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The effects of cyclic stretching on tensile properties of the rabbit's skeletal muscle

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Abstract

Objective. To define the threshold of muscle injury with cyclic passive stretch.
Design. The changes in the load–deformation curve of muscle–tendon unit were monitored
until the failure point by an *in vivo* rabbit model.
Background. Muscle injuries range in severity from a simple strain to complete rupture.
Although strains occur more frequently than complete failures, only a few studies have
investigated the phenomena of these sub-failure injuries. Monitoring of the continuum for
stretch-induced injury allows us to define the threshold of stretch injury.
Methods. Thirty rabbits' triceps surae muscle–tendon unit preparations were used. One of
the pairs (control) was stretched until failure; the other (experimental) was first cyclic
stretched to either 12, 20 or 25% of the initial length of the muscle–tendon unit and then
stretched to failure. Comparisons were made between the load–deformation curves of the
experimental and control specimens.
Results. When cyclic stretched to 12 or 20%, there were no significant changes existed in
the biomechanical parameters except the deformation at the peak load. In contrast, all the
biomechanical parameters except the ration of the energy absorption changed significantly
after 25% strain cyclic stretch.
Conclusions. A threshold for stretch-induced injury does exist. This can be reproduced at
the 25% strain of the triceps surae muscle–tendon unit.

Relevance

Muscle–tendon injuries, primarily muscle strains or tears, are extremely common in profes-
sional and amateur athletes. This experimental study with rabbits, give evidence that
changes in the biomechanical properties after sub-failure injury does exist in the muscle
tendon unit. More importantly, stretching before competition should not exceed 25% strain
in the triceps surae muscle–tendon unit. © 1998 Elsevier Science Ltd. All rights reserved

Introduction

Muscle–tendon injuries, primarily muscle strains or
tears, are extremely common in professional and

amateur athletes[1,2]. These injuries account for almost
half of all injuries in certain sports[3]. The triceps surae
muscle is one of the most common muscles damaged
in the lower extremities[4]. In the past two decades,
much literature has been devoted to the preven-
tion[2,5], understanding[6], and treatment of these
injuries[7].

During the activities of daily living, the musculo–
skeletal system is subjected to a wide range of joint
motion. Both muscle and tendon components are

susceptible to trauma or wear and tear. It is believed that warming up before an athletic event is important for both performance and injury prevention. In sports medicine, stretching exercise has been recommended to prevent injury[6,8] and to improve performance[9]. Previous experimental studies in animals have shown that passive warming increases the extensibility of the musculo–tendinous unit and that warmed muscle has greater deformation and less stiffness than cold muscle, and offers support to the theory that warming up muscles can aid in injury prevention and improvement in athletic performance[10].

However, intensive exercise training can also result in muscle damage and muscle soreness. There are a large number of biomechanical studies of ligaments or muscles stretched to failure, but only a few studies were done to determine the effect of sub-failure stretch on the elastic behavior and failure properties of the muscle–tendon unit. Our recent experimental studies of indirect muscle strain injury have concentrated on the muscles injured in response to excessive stretch alone[11] or stretch and activation[12]. Previous studies did not determine a threshold or minimal force–displacement necessary to create injury.

Measuring joint range of motion is not practical for the athlete or patient attempting to assess improvement in flexibility, therefore stretch distance is a more useful method of measuring flexibility. In contrast to previous human study using joint range of motion to determine the effects of stretching of flexibility, we measured stretch distance relative to a fixed reference point. The present study investigates a repetitive stretch of muscle–tendon units using the minimal strain necessary to create injury, and thus, define a threshold for injury with cyclic passive stretch. We chose the New Zealand white rabbit for our investigation, because their architectural design, including the soleus in the hindlimb, were considered to be similar to that found in humans[13].

Methods

Thirty New Zealand White rabbits (mean weight, 2.5, SD, 0.2 kg) were divided into three groups. The preparation was the same as previously reported[11]. After subcutaneous ketamine (dosage, 50 mg/kg) general anesthesia, an incision from the mid calf to the plantar surface of the foot was made on the lateral aspect of each hind limb. The Achilles tendon was isolated taking special care to maintain the neurovascular bundle and the tendon insertion intact. For determining the *in situ* muscle–tendon unit (MTU) length, dial calipers (accurate to 0.05 mm) were used to measure the distance between the origin of the triceps surae at the femur and the insertion at calcaneus with the knee and the ankle at 90° angulation. The anesthetized rabbit was placed in a frame attached to a MTS machine (MTS BionixTM 858 test

system). The hind limb was immobilized with a K-wire transfixation through the proximal tibia. The distal tendinous insertion was freed by osteotomization at the calcaneal tuberosity and clamped to the MTS load cell. A 3 N preload was applied on the muscle, and then the muscle length was again measured[11].

The muscle–tendon unit of one hind limb was cyclically loaded for one hour at a rate of 0.5 cm/min to any of three strain amplitudes (12, 20 and 25%). Once the peak stretch amplitude was reached, the stretching was discontinued and the muscle–tendon unit returned to its initial resting length. After cyclic passive stretch, the muscle was stretched at a constant rate (0.5 cm/min) until a macroscopic tear or full division of the ruptured muscle fragments occurred. In the other hind limb, the muscle–tendon unit was stretched at the same rate to failure of the muscle–tendon unit. The load and deformation required to deform the muscles were simultaneously recorded on a PC by the TestlinkTM system Software (PCLABTM Data Translation, Data Translation Inc., Locke Drive, Marlboro, USA). All muscles were kept moist and at physiologic temperature using warm normal saline irrigation. Additional anesthesia was given as needed. This study received prior approval of the National Taiwan University Medical College's Animal Research Committee. After completion of the experiments, the rabbits were sacrificed at the conclusion of the study.

For each triceps surae muscle, the load and deformation of the muscle–tendon unit were recorded and plotted directly using a PC. Deformation of the muscle–tendon unit was measured when peak load was evident. The deformation of the muscle–tendon unit was calculated by muscle length at peak load minus muscle length before distraction. The area under the load–deformation curve before the failure point represents the relative energy absorbed by the muscle–tendon unit prior to failure. The difference of the energy absorbed by the muscle–tendon unit before the point of peak load and the point of full separation of the ruptured fragments as well as the differences between the two limbs were evaluated by the paired *t*-test. Because of the great individual variation of the triceps surae muscle strength, only the statistic method of the paired *t*-test was used to evaluate the difference between the two limbs of the rabbits in each group. The level of statistical significance was set at 5%.

Results

All of the triceps surae muscle–tendon units under distraction had a similar curve pattern, as shown previously[11]. The load–deformation curve began with an initial increasing slope and ultimately reached the peak load. After the point of peak force, there is a

steep drop followed by a curve with gradual increasing and decreasing of the forces. The slopes of these curves were measured at every linear portion of the curve. The means of peak tensile force, displacement at peak tensile force and slope are summarized in Table 1.

After cyclically stretched at either 12 or 20% strain, the shape of the load–deformation curve of triceps surae muscle–tendon unit did not show any significant change [Figure 1(A)]. When the muscle–tendon unit was cyclically stretched after 25% strain, a statistically significant difference in the biomechanical parameters of the triceps surae muscle–tendon unit between the control and experimentally cyclic stretched groups can be noted [Figure 1(B)]. In the group of 12 and 20% cyclic stretch, all biomechanical parameters between the control and experimental cyclic stretched muscle–tendon unit showed no significant difference except in deformation at the peak load (Tables 1 and 2). In the group of 25% cyclic stretch, all biomechanical parameters between the control and experimental cyclic stretched muscle–tendon unit showed a statistically significant difference, except for the ratio of energy absorption (Tables 1 and 2). After 60 min cyclic stretch to 25%, the peak load decreased 10.3%, deformation at peak load decreased 19.9% and slope of the load–deformation increased 27.9% (Table 1).

The energy absorption before complete separation of the disrupted fragments of the muscle–tendon unit was expressed by the area beneath the load–deformation curve. The means of total energy absorption, energy absorption before peak tensile force and the ratio of energy absorption before peak tensile force are shown in the Table 2. In the rabbits group after either 12 or 20% cyclic stretch, the means of the total energy absorption and the energy absorption before peak load remained constant; while after 25% cyclic stretch, the means of the total energy absorption and the energy absorption before peak load decreased significantly. The differences were statistically significant ($P = 0.0025$ and 0.0017 respectively). No statistically significant difference appeared between the ratio of energy absorption in both groups ($P > 0.05$). The total energy absorption before muscle–tendon

unit failure decreased 33.1%; the energy absorption before peak load decreased 35.7% (Table 2).

The sites of failure were within 0.1–1.0 mm from the distal musculo–tendinous junction for soleus muscle and within 5–10 mm from the distal musculo–tendinous junction in the lateral head of gastrocnemius muscle. While in the medial head of gastrocnemius muscle, failure occurred within 15–30 mm from the distal musculo–tendinous junction as previous reported[11].

Discussion

Musculo–tendinous strain injuries have been cited as the most common injury in competitive athletics[1,9,14]. Their frequency and disabling effects have been documented in many epidemiologic studies[15-18]. Strains not only cause a significant loss of time from sports, but are also a common source of pain, and impair performance following return to competition. Despite their common occurrence, there have been relatively few studies investigating the effects of these injuries. Possibly as a result of the incomplete study of these injuries, treatment is extremely variable, ranging from complete rest and immobilization of the injured muscle to immediate return to athletic competition, sometimes after local injections into the injury site[19]. Previous studies involving stretching injury have used large total displacements (beyond the muscle's physiologic range of motion) to create injury[11,20]. These studies did not determine a threshold or minimal force–displacement necessitated to create injury. Muscle injuries can range in severity from a simple strain to a complete rupture. However, strain or sub-failure comprise 80% or more of all muscle injuries and are much more frequent than complete failures. Only a few studies have investigated the phenomena of these sub-failure injuries[21]. Despite this prevalence, there is a paucity of information on the mechanics of muscle strains. The present study has investigated a repetitive stretch of muscle–tendon units using the minimal strain necessary to create injury, and thus, define a threshold for injury with passive stretch. The hypothesis of this work was that sub-failure injury

Table 1. Biomechanical data of the slope, peak tensile load and deformation at peak tensile load of the composite triceps surae muscle–tendon unit ($N = 10$)

		12%	20%	25%
Peak load (N)	Control	482.2 (SD, 68.0)	463.5 (SD, 58.4)	447.4 (SD, 56.9)
	Study	485.0 (SD, 63.8)	459.7 (SD, 69.6)	401.4 (SD, 90.2)
	P value	0.8290	0.8627	0.0256
Deformation at	Control	42.1 (SD, 5.3)	40.1 (SD, 4.6)	43.7 (SD, 5.8)
Peak load (mm)	Study	38.5 (SD, 5.7)	36.6 (SD, 4.1)	35.0 (SD, 6.7)
	P value	0.0098	0.0058	0.0055
Slope (N/mm)	Control	19.9 (SD, 1.6)	18.1 (SD, 2.5)	17.9 (SD, 3.7)
	Study	20.1 (SD, 2.0)	19.7 (SD, 2.8)	22.9 (SD, 4.0)
	P value	0.7249	0.2677	0.0010

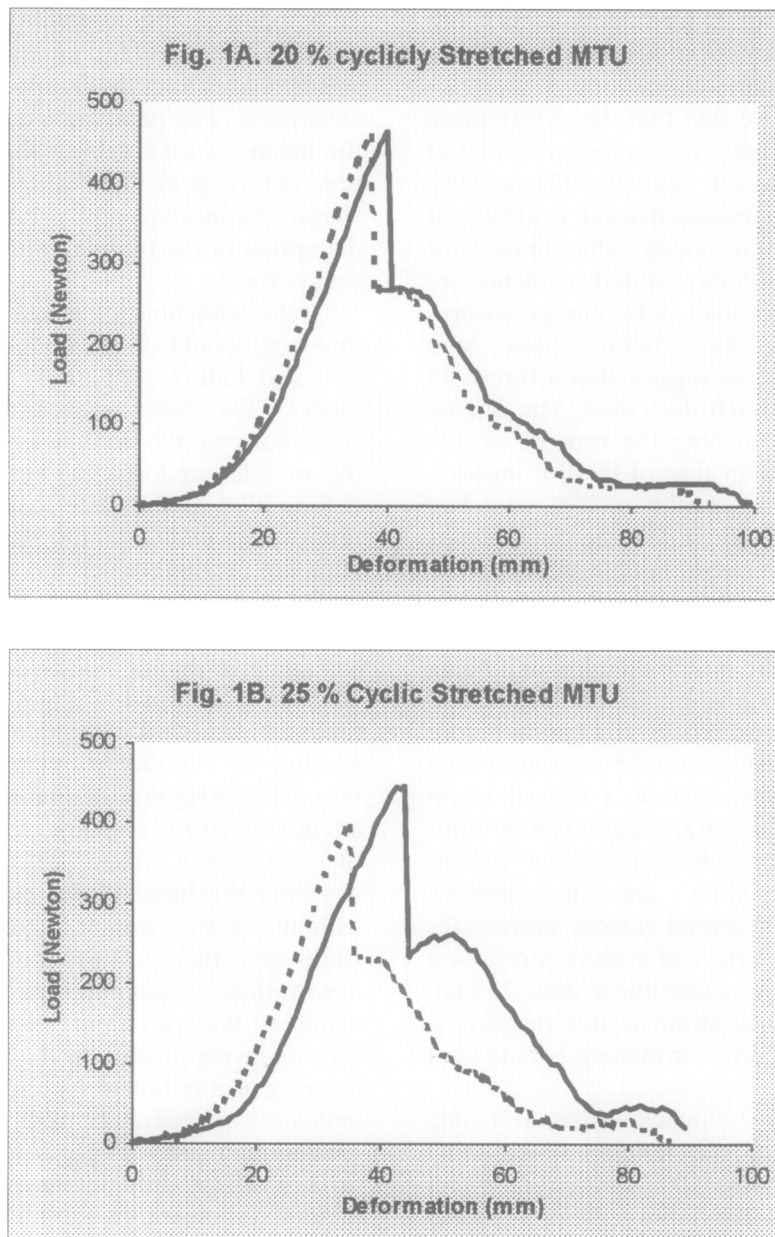Data are presented as mean (SD = standard deviation).

Figure 1. Load–deformation curve of triceps surae muscle-tendon unit after 20 or 25% cyclic stretch. The area below depicts the relative energy absorbed to failure under various conditions: (———) control; (– – –) experimentally cyclic stretched to 20 or 25%.(A) After cyclically stretching to 20% strain, the shape of the load–deformation curve of triceps surae muscle-tendon unit showed no significant change. (B) When the muscle-tendon unit was cyclically stretched at 25% strain, the peak load, deformation at peak load, total energy absorption and energy absorption before peak load all significantly decreased, while the slope of the load-deformation increased significantly.

Table 2. Energy absorption of composite triceps surae muscle–tendon unit ($N = 10$)

		12%	20%	25%
Total energy	Control	13068.2 (SD, 3056.0)	12245.6 (SD, 4202.0)	12797.0 (SD, 2879.4)
Absorbed (N/mm)	Study	12992.0 (SD, 3513.8)	10494.1 (SD, 2505.8)	8564.6 (SD, 3610.1)
	P value	0.9002	0.1754	0.0025
Energy absorbed	Control	7343.3 (SD, 2138.6)	6761.3 (SD, 1659.2)	7102.0 (SD, 2269.5)
Before peak	Study	6934.3 (SD, 1670.1)	5937.9 (SD, 1525.4)	4566.9 (SD, 2063.3)
Load (N/mm)	P value	0.4730	0.1339	0.0017
Ratio of energy	Control	56.0 (SD, 7.9)	57.3 (SD, 11.4)	55.1 (SD, 10.0)
Absorption	Study	54.2 (SD, 6.5)	57.3 (SD, 10.9)	54.1 (SD, 10.4)
(%)	P value	0.5913	0.9945	0.7812

Data are presented as mean (SD = standard deviation).

initiates within the linear region of the load deformation curve and that the sub-failure injury initial does not alter the load–deformation behavior above the level of the sub-failure injury.

As shown in Table 1, we find that the deformation at the peak load diminished after cyclic stretching at all ranges of strain. However, both the 12 and 20% groups demonstrated no biomechanical evidence of injury in muscle–tendon unit injury, while muscles of the 25% strain group demonstrated evidence of injury by diminished peak load, total energy absorption, and energy absorption before peak load (Tables 1 and 2). These data suggest that a threshold for injury with passive stretch does exist. The finding that after 25% cyclic stretching the muscle–tendon unit became more stiff than that of control muscle–tendon unit supports the possibility of 25% stretched muscle being injured. This difference in stiffness implies that, for a given increase in deformation, 25% cyclic stretched muscle develops more force when compared with 12% or 20% stretched muscle (Figure 1). Further evidence of the muscle–tendon unit being injured is obvious by observing that the peak load, total energy absorption, and energy absorption before peak load were all diminished after 25% cyclic stretch (Tables 1 and 2). It assumes that a critical strain needs to be reached before strain injury can be initiated. Before exercise, stretching has been recommended to prevent injury and to improve performance[6,8,9]. The widespread clinical impressions regarding the protective effect of passive stretch on muscles[6] are certainly not against these data. In fact, it is clear that if a critical strain is not reached, a detrimental effect of cyclic stretching would not appear.

Recently, Panjabi et al.[21] elucidated that stretching the ligament to 80% of the failure subsequently increased deformations below 80%, but had no effect on any mechanical properties above 80%, including the peak load and deformation. This may result in increased joint laxity, additional loads will be applied to other joint structures and to the joint. In this study, we demonstrated that when the muscle–tendon unit was cyclically stretched, a statistically significant decrease in the deformation at the peak load of the triceps surae muscle–tendon unit was noted, even though all other biomechanical parameters showed no significant change (Tables 1 and 2). The anesthetic used in this study was ketamine and this drug has no muscle-relaxant effect[22]. The nerve function and related muscle tone were well preserved[23]. This fact suggests that the presence of nerve function can respond to the cyclic stretch by increasing muscle tone and then the deformation at the peak load is decreased. This suggestion is further consolidated by the fact that after 25% cyclic stretch, the slope of the load–deformation curve in the muscle–tendon unit is significantly increased before the peak deformation.

After 25% cyclic stretch, all biomechanical parameters between the control and experimental cyclic stretched muscle–tendon unit showed a statistically significant difference except in the ratio of energy absorption. The peak load, deformation at peak load, the means of total energy absorption, energy absorption before peak load decreased (Table 1 and 2). These biomechanical parameters indicated that disruption in the muscle–tendon unit has occurred to some extent.

Cyclic stretching of muscle–tendon units above a threshold would drastically alter both load–deformation and failure properties. Using an in vivo rabbit model, we have demonstrated that the 20% sub-failure stretch does not alter the failure point of the muscle–tendon unit, but significantly alters the deformation at the peak load, possibly by the nerve reflex. Above 25% strain, the biomechanical parameters are all significantly changed. The fact that there is significant fiber damage without gross changes in the mechanical behavior below the sub-failure point has some intriguing consequences. The most prominent morphological changes in the injured muscle fibers were the loss of desmin staining occurred in the absence of contractile or metabolic protein disruption. The disruption of the cytoskeletal network and an inflammatory response could further deteriorate the contractile response[24]. Clinically, it is well observed that mild strains can cause significant pain without altering muscle activity. The results of this study are, then, in general agreement with those observations, assuming pain is associated with damage. We postulate that fiber failure without accompanying change in the gross load–deformation behavior is a potential mechanism for stimulating the biological maintenance and repair of the muscle–tendon unit. This suggests that this model can be further validated by documenting a biological/inflammatory response to a sub-failure injury, which would not notably alter the load–deformation behavior.

In summary, this study shows that a threshold and continuum for stretch-induced injury does exist. It is possible that the muscle fiber disruption occurred initially and connective tissue disruption occurred only with larger muscle displacements[25]. More importantly, in our model, injury can be reproduced at 25% strain of the triceps surae muscle–tendon unit.

Acknowledgements

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