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The role of warmup in muscular injury prevention*

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ABSTRACT

This study is an attempt to provide biomechanical support for the athletic practice of warming up prior to an exercise task to reduce the incidence of injury. Tears in isometrically preconditioned (stimulated before stretching) muscle were compared to tears in control (nonstimulated) muscle by examining four parameters: 1) force and 2) change of length required to tear the muscle, 3) site of failure, and 4) length-tension deformation. The tibialis anterior (TA), the extensor digitorum longus (EDL), and flexor digitorum longus (EDL) muscles from both hindlimbs of rabbits comprised our experimental model.

Isometrically preconditioned TA ($P < 0.001$), EDL ($P < 0.005$), and FDL ($P < 0.01$) muscles required more force to fail than their contralateral controls. Preconditioned TA ($P < 0.05$), EDL ($P < 0.001$), and FDL ($P < 0.01$) muscles also stretched to a greater length from rest before failing than their nonpreconditioned controls. The site of failure in all of the muscles was the musculotendinous junction; thus, the site of failure was not altered by condition. The length-tension deformation curves for all three muscle types showed that in every case the preconditioned muscles attained a lesser force at each given increase in length before failure, showing a relative increase in elasticity, although only the EDL showed a statistically significant difference. From our data, it may be inferred that physiologic warming (isometric preconditioning) is of benefit in preventing muscular injury by increasing the and length to failure and elasticity of the muscle-tendon unit.

Musculoskeletal injuries, primarily muscle strains and tears, account for almost half of all injuries in certain sports. Their frequency and disabling potential have been documented in epidemiologic studies of many sports: football, basketball, hockey, soccer, lacrosse, squash, gymnastics, rugby, and track and field. Strains not only result in significant loss of time from sports and other activities, but are also a frequent source of pain and impaired performance following return to competition.

With the intention of improving performance and reducing the incidence of injuries, athletes commonly “warm up” prior to an exercise task. The performance benefits have been widely debated. There is widespread opinion among athletes, coaches, trainers, and physicians that this warmup reduces the risk of injury although this has not been demonstrated conclusively.

The warm-up period often consists of both stretching exercises and a period of active muscle contraction or exercise. A warm-up period should increase the range of motion of the joints and muscle-tendon units as well as increase the muscle temperature and the efficiency of the muscle contractions. Various authors attribute the apparent protective effect of warmup to the increase in the range of motion or to the reduced stiffness resulting from the increase in muscle temperature.

We are unaware of experimental data showing the effects of a warmup or preconditioning period on the behavior of muscle-tendon units. Our laboratory has previously shown that muscle-tendon units undergoing repetitive passive stretch do show relaxation of the tension in the muscle-tendon units. The purpose of this study is to test the effects of muscle activation without stretch on the behavior of muscle-tendon units. We have examined four biomechanical parameters: the force to failure, the amount of stretch required to tear a muscle, the site of failure, and the length-tension relationship. We are specifically interested in determining whether there is a protective effect of a warming up or preconditioning period without applied stretch.

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MATERIALS AND METHODS

In 10 New Zealand White rabbits (average weight, 5 to 6 pounds), three muscles from each hind leg were tested (N = 60). After intramuscular administration of an anesthetic mixture of Ketamine, 100 mg/kg (Bristol Laboratories, Syracuse, NY), Xylazine, 12.5 mg/kg (Miles Laboratories, Shawnee, KS), and Acepromazine, 3 mg/kg (Aveco Company, Fort Dodge, IA), the hindlimbs were shaved. An incision was made on the ventral surface of the hindlimb 3 to 4 cm proximal to the knee and extending to the foot. The TA, EDL, and FDL muscles were isolated, maintaining their neurovascular supply and tendon insertions. Using Vernier calipers, the TA muscle length was measured from its origin to the first cruciate ligament with the foot plantar flexed, the EDL was measured from its origin to the second cruciate ligament with the knee extended and foot plantar flexed, and the FDL was measured from its origin to the calcaneus with the foot dorsiflexed to 90°. The muscles were kept moist and at physiologic temperatures throughout the experiment using warmed normal saline irrigation. Further anesthesia was administered as needed.

The hindlimb was immobilized with K-wires through the tibia and femur in a frame attached to an Instron Universal Testing Instrument (Instron Corporation, Canton, MA). The distal tendon was freed from its insertion and clamped to the crosshead of the Instron with a 100 gram tensile preload. The motor nerve to each muscle was stimulated with a Grass S44 stimulator (Grass Instruments, Quincy, MA) to determine the threshold (T), defined as the smallest voltage necessary to produce a measurable tension increase in the muscle. Each muscle was then stimulated to its maximal wave-summated tension at a frequency of 20 Hertz, 0.5 ms duration, 0.5 ms pulse, and a voltage of 10 T. Stimulation was discontinued at the plateau of the maximal force generated by the muscle. This single stimulation to maximal voltage was defined as isometric preconditioning for the purpose of this study. The nerve and muscle were stimulated for an average of 15 seconds to achieve this preconditioning. The electrode was then moved away from the nerve and the muscle was pulled to failure at a rate of 10cm/min. The contralateral muscle was prepared in a similar fashion and pulled without preconditioning, thus serving as a control.

In an additional four animals, the temperature changes following isometric preconditioning were measured in eight EDLs as follows. The EDL muscles were prepared as above and the distal tendon clamped to the Instron crosshead. Intramuscular temperatures were then recorded in the middle of the muscle belly before stimulation, and at 10, 30, and 60 seconds following stimulation by inserting a hypodermic temperature probe (Yellow Springs Instrument Co., Inc, Yellow Springs, OH; model YSI 524) attached to a YSI Thermometer (model 43TD, Yellow Springs, OH).

Statistical analyses of the maximal force to failure, the increase in length from rest, and the stress-strain deformation were performed using a paired Student's t-test to eliminate interindividual variation. The percentage increase in length to failure was calculated from the sum of the elongation to failure recorded by the chart recorder plus the measured length of the muscle with the preload in the Instron, divided by the rest length with the knee and ankle at 90°.

RESULTS

Force

Figure 1 shows the average force to failure of isometrically preconditioned and unconditioned (control) TA, EDL, and FDL muscles. The average force to tear preconditioned TA muscles was 40.00 ± 3.55 N, whereas nonstimulated control TA muscles required an average 38.45 ± 3.26 N to tear (difference = 1.56 ± 0.29 N; P < 0.001). Thus, an average of 4% more force was required to tear the preconditioned TA muscles. The average force required to cause the preconditioned EDL muscles to fail was 91.32 ± 14.54 N, whereas an average of 84.39 ± 12.01 N was necessary to tear the unstimulated EDL muscles (difference = 6.93 ± 1.76 N; P < 0.005). This is an average of 8% more force necessary to tear preconditioned EDL muscles. The average force needed to tear the prestimulated FDL muscles was 106.67 ± 13.69 N, while the control FDL muscles averaged 97.93 ± 12.37 N (difference = 8.74 ± 2.76 N; P < 0.01). Thus, 9% more force was necessary to tear the preconditioned FDL muscles.

Length

Figure 2 shows the average percent increase in length to tear all three muscles in both states. The isometrically preconditioned TA muscles required an average 27.7 ± 2.6% increase in length from rest before tearing, while the contralateral controls averaged 26.2 ± 2.1% (difference 1.5 ± 0.6%; P < 0.05). The average increase in length from rest of preconditioned EDL muscles was 15.4 ± 1.0%, while control EDL muscles averaged 14.0 ± 1.2% (difference = 1.5 ± 0.3%; P < 0.001). Finally, the prestimulated FDL muscles stretched to 20.3 ± 2.4% over rest length before failure. In comparison, control FDL muscles required an average increase in length of 18.8 ± 3.0% (difference = 1.6 ± 0.6%; P < 0.01).

Site of failure

The site of failure in all preconditioned and control TA and EDL muscles was at the distal musculotendinous junction (MTJ). In nine rabbits all of the preconditioned and control FDL muscles tore at the distal MTJ while in one rabbit the FDL tore at the proximal MTJ in both conditions. Thus, all of the preconditioned muscles tested failed at the same site as their controls.

Length-tension deformation curves

In all cases, the unconditioned muscles attained a greater force at any given increase in length prior to failure, as
Figure 1. Average force to failure for preconditioned and unconditioned TA, EDL, and FDL muscles. All values are means ± SD (N = 60).

shown by the length-tension deformation curve (Fig. 3). However, only the EDL muscles showed statistical significance (at 2.5%, P < 0.01; at 5%, P < 0.006; at 7.5%, P < 0.001; at 10%, P < 0.09; at 12.5%, P < 0.42; at 15%, P < 0.001).

Temperature

Following isometric preconditioning, intramuscular temperatures were seen to rise an average of 1°C within the first 10 seconds, followed by a decrease in temperature thereafter (range, 0.6 to 1.4°C increase).

DISCUSSION

All of the muscles tested, the TA, EDL, and FDL, are composed of predominantly fast twitch (Type II) fibers. Fast twitch fiber predominant muscles have been shown to be the muscles most likely to be injured in humans.27

Our results from this model indicate that a statistically greater force and length of stretch are necessary to tear isometrically preconditioned muscles. There are at least two possible reasons for this phenomenon. One explanation is based on the alteration of the viscoelastic properties of the intramuscular connective tissue as a result of an actual muscle temperature increase with contraction. The second possible explanation is that the isometric contraction caused by nerve stimulation may stretch the connective tissue of the muscle-tendon unit, resulting in a viscoelastic load/stress relaxation. These two possible explanations are not mutually exclusive, and each may play a role.

The main histologic components of muscle tissue are the contractile muscle fibers themselves and a large amount of connective tissue. A connective tissue framework is associated with the muscle cell membranes as the sarcolemma. Connective tissue also surrounds single fibers and groups of fibers as the endomysium and perimysium. There are also connective tissue specializations at the muscle-tendon junctions.69 With large stretches, most of the tension developed in muscle is due to the connective tissue elements. Some of the passive tension in muscle is also due to the contractile proteins in the muscle fibers themselves.69 However, as muscle length increases, more of the tension is due to connective tissue elements outside the muscle fibers.15,31,62,70

Collagen is the principle component of the connective tissue in muscle, and the properties of collagenous tissue have been studied in detail. Collagenous tissues such as tendons and ligaments are generally considered to behave as rigid structures.23,41,66 However, it has been shown that the extensibility of collagenous tissue can be increased by raising the temperature.32,41,59,60,71 LaBan40 showed a 0.75% increase in the length of a stretched tendon with a 2.8°C increase in temperature from 37° and a 1.5% increase in length with a 5.5°C increase in temperature. Warren et al.71 showed an increase in both length and force to failure for...
Figure 2. Average percent increase in length before failure of preconditioned and unconditioned TA, EDL, and FDL muscles. All values are means ± SD (N = 60).

Figure 3. Average length-tension curves for preconditioned and unconditioned EDL muscles. All values are means ± SD (N = 60).

The warming effect of contraction lasts up to one-half hour after the contraction. Therefore, part of the difference in preconditioned muscle may be due to alteration of the biomechanical properties of muscle as a result of the temperature increase associated with contraction. We measured the actual temperature changes using an intramuscular needle temperature probe and found that muscle temperatures rise 0.6 to 1.4°C (average, 1.0°C) with this preconditioning protocol. Although the temperature increase is small, it can be seen from the work of LaBan and Warren et al. that it may indeed have an appreciable effect on the length and tension changes in muscle prior to failure.

In addition to the effects of temperature, a second independent factor to be considered is the effect of viscoelastic stress relaxation. Connective tissue subjected to a sustained constant stress shows elongation with time, and if stretched to an initial tension will show a fall in that tension with time. Similar findings for whole muscle preparations in this laboratory have shown that cyclic stretching to the same length is accompanied by a decrease in muscle tension, especially over the first few cycles.

The protocol for these experiments involved an isometric contraction. Although the total length of the muscle-tendon unit remained constant, there is reason to believe that the series elastic components were subjected to a stretching effect. Nerve stimulation produces contraction of the muscle fibers. As the fibers are activated they begin to shorten and
apply tension and stretch to the tendon and to the muscle-tendon junction (Fig. 4). Although the total length of the muscle-tendon unit remains unchanged, the active muscle fibers shorten slightly and the muscle-tendon junction and the tendon are lengthened slightly, with the two effects being equal and opposite. In this manner it can be seen how isometric contraction can lead to the same stress-relaxation effect as passive stretching. The muscle-tendon junction is subjected to stretch, and it is the location of the actual rupture of the muscle-tendon unit. Therefore, stress relaxation effects in this region may help explain the altered mechanical behavior noted in these experiments as a result of preconditioning.

In the clinical setting, indirect muscle injuries or strains often involve the region of the muscle-tendon junction. This is seen in the biceps brachii, triceps brachii, pectoralis major, gastrocnemius, hamstrings, rectus femoris, adductor longus, iliopsoas, and flexor pollicus longus muscles. We feel that delayed onset muscular soreness may be the beginning of a continuum that progresses to first degree, second degree, and third degree strains with increasing injury. Delayed onset muscular soreness which is thought to be a result of connective tissue breakdown, has been noted through clinical evaluation to be localized primarily at the distal MTJ. One of the few experimental studies of muscle tears was performed in 1933 by McMaster. He showed that normal muscle-tendon units fail at the tendon of origin or insertion, the muscle belly, or the MTJ, but not in the tendon itself. Previous experimental studies on passive muscle tears in physiologic vascular preparations have shown the distal MTJ to be the site of damage in stretching injuries, regardless of rate of strain, architecture of muscle, and end from which the muscle was pulled. Our experimental warmup did not alter the site of muscle failure. The muscles in this study consistently failed at the muscle-tendon junction, with a 0.1 to 1.0 mm remnant of muscle fibers retained on the distal tendon. Why the MTJ is the site of failure is not known.

The length-tension curves of isometrically preconditioned and control muscles (Fig. 3) show that preconditioned muscles follow a deformation curve similar to that of the control muscles, with lesser forces at given lengths, while ultimately failing at greater forces and lengths. Statistically, the control EDL muscle is more inelastic, requiring a greater force at given lengths until nearing failure. At this point it is surpassed in force and length by the preconditioned muscle. The elasticity change in the TA and FDL may not have been statistically significant due to 1) the greater ranges (standard deviations) of lengths required to tear the TA and FDL muscles, leading to variations in the location of the inflection point (toe-in), or 2) the relatively small but statistically significant difference in force to tear the preconditioned and control TA muscles.

It appears that warmup stretches the musculotendinous unit and results in an increased length at a given load, putting less tension on the MTJ and resulting in a reduced incidence of injury to the muscle-tendon junction. These findings have significance with respect to muscle strains and tears seen in clinical practice, lending scientific support to the athletic practice of warming up prior to an exercise task to reduce the incidence of injury.

CONCLUSIONS

This paper presents biomechanical and pathophysiologic data on muscle tears in two simulated physiologic states: “warm” and “cold.” Our results show that a greater force and increase in length are needed to tear isometrically preconditioned (warmed) muscle. The MTJ was the site of failure in all of the muscles and the physiologic state did not alter the site of failure. The unconditioned (cold) muscles appear more inelastic at each increase in length. This study provides a biomechanical explanation of the mechanisms by which warmup may reduce the incidence of musculotendinous injury. An increased understanding of the benefits of muscle warmup is of great importance to athletes, sports medicine physicians, and orthopaedic surgeons in general.

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**DISCUSSION**

Michael J. Smith, MD, St. Petersburg, Florida: In athletic training, it has long been entrenched that a good warmup is essential. Clinically, a warmup improves the range of motion of a joint, increases the heart rate of the athlete, and also increases the metabolic rate. This hopefully reduces the risk for injury and perhaps even enhances athletic performance. This paper gives a scientific credence for the premise of injury prevention with warmup. The authors need to be congratulated for this good undertaking. The experimental protocol was concise, simple, and well-done on 10 New Zealand rabbits. It is a good preliminary study and has potential to give birth to many future studies. I have three
questions for the authors. If extensibility of collagen tissue is increased, by raising the cell temperature, and by internal active means, can the same result be obtained with external, passive modalities, such as moist heat? The authors did monitor the degree of temperature rise and this average was 1°C. My second question is what was the optimal temperature rise? Is the 1°C rise optimal, or would further heating externally possibly improve the function or benefit? And three, what effect does this increased temperature have on athletic performance and function?

Authors' Reply: If the extensibility increases seen with preconditioning are due primarily to the active raising of temperature, then one would hypothesize that passive increases in temperature may also increase extensibility. This is a question we plan to pursue in the future. As far as optimal temperature rise, again we do not know the answer but plan to examine this question further. Lastly, would increased temperature lead to enhanced performance? This question has been debated for a long time and our study does not directly address this point. We would like to know the answer.