspink 1971, 1973, 1978). The ability to develop new sarcomeres and the plasticity at the terminal sarcomeres, located within the muscle fibers, demonstrate the available plasticity of the skeletal muscle (Williams and Goldspink 1971, 1973, 1978). The plasticity allows for localized change within the muscle-tendon junction, in addition to the possibility of regional structural and functional specializations (Garrett et al. 1988).

Important in the overall structure and function of the muscle-tendon unit are the tendon and ligament. Previous studies have examined the viscoelastic characteristics of tendons and ligaments (Butler et al. 1978; Solveborn 1983). When a tendon or a muscle is loaded with a constant force, the length slowly increases without increase in a time-dependent (therefore viscoelastic) manner (Solveborn 1983; Butler et al. 1978). Elastic structures produce a constant deformation for a constant load (Butler et al. 1978). The length-tension relation changes with load and therefore displays viscoelastic behavior rather than strict elastic behavior (Butler et al. 1978). Increased length in response to a constant force is called creep (Butler et al. 1978). If a muscle or tendon is stretched to a constant length, the measured tension decreases (Butler et al. 1978). The decrease in tension over time is also referred to as stretch relaxation (Solveborn 1983; Butler et al. 1978). Cyclic stretching of the ligament and tendon to the same length results in a decrease in tension with each stretch performed (Butler et al. 1978; Solveborn 1983). Descriptions of the muscle-tendon unit under two types of cyclic repetitive stretching have been provided by previous research (Taylor et al. 1985b, 1990). With repetitive cyclic stretching to the same length, there is an 80% increase in total length during the first four stretches (Taylor et al. 1985b). The stretch relaxation curve following the first stretch is significantly different than the stretch relaxation curve following the second stretch (Taylor et al. 1985b). The third and fourth stretch relaxation curves are also different, but the final six curves demonstrate no differences between them (Taylor et al. 1985b, 1990). Another study, similar to the previously described study, demonstrated that the peak tension decreased with each stretch, in addition to an overall drop in peak tension of 16.6% between the first and tenth stretch (Taylor et al. 1985c). There is a significant decrease in peak tension between the first four stretches, but the peak tension in cycles five through ten do not differ (Taylor et al. 1985c). This information suggests that repetitive stretching will lead to a reduction of load on the muscle-tendon junction at a given length (Taylor et al. 1985c). This effect is independent of any reflex effects or other influences mediated by the central nervous system; reflex effect and central nervous system influences may be involved in addition to the viscoelastic response (Taylor et al. 1985a).

The muscle-tendon unit is viscoelastic (Taylor et al. 1990). The decline in peak tension, which occurs because of the viscoelastic property of stretch relaxation, is associated with the internal changes in the structure of the muscle (Taylor et al. 1990). With each stretch, the relaxation curve for the muscle-tendon unit gradually levels off at higher tensions than the preceding relaxation curve (Taylor et al. 1990). Although a constant amount of tension is not always maintained to cause a length increase, varying amounts of tension up to a maximum lead to elongation (Taylor et al. 1990). This is demonstrative of the creep property of the muscle-tendon junction, forming a curve toward a maximum deformation

(Taylor et al. 1990). Both peak tensile force and absorbed energy are dependent upon the rate of stretch applied (Taylor et al. 1990). Stretch rate dependency is the amount of stress relaxation that occurs in a given amount of time (Taylor et al. 1990). Slower stretches allow for a greater degree of stretch relaxation to occur, resulting in lowered peak forces (Taylor et al. 1990). Energy is absorbed by the muscle-tendon unit during the loading process and is then dissipated during the unloading process (Taylor et al. 1990). During any one stretch, the rate at which the muscle-tendon unit absorbs energy is different from the rate at which it dissipates energy (Taylor et al. 1990). This creates a discrepancy between energy put into the system and the energy released form the system (Taylor et al. 1990). This difference may be explained by heat transfer and/or by internal changes within the ultrastructure of the muscle (Taylor et al. 1990).

5.3 Function

The function of the muscle and muscle-tendon junction has implications for muscle strain injury. The amount of stretch that a muscle may endure impacts the likelihood of having a muscle strain injury (Garrett 1990). Muscles that act on two joints are subjected to more stretch than muscles that act on one joint and are, therefore, more likely to suffer muscle strain injuries (Brewer 1960). The ability to be stretched at more than one joint, based on anatomical location, may play a role in the risk of muscle strain injury associated with these two joint muscles (Brewer 1960; Garrett 1990). These two joint muscles have an intrinsic tightness; in turn they can limit the ability to produce range of motion, but normal physiological joint motion can place the muscles in a position of increased passive tension (Brewer 1960; Garrett 1990). Often times these types of muscles control the regular movement when they are eccentrically contracted (Brewer 1960; Garrett 1990). More specifically, they act to produce joint motion control or decelerate the joint, both actions that are eccentric in nature (Brewer 1960; Garrett 1990).

Normal muscle activated by way of nerve stimulation, which has been previously theorized to lead to muscle strain injury, does not cause complete or incomplete disruption of the muscle (Garrett et al. 1984a). Although force is diminished and there is a failure of the excitation, there is no disruption of the muscle and no muscle strain injury (Garrett et al. 1984a). In order to see any gross microscopic muscle injury, stretch of the muscle is required, as opposed to solely nerve stimulation of the muscle (Garrett et al. 1984a). At the time of muscle failure, the forces produced without muscle activation are several times higher than the maximum isometric force produced by the activated muscles (Garrett et al. 1988). In turn, the passive forces within the muscle may play as much of a role as the active forces that occur in the muscle during muscle strain injury (Garrett et al. 1988).

The passive components of a stretched muscle have the ability to absorb energy, but that potential is greatly increased by active contraction of the muscle (Garrett 1990). This may provide an explanation as to why muscles can be injured when they are incapable of withstanding strain (Garrett 1990). A muscle's ability to withstand strain is a measure of the energy absorbed by the muscle prior to failure (Garrett 1990). Strain energy is represented as the area underneath the curve as it relates stress to strain (Garrett 1990). The two components that compose a muscle's ability to absorb energy include the passive component which is not dependent on the muscle activation and is a property of the connective tissue elements within the muscle, including muscle fiber and the connective tissue in the cell surface and existing between the fibers (Garrett 1990). There is an additional ability to absorb energy based on the contractile ability of the muscle (Garrett 1990). The active muscle components can double the ability of a muscle to absorb energy; therefore, conditions which diminish the ability of a muscle to contract might also diminish the ability of the muscle to absorb energy (Garrett 1990). Muscle fatigue and weakness are considered predisposing factors for muscle injury; this implies that the active ability to absorb the energy is diminished (Garrett 1990).

The ability of a muscle to absorb energy can protect the bone and joint, as well as the muscle itself (Radin et al. 1979). When the muscle is under a low strain, the energy absorption is due to the active component as opposed to the passive component (Radin et al. 1979).

5.4 Injury

A muscle strain is the response of a muscle following forceful stretching in an active or passive manner (Garrett 1990). Muscle strains may be partial or complete (Garrett 1990). These injuries are typically acute and usually a painful event, which is recognized by the patient as the event of injury (Garrett 1990). Two types of muscle strain injuries are possible, direct injury and indirect injury (Garrett 1990). Direct muscle strain injury is characterized by a contusion, which in turn causes an injury based on direct contact to a portion of the muscle (Garrett 1990). An indirect injury is typically at or near the muscle-tendon junction or the tendon-bone junction (Garrett 1990). A complete tear indicates that the muscle is asymmetric when compared to the non-injured contralateral side when the patient is at rest (Garrett 1990). When a complete tear occurs, typically when the muscle is contracted, there is a bulge at the muscle-tendon junction where the muscle is still attached to the bone (Garrett 1990).

Muscle strain injury occurs in two ways, a stretch of the muscle to complete rupture or, more commonly, an incomplete injury in which there is not a complete separate of the tissue to the level of complete tear (Garrett 1990). A strain injury tends to result in bleeding; this bleeding may occur immediately post-injury, but there is a possibility of experiencing a delay prior to the detection of the subcutaneous ecchymosis of one or more days (Garrett 1990). This bleeding is not confined to the muscle proper; the bleeding also escapes through the perimysium and the fascia into the subcutaneous space (Garrett 1990). Computed tomography has confirmed that within the muscle tissue there is also an inflammatory or edematous response that occurs in response to muscle strain injury (Garrett et al. 1989). Ultrasonography of strain injury has demonstrated that bleeding and hematoma can collect between the muscle tissue and the surrounding fascial compartment as a result of the injury (Fornage et al. 1983).

McMaster, in 1993, completed one of the first studies describing injuries at the muscle-tendon junction (McMaster 1933). When there is a normally occurring stretch in the muscle-tendon junction, disruption of the tendon is not present (McMaster 1933). More likely than injuries to the muscle-tendon unit are injuries at the bone-tendon junction, the muscle-tendon junction, and the muscle substance tears (McMaster 1933). Immediate changes are demonstrated in the muscle-tendon junction following a controlled passive strain injury limited to the plastic region of the deformation curve; there is also a limited rupture of the most distal muscle fibers along with hemorrhage (Nikolaou et al. 1987). This is in agreement with previous studies examining the failure properties of the muscle-tendon units under passive extension, demonstrating that the muscle-tendon junction is the site of rupture and therefore the weakest point in the muscle-tendon unit (Nikolaou et al. 1986).

When failure occurs, the muscle fibers near the muscle-tendon junction fail as opposed to a clear separation of the tendon from the muscle fibers; in other words, a small amount of muscle tissue remains attached to the tendon (Garrett et al. 1988). When passive extension is utilized to create muscle strain injuries, those muscle-tendon systems that are deemed normal do not rupture within the tendon (Garrett et al. 1988). The passive extension leads to rupture at the tendon insertion, an avulsion of the muscle organ, a rupture in the muscle belly, a separation of the muscle-tendon junction, or a fracture of the bone (Garrett et al. 1988). During passive extension, the weakest point in the muscle-tendon unit has been identified as the muscle-tendon junction (Garrett et al. 1988). The distal end of the muscle-tendon junction, more specifically, has been identified as the weakest point in the muscle-tendon unit (Garrett et al. 1988). The muscle architecture plays an important role in the failure point, when the muscle is stretched (Garrett et al. 1988). A majority of muscles, regardless of type, fail at the

muscle-tendon junction, more specifically at the distal muscle-tendon junction (Garrett et al. 1988). This does not hold true for the gastrocnemius, which is a multipennate muscle (Garrett et al. 1988). The multipennate architecture results in an arrangement of the muscle-tendon junction which is more complicated than other arrangements (Garrett et al. 1988). This architecture diminishes the ability to categorize the distal and proximal tears of the muscle-tendon junction, like can be done with more simplistic muscle architecture types (Garrett et al. 1988). For example, in experiments, 55.5% of gastrocnemius muscles stretched into passive extension have a clear failure point at the distal muscle-tendon junction (Garrett et al. 1988). The remaining percentage of failures occurs between the muscle-tendon junctions, separating the two heads of the gastrocnemius or within each gastrocnemius head along the deep tendon expansions (Garrett et al. 1988). Although these remaining failures cannot be categorized into distal or proximal muscle-tendon junction failures, they still follow a similar pattern of failure when compared to the other muscles tested and still occurred in the muscle-tendon junction (Garrett et al. 1988). Regardless of muscle architecture, when muscles are passively extended, no failures occur mid-belly of the muscle fibers, within the tendon, or at the origin or insertion of the tendon to the bone (Garrett et al. 1988). Ruptures of the muscle-tendon junction do not cleanly separate the tendon from the muscle; during passive extension, when failure occurs, the tendon which is avulsed carries approximately 0.5 mm of the muscle fibers away with it (Garrett et al. 1988).

Rate of strain is necessary to create a strain injury in human muscle and alters where the failure will occur (Garrett et al. 1988). A range of rates is present because of the influence of internal factors including muscle type, muscle architecture, physiologic conditioning, and strength (Garrett et al. 1988). Rate of strain may also be influenced by external factors including warm-up prior to vigorous activity and fatigue (Garrett et al. 1988). During passive extension, regardless of the rate of strain, the point of failure is always the muscle-tendon junction (Garrett et al. 1988). Studies examining passive stretch have demonstrated that disruption within the muscle-tendon junction is predictable within a range of strain rates (Garrett et al. 1984a). This is true for all muscle types during passive stretch; regardless of architectural features and direction of strain, the injury still occurs at the muscle-tendon junction (Garrett et al. 1984a). A stretch of the muscle from either the proximal or distal tendon, without preconditioning or muscle activation, still fails and demonstrates disruption on or near the muscle-tendon junction (Garrett et al. 1984a). The biomechanical response of the muscle when the stretch occurs may be related to the muscle fiber length (Wikiewicz et al. 1983). Muscle strain injury does not occur after a relatively constant muscle fiber strain (Huxley and Peachey 1961). The ends of the muscle fibers near the muscle-tendon junction do not strain as much as the fibers that are in the more central area of the muscle (Huxley and Peachey 1961).

Decreases in contractile ability, which has been measured as maximal isometric tension, come before the decrement of tensile stretch in muscles which undergo strain injury (Noonan et al. 1994). This suggests that structural changes in the muscle affect primarily the contractile apparatus, leaving the connective tissue framework of the muscle essentially unaffected (Noonan et al. 1994). This would cause a decrease in the contractile ability without altering tensile parameters (Noonan et al. 1994). In contrast, it is possible that connective tissue damage does occur before or simultaneously with contractile tissue injury, because the conclusion of unaltered tensile properties is based on the idea that failure properties do not change (Noonan et al. 1994). Functional impairment may occur without a concurrent decrease in tensile strength, which may be clinically important in consideration of muscle strain injury (Noonan et al. 1994). There is a general sense that muscles are injured during powerful eccentric muscle activation (Noonan et al. 1994). During these eccentric contractions, the muscle absorbs energy and continues to function throughout the contraction to aid in prevention of injury (Noonan et al. 1994). If the muscle's contractile ability is impaired, its ability to absorb

energy is impaired; this would put the muscle as significant risk of new injury or reinjury (Noonan et al. 1994). In turn, functional impairment even in the face of preserved tensile strength may lead to an increased risk for subsequent more severe muscle injury (Noonan et al. 1994).

In a study by Nikolaou et al., the passive stretch injury in a rabbit's tibialis anterior muscle was stretched to a force equal to 130% of their body weight, which is approximately 80% of the ultimate rupture force of the muscle-tendon unit at a rate of 10 cm/min (Nikolaou et al. 1987). The maximum force generation from a tetanic contraction in the tibialis anterior muscle was 70.5% of the control muscle immediately after the injury; by 24 h post-injury, the tetanic contraction was 51.1% of the control (Nikolaou et al. 1987). By 48 h post-injury, the contraction was 74.5% of the control muscle, and at 7 days, the contraction was 92.5% of the control muscle (Nikolaou et al. 1987). Histology studies showed that there were limited distal fiber rupture and hemorrhage immediately following the muscle strain injury, but within 24 h, there were fiber necrosis, inflammation, and edema (Nikolaou et al. 1987). Forty-eight hours post-injury, there were complete fiber breakdown and more intense inflammation, followed by collagen fibrosis 7 days post muscle strain injury (Nikolaou et al. 1987). In a similar study on rabbit extensor digitorum longus that were stretched at 10 cm/min to deformation, the contractile ability of the muscle was dramatically decreased when tested immediately after the muscle strain injury (Taylor et al. 1986). The muscle strain injury caused a statistically significant decrease reported in the total deformation and the load to failure (Taylor et al. 1986).

Clinical findings have indicated that muscle injuries occur most often during powerful eccentric contractions (Glick 1980; Peterson and Renstrom 1986; Zarins and Ciullo 1983). This has been evaluated in the research setting while stretching muscles to failure under three conditioning of motor nerve activation (Garrett et al. 1987). The conditions tested included tetanically stimulated, submaximally stimulated, and unstimulated (Garrett et al. 1987). In the groups that failed and a muscle strain injury was produced, only 15% higher forces were generated in the stimulated muscles (Garrett et al. 1987). Between all of the groups, the location of failure, which was at or near the muscle-tendon junction, did not change (Garrett et al. 1987). In contrast, muscles that were stretched to failure while activated had energy absorptions rates that were about 100% greater (Garrett et al. 1987).

Eccentric contractions, which stretch or lengthen the muscle, have the potential to produce contractions of much higher force when compared to other contraction types, including when the muscle stays at a constant length (isometric contraction) and when the muscle is allowed to shorten (concentric contraction), which shortens the muscle (Garrett 1990). Therefore, the development of excessive force within the muscle-tendon unit is increased (Stauber 1989). During eccentric contraction, the passive elements of the muscle, which include the connective tissue, provide a low level of force throughout the contraction (Elftman 1966). In order for these passive elements to provide resistance, there must be enough stretch applied to the muscle; prior to the stretch or prior to this threshold of stretch, there is no resistance or very little resistance provided by these passive elements (Elftman 1966). In the muscle-tendon unit, passive forces do provide some limit to range of motion (Garrett 1990). The role of passive forces means that there is an association between excess strain, which causes muscle strain injury, and eccentric muscle contraction, which by definition is associated with muscle stretch (Garrett 1990).

5.5 Recovery

Functional recovery of muscle can be identified through physiological testing which includes maximal force production in response to a nerve stimulation (Nikolaou et al. 1987; Garrett 1990). Following acute injury, muscles produce 70% of their normal force production (Nikolaou et al. 1987). Within 24 h of initial injury, the force production of a muscle declines to about 50% of the normal force production of the muscle; the comparison is the force production of the healthy,

non-injured muscle (Nikolaou et al. 1987). Seven days following the initial injury, the force production of the muscle is greatly improved, with measures up to 90% of the normal force production of the muscle (Nikolaou et al. 1987). This represents recovery of contractile ability, which is relatively rapid (Nikolaou et al. 1987). Even when the contractile ability is initially diminished, it returns to its normal strength within 7 days of the initial acute injury (Nikolaou et al. 1987).

Recreation of a nondisruptive muscle strain injury has been accomplished by stretching an unstimulated muscle and observing the force-displacement relationship (Nikolaou et al. 1987). If the slope is no longer linear and the muscle is considered to have undergone a "plastic" deformation, the resulting alterations are a material structure (Nikolaou et al. 1987). The ability of the muscle to recover has been demonstrated through physiology and histology studies (Nikolaou et al. 1987). These types of injuries can be recreated by stretching the muscle to 80% of the force necessary to disrupt the contralateral muscle (Nikolaou et al. 1987). The histological studies show that injuries nondisruptive to the whole muscle can cause a disruption of a small number of muscle fibers near the muscle-tendon junction (Nikolaou et al. 1987). The fibers do not actually tear at muscle-tendon junction; rather they tear within the fibers a short distance from the tendon; rarely does the tear occur near the middle of the muscle fiber (Nikolaou et al. 1987). When these types of injuries are demonstrated acutely, there is some hemorrhage within the muscle which is a mark of the distribution that has occurred (Nikolaou et al. 1987). Within 1 to 2 days, the inflammatory reaction becomes more pronounced, there is an invasion of inflammatory cells, and the edema is now present (Nikolaou et al. 1987). When the injury reaches day seven, the inflammation reaction is being replaced by an increase in the fibrous tissue near the region of the actual injury; although some of the muscle fibers have begun the regeneration process, normal histology is not restored at this point, and the scar tissue is still present (Nikolaou et al. 1987).

Speed is also a consideration for recovery following muscle strain injury (Nikolaou et al. 1987). There are no differences in force or elongation to failure when examining the speed at which muscles are pulled to failure at speeds of 10 cm/min and 100 cm/min (Nikolaou et al. 1987). Biomechanical studies on rabbits demonstrated that passively strained muscle can recover function within 48 h, in contrast to the histological findings at 48 h, which demonstrate inflammation and active healing (Nikolaou et al. 1987). In patients with acute muscle strain, decreased function is observed at periods of up to 48 h following injury (Nikolaou et al. 1987). This decreased function is not due to the further degradation of contractile function of the injured muscle, but due to the edema and the increased pain caused by the inflammatory nature of the healing process (Nikolaou et al. 1987). The formation of scar and localized fibrosis seen at 7 days may also play an important role in the tendency for these muscle strain injuries to reoccur (Nikolaou et al. 1987).

5.6 Clinical Implications

Muscle strain injury has a significant impact on sport and recreation (Garrett 1990). Sports requiring large amounts of sprints and bursts of speed and/or rapid acceleration have a high likelihood of muscle strain injury (Peterson and Renstrom 1986). These sports include speed athletes, those who participate in American football, basketball, rugby, and soccer (Peterson and Renstrom 1986). In addition to requirements for sport participation, intrinsic factors also influence risk of muscle strain (Garrett et al. 1984b). Generally, the injured muscles are more superficial and cross two or more joints (Garrett et al. 1984b). These muscles also tend to have a relatively high percentage of type II, fast-twitch muscle fiber (Garrett et al. 1984b). Muscles that have a high percentage of fast-twitch, type II muscle fiber require faster contractions within the muscle (Garrett et al. 1984b). Because of this requirement for faster contractions, kinesiology, and the high speeds that the contractions can reach, these

muscles are predisposed to injury during sport participation (Garrett et al. 1984b).

Establishing the effectiveness of commonly used clinical modalities is important for the prevention and treatment of muscle strain injury (Noonan et al. 1994). The effects of commonly utilized modalities such as stretching have been previously evaluated (Noonan et al. 1994). It may be most appropriate to evaluate injury prevention through testing the ability to prevent or minimize a smaller-scale injury instead of a complete rupture injury (Noonan et al. 1994). This would indicate that the injury is closer to the injury threshold, as opposed to being at the extreme end of total rupture and total failure (Noonan et al. 1994). A 30% stretch has been identified as the point in which there is a failure of force within the muscle, translating to a loss of the ability of the muscle to contract following the stretch (Noonan et al. 1994). In contrast, muscle-tendon junctions stretched to 20% of failure force suffered no decrement in contractile ability (Noonan et al. 1994). This suggests that there may be a threshold for passive stretch injury in the muscle-tendon junction (Noonan et al. 1994). This is supported by histologic examinations; fiber disruption and hemorrhage are only found in muscles stretch to 30% of the force failure (Noonan et al. 1994). This information is meaningful because the identification of a threshold may be useful for evaluating the effect of various treatments or routines on passive stretch-induced muscle injury (Noonan et al. 1994). By finding this threshold for injury, there are, perhaps, exact mechanisms by which passive stretch-induced injury occurs can be elucidated (Noonan et al. 1994).

The inclusion of warm-up periods prior to an exercise task has been debated, due to the performance and injury prevention benefits warm-ups may provide (Safran et al. 1988; Asmussen, Boje 1945; DeBruyn-Prevost 1980; Genovely and Stamford 1982; Ingjer and Stromme 1979; Karpovich and Hale 1956). The intention of a warm-up period is to improve performance and reduce risk of injury, although this has not been demonstrated (Asmussen and Boje 1945; DeBruyn-Prevost 1980; Genovely and Stamford 1982; Ingjer and Stromme 1979; Karpovich and Hale 1956; Safran et al. 1988). The warm-up period should include stretching and active muscle contractions, increasing the range of motion of the joints and muscle-tendon units, in addition to increasing the temperature of the muscles and the efficiency of the contracts that the muscles produce (Kulund and Tottossy 1983; Beaulieu 1981; Williford et al. 1986). The protective effect that the warm-up period may have has been attributed to the increased range of motion and reduced stiffness that is a direct result of the increase in muscle temperature (Safran et al. 1988). With a four-degree increase in the temperature of the muscle, the amount of elongation which can occur without subsequent rupture is increased (Strickler et al. 1990). Evidence is available to show that greater force and increased length are needed to tear isometrically preconditioned muscle or muscle that has gone through a warm-up period (Safran et al. 1988). Regardless of the muscle having gone through a warm-up period, the site of failure is always the muscle-tendon junction (Safran et al. 1988). Muscles which have not gone through a warm-up period appear to be inelastic when the length of the muscle is increased (Safran et al. 1988). This is the biomechanical evidence necessary to prove that warm-up periods may reduce the incidence of muscle strain injury (Safran et al. 1988).

Conclusion

This chapter has reviewed the nature of muscle strain injury in animal model and correlated with clinical studies on muscle strain injury. It is known that most indirect muscle injuries occur in response to stretch and usually while the muscles are activated, therefore resisting stretch. Injury locations are not random in the muscle belly, but rather in the muscle-tendon junction. Most of the research presented was studied utilizing specialized equipment requiring incisions of the tendon. Recent advances in technique allow for the creation of injury in a nondisruptive manner that allows for the study of treatment therapies and general recovery. Muscle injuries cause a great deal of time loss and disability. Much remains in our understating of muscle strain injury.

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