

Factors Affecting Force Loss With Prolonged Stretching

David G. Behm, Duane C. Button, and Jeremy C. Butt

Catalog Data

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Mots clés : antagoniste, électromyographi.e., contraction maximale volontaire, activation musculaire, secousse, tétanos

Abstract/Résumé

The purpose of this study was to investigate factors underlying the force loss occurring after prolonged, static, passive stretching. Subjects were tested before and 5–10 min following 20 min of static, passive stretching of the quadriceps (N = 12) or a similar period of no stretch (control, N = 6). Measurements included isometric maximal voluntary contraction (MVC) force, surface integrated electromyographic (iEMG) activity of the quadriceps and hamstrings, evoked contractile properties (twitch and tetanic force), and quadriceps inactivation as measured by the interpolated twitch technique (ITT). Following stretching, there was a significant 12% decrement in MVC with no significant changes in the control group. Muscle inactivation as measured by the ITT and iEMG increased by 2.8% and 20.2%, respectively. While twitch forces significantly decreased 11.7%, there was no change in tetanic force post-stretch. Although possible increases in muscle compliance affected twitch force, a lack of tetanic force change would suggest that post-stretch force decrements are more affected by muscle inactivation than changes in muscle elasticity.

Le but de cette étude est d'analyser les facteurs relatifs à la réduction de la force musculaire après une longue période d'étirement statique et passif. Les sujets sont évalués avant et 5-10 min après un étirement statique et passif du quadriceps (n = 12) d'une durée de 20 min;

The authors are with the School of Physical Education, Recreation, and Athletics, Memorial University of Newfoundland, St. John's Newfoundland Canada A1C 5S7.

la même évaluation est faite 5-10 min après une période d'égale durée sans étirement (contrôle, n = 6). Les variables analysées sont les suivantes : la force de contraction isométrique maximale volontaire (MVC), l'intégration de l'activité myoélectrique de surface (iEMG) du quadriceps et des ischiojambiers, les propriétés contractiles évoquées (tension de secousse et tétanique) et l'inactivation du quadriceps indiquée par la technique de la secousse interpolée (ITT). Après l'étirement, on observe une réduction de 12% de MVC mais pas chez le groupe de contrôle. L'inactivation du muscle indiquée par ITT et par iEMG augmente de 2,8% et 20,2% respectivement. Malgré la diminution significative (11,7%) de la tension de secousse, la tension tétanique après la période d'étirement n'est pas affectée. Même si l'augmentation possible de la compliance musculaire s'est répercutée sur la tension de secousse, l'absence de variation de tension tétanique indique que la réduction de la force consécutive à un étirement est plus affectée par l'inactivation du muscle que par des modifications de l'élasticité du muscle.

Introduction

Stretching is pervasive throughout sport and therapy. It has been reported to increase range of motion (ROM; Safran et al., 1989), prevent injuries (Worrel et al., 1995), and increase performance (Worrel et al., 1995). Recently, there have been reports (Fowles et al., 2000; Kokkonen et al., 1998; Nelson et al., 1998) of decreases in force output after extensive stretching. It would be difficult to imagine that overall athletic performance would be consistently enhanced if, following acute bouts of prolonged stretching, force output was diminished. While the duration of stretching for one muscle group in this study exceeds typical sport applications (Alter, 1996), the body of scientific literature investigating the effects of pre-exercise stretching on force output is still limited in its scope.

Investigations examining the effects of pre-exercise stretching on subsequent performance have reported decreases in 1 repetition maximum (RM) for both knee flexion and extension following stretching of the quadriceps and hamstrings (Kokkonen et al., 1998). Nelson et al. (1998) examined the effects of acute stretching on vertical jump performance and found that after three different stretches of the knee and hip extensors, counter movement jump heights were significantly decreased. Fowles et al. (2000) monitored the time course of plantar flexor strength deficit following 30 min of maximum, passive stretching, finding voluntary strength decreased for up to an hour. While flexibility exercises can improve ROM and may prevent injuries, the causes and scope of force loss associated with extensive stretching should be thoroughly documented.

Some authors have suggested that a portion of the stretch-induced force loss can be attributed to changes in muscle compliance (Kokkonen et al., 1998), based on decreases in twitch force output (Fowles et al., 2000) and tendon tap reflex activity (Rosenbaum and Hennig, 1995). However, there have not been any studies to investigate the effects of prolonged stretching on the evoked force output of a high frequency tetanic contraction. Furthermore, all myoelectric and evoked muscle testing post-stretch has been performed on the triceps surae muscle group (Fowles et al., 2000; Guissard et al., 1988; Rosenbaum and Hennig, 1995). The greater size and differing fibre composition (Johnson et al., 1973) of the quadriceps may result in a muscle specific response.

Therefore, the purpose of this study was to determine the extent and causes of the acute force deficit following a single 20-min bout of prolonged, static, passive stretching of the quadriceps.

Materials and Methods

SUBJECTS

Sixteen subjects originally volunteered for the experiment. Four of the subjects were disqualified since they were unable to activate more than 80% of their quadriceps as measured by the interpolated twitch technique (ITT). The remaining 12 healthy, active, male subjects from the university population (20–43 years, 181.6 cm \pm 14.8, 87.3 kg \pm 15.2) volunteered for the experiment. A control group of 6 subjects from the experimental group repeated the testing procedure over a 20-min period without the stretching intervention approximately a week following the experiment. Subjects were verbally informed of the procedures and read and signed a consent form prior to participation. The study was sanctioned by the Ethics Committee of the School of Physical Education, Recreation, and Athletics at Memorial University of Newfoundland.

EXERCISE PROTOCOL

Subjects performed a 5-min sub-maximal warm-up on a cycle ergometer to increase muscle temperature. Five sets of stretches were then performed for a total duration of 20 min. Each stretch was held for 45 s and followed by a 15-s relaxation period.

The first stretch performed was the standing quadriceps stretch. The subject stood upright with knee flexed, heel raised to buttocks, and pulled the heel toward the buttocks attempting to not over-compress the knee, while extending the hip. The second stretch was the hurdler quadriceps stretch. This stretch was performed while seated on the floor, hips flexed, with one leg extended in front of the body, while the leg to be stretched was abducted, internally rotated, and knee flexed. The quadriceps were stretched when the subject leaned back toward the floor. The third stretch was kneeling hip extensions. This stretch was performed by kneeling on a mat with knees flexed at an angle greater than 90° and then attempting to extend the hips. The position was supported by extended arms behind the body. The final stretch was the assisted quadriceps stretch. The subject would lie on the mat, face down with one hip extended and knee flexed. A partner would place one hand under the subject's knee and the other hand rested above the buttocks, and the partner would passively extend the hip and leg with the knee flexed. All stretches attempted to reach and stress the subjects' range of motion limits.

TESTING

All testing was conducted prior to and 5–10 min following the stretching regime. Twitch contractile properties were tested at the 5-min recovery mark, followed by a random selection of two tetanic and three voluntary contractions from 6–10 min of recovery. One minute rest was allocated between each contraction. For all voluntary and evoked testing, subjects sat on a bench with hips and knees flexed at 90°, their upper leg and hips restrained by two straps. The ankle was inserted into

a padded strap attached by a high-tension wire to a Wheatstone bridge configuration strain gauge (Omega Engineering Inc. LCCA 250). Subjects performed three isometric maximum voluntary contractions (MVC) of the quadriceps during a single leg extension movement (before stretching protocol and 6–10 min after stretching protocol). Force, EMG, and inactivation as measured by the ITT were recorded from the MVC with the greatest force output. All voluntary and evoked torques were detected by the strain gauge, amplified (Biopac Systems Inc. DA 100 and analog to digital converter MP100WSW), and monitored on computer (Sona Phoenix PC). All data were stored on a computer at a sampling rate of 2000 Hz. Data were recorded and analyzed with a commercially designed software program (AcqKnowledge III, Biopac Systems Inc.).

Surface electromyographic (EMG) recording electrodes were placed approximately 3 cm apart over the mid-portion of the rectus femoris and biceps femoris. A ground electrode was secured on the fibular head and tibial shaft. Thorough skin preparation for all electrodes included removal of dead epithelial cells with an abrasive (sand) paper around the designated areas followed by cleansing with an isopropyl alcohol swab. EMG activity was amplified (X 1000), filtered (10–1000 Hz), rectified, monitored and stored on computer. The integrated EMG (iEMG) activity was measured over a 1-s period, 1.5 s after the beginning of the MVC, in order to allow peak forces to be generated.

Bipolar surface stimulating electrodes were secured to the superior and distal portion of the quadriceps. Stimulating electrodes, 4–5 cm in width, were constructed in the laboratory from aluminum foil, paper coated with conduction gel (Aquasonic), and immersed in water. The electrode length was sufficient to wrap the width of the muscle belly. The electrodes were placed in approximately the same position for each subject. Peak twitch torques were evoked with electrodes connected to a high-voltage stimulator (Digitimer Stimulator, Model DS7H+). The amperage (10 mA–1A) and pulse duration (50 μ s) of a 100 volt square wave pulse were progressively increased until a maximum twitch torque was achieved. The average of 3 trials was used to measure twitch amplitude, time to peak twitch torque (TPT), and peak twitch half relaxation time (1/2 RT).

In order to investigate the summated and fused evoked force of the quadriceps, two trains of tetanic stimulations (100 Hz) were administered at the same stimulus intensity as the twitch for a 300 ms duration. Measures were generated from the tetanus with the greatest torque. Greater stimulation durations (> 300 ms) could not be used due to the pain tolerance of the subjects. All stimulation parameters were identical for pre- and post-stretch testing.

The interpolated twitch technique (ITT) was administered as a measure of the extent of muscle inactivation (Behm et al., 1996; Belanger and McComas, 1981). The ITT involved superimposing 2 electrically stimulated doublets with an interpulse interval of 10 ms upon a voluntary contraction. Torque signals were sent through a high gain amplifier with the superimposed force isolated and further amplified by the software computer program (AcqKnowledge III). An interpolation ratio was calculated comparing the amplitudes of the superimposed stimulation with the post-contraction stimulation to estimate the extent of inactivation during a voluntary contraction (interpolated doublet torque / potentiated doublet torque \times 100 = % of muscle inactivation). Three-minute rest periods were provided between all pre-stretch contractions.

STATISTICAL ANALYSIS

Data were analyzed using both a 1-way (12 experimental subjects pre- and post-stretch) and 2-way ANOVA (2×2) with repeated measures. The 2-way ANOVA factors included groups (6 control versus 6 experimental) and testing (pre- and post-stretch). Control and experimental subjects were the same individuals. F ratios were considered significant at $p < .05$. A Bonferroni (Dunn's) procedure test was conducted if significant main effects were present. Descriptive statistics include means \pm standard deviation (SD).

Results

There were no significant changes in any voluntary or evoked measures in the control group, pre- and post-testing.

VOLUNTARY MEASURES

MVC was significantly ($p < .05$) decreased 12.2% (Figure 1) between 6–10 min post-stretching. Muscle inactivation as measured by the ITT significantly ($p < .05$) increased in the experimental group by 2.8% (pre: 5.7% \pm 2.2, post: 8.5% \pm 6.0) following the stretching protocol (Figure 2). There were significant ($p = .02$) and non-significant ($p = .11$) decreases in quadriceps and hamstring iEMG activity, respectively (Figure 3). Post-stretch quadriceps iEMG activity decreased 20.2% while hamstrings iEMG decreased 16.8% from pre-stretch measures.

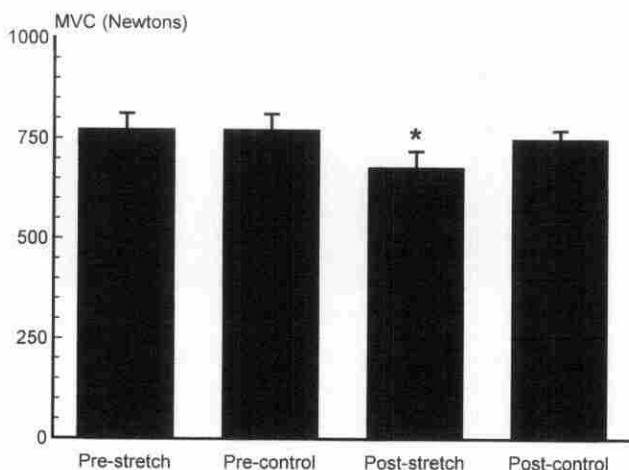


Figure 1. Changes in control and experimental MVC leg extension force (Newtons) prior to and 6–10 min after a 20-min, static, passive stretching protocol. Vertical bars indicate standard error of the means. A single asterisk (*) represent significant differences at the $p < .05$ level.

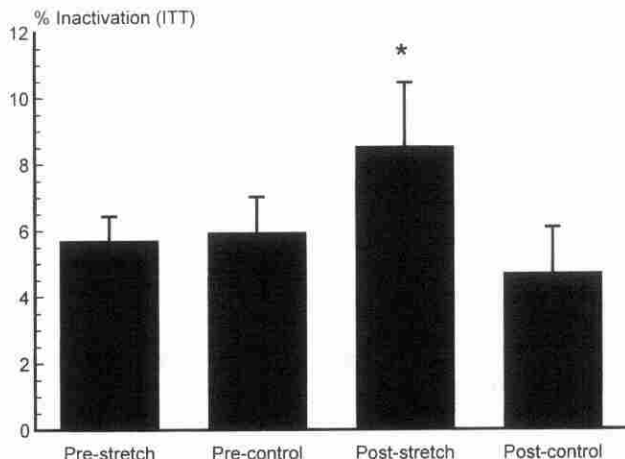


Figure 2. Changes in control and experimental muscle inactivation as measured with the interpolated twitch (IT) ratio prior to and 6–10 min after a 20-min, static, passive, stretching protocol. Vertical bars indicate standard error of the means. A single asterisk (*) represent significant differences at the $p < .05$ level.

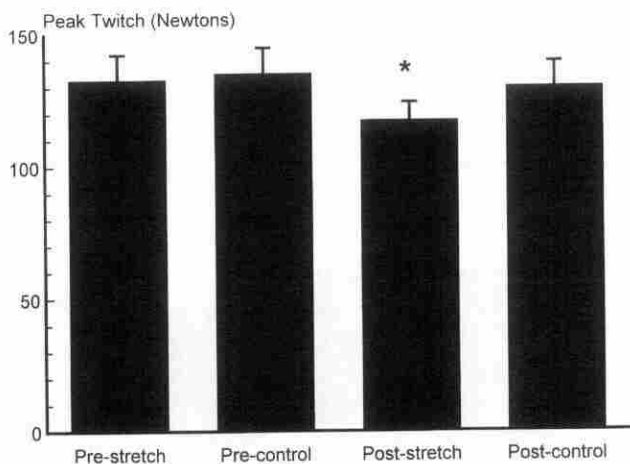


Figure 3. Changes in control and experimental peak twitch force (Newtons) prior to and 5 min after a 20-min, static, passive stretching protocol. Vertical bars indicate standard error of the means. A single asterisk (*) represent significant differences at the $p < .05$ level.

EVOKED MEASURES

Although post-stretch peak twitch force significantly ($p < .05$) decreased 11.7% (Figure 4), prolonged, static, passive stretching did not significantly affect tetanic force (pre: 311.3 N \pm 126.1, post: 309.4 \pm 125.8), TPT (146.0 ms \pm 16.5, post: 144.3 ms \pm 16.4), or 1/2 RT (pre: 49.9 ms \pm 15.2, post: 45.4 \pm 14.5).

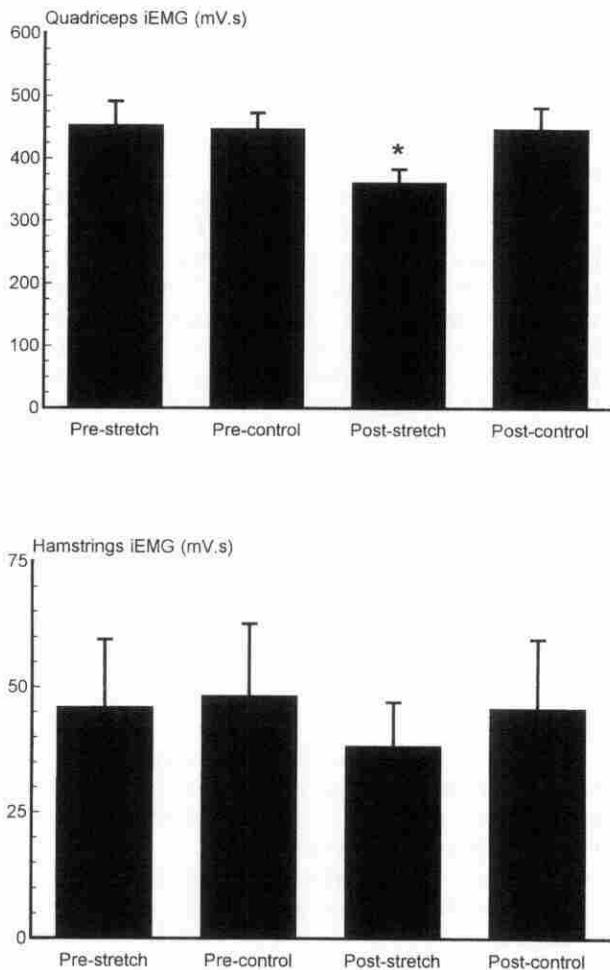


Figure 4. Changes in control and experimental integrated electromyographic (iEMG) activity of the agonist quadriceps (upper graph) and antagonist hamstrings prior to and 6–10 min after a 20-min, static, passive stretching protocol. Vertical bars indicate standard error of the means. A single asterisk (*) represent significant differences at the $p < .05$ level.

Discussion

This study's most important contribution was that the stretch-induced decrease in MVC could be partially attributed to decreases in muscle activation. Both measures of muscle activation (ITT and iEMG) were decreased following the stretching protocol. While muscle inactivation as measured by the ITT increased (or activation decreased) 2.8%, iEMG activity decreased by 20.2%. This disparity in the level of activation may be attributed to the non-linearity of both the interpolated

twitch (IT) ratio—force and EMG—force relationships. It has been reported that the superimposed or interpolated force to voluntary force relationship is not linear, but actually curvilinear (Behm et al., 1996; Belanger et al., 1981; Dowling et al., 1994; Lloyd et al., 1991; Norregard et al., 1994; Rutherford et al., 1986; Siemionow et al., 2000). Similarly, the EMG—force relationship has been reported to be non-linear in a number of muscle groups (quadriceps femoris; Alkner et al., 2000), dorsiflexors, plantar flexors (Genadry et al., 1988), tibialis anterior (Bigland and Lippold, 1954), and a variety of other muscles (Perry and Bekey, 1981). A non-linear relation would not permit an accurate extrapolation of muscle activation from either a single IT ratio or a change in iEMG. Thus, the use of iEMG or single IT ratios may not provide a precise estimate of muscle activation, although they can still be useful as a gross indication of increases or decreases in muscle activation.

Yue et al. (2000) suggest that the conventional ITT overestimates the activation level. This hypothesis is supported by estimates of muscle activation with magnetic resonance techniques that are routinely lower than ITT (Adams et al., 1993). The problem with the prediction may partially arise from the amplitude of the superimposed or interpolated force. Upton et al. (1971) suggested that the evoked force cannot be fully developed due to collisions from the anti-dromic volley of electrical stimulation. Herbert and Gandevia (2000) using a computerized model also found that antidromic collisions as well as spinal reflexes could reduce the amplitude of the interpolated twitch. This reduction was most significant with contractions between 40–80% of MVC but also occurred to a lesser degree with MVC. The decrement in force would artificially lower the amplitude of the superimposed force, falsely indicating that less muscle fibres had been activated by the stimulation. In summary, disparities in the percentage change of muscle activation after stretching may be ascribed to the non-linearity of both measures and the tendency for the ITT to underestimate the extent of muscle inactivation. However, the main message was consistent; extensive static passive stretching results in decreased muscle activation affecting force output.

Decreased activation following prolonged stretching is consistent with other research as well. Fowles et al. (2000) reported a 20% decrease in force 5 min after stretching, which was accompanied by a significant 13% decrease in activation as measured by the ITT and a non-significant 15% decrease in EMG activity. In their discussion, they reviewed a number of factors that could have contributed to the post-stretch inactivation. The extent of autogenic inhibition provided by the Golgi tendon reflex is related to tension development, and thus this inhibitory reflex could contribute to the inexcitability of the motoneurons. However, as pointed out by Fowles et al. (2000), Golgi tendon organ discharge rarely persists during maintained stretch and the inhibitory effects are transitory (Alter, 1996).

Type III (mechanoreceptor) and IV (nociceptor) afferents could contribute as well. Rutherford et al. (1990) reported extensive quadriceps inactivation with muscle pain. However, both deAndrade et al. (1965) and Wood et al. (1988) reported that swelling-induced reflex inhibition of the quadriceps was independent of pain. Behm and St. Pierre (1997a) reported only a 0.1 correlation between pain and inactivation in previously immobilized ankle fracture patients. Stokes and Young (1984) infiltrated human knee joints with bupivacaine to block the pain of post-surgery meniscectomies and reported no change in the severity of inhibition. Again,

Fowles et al. (2000) commented that the discomfort and pressure would be present only during the stretch, with these inhibitory components absent 5–10 min following the stretching protocol, making it unlikely that inhibition induced by mechanoreceptors or nociceptors provided substantial inhibition during the testing period.

Similarly, fatigue-induced inhibition (Behm and St. Pierre, 1997b, 1998) would be an unlikely candidate, since muscle activation was absent during the stretch as evidenced by the lack of increase in iEMG over resting conditions (Figure 5).

In three of the four stretches used in the protocol, the knee was flexed to the limit of the individual's ROM. Knee flexion during a quadriceps stretch would increase intra-articular knee pressure (Eyring and Murray, 1964; Jayson and Dixon, 1970) as well as compress the patella upon the joint. In addition, dislocating torques would be placed upon the tibial portion of the knee joint, by forces pulling or pushing the distal portion of the tibia toward the pelvis. Prolonged stress on the joint receptors could possibly lead to inhibitory effects upon the motoneuron. McComas et al. (1983) demonstrated greater inactivation in patients with joint pathologies. However, Sabbahi et al. (1990) desensitized healthy ankle joint receptors with xylocaine and then observed motoneuron excitability by monitoring H-reflex activity. They found no significant changes in H-reflex activity suggesting

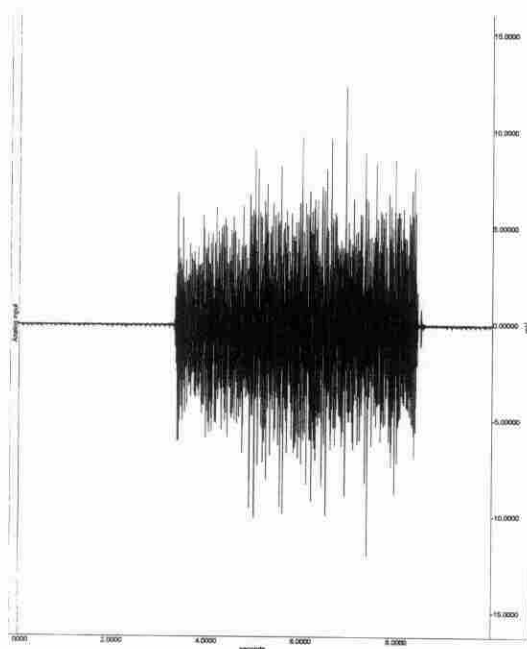


Figure 5. The graph depicts the rectified electromyographic (EMG) activity of the quadriceps for a single subject; while stretching (approximately first 2.5 s), performing a maximal voluntary contraction (MVC; approximately 2.5–8 s) and then at rest (8–10 s).

the joint receptors have minimal inhibitory effects on the excitability of the motoneurons. Similar to other reflex actions, any inhibitory actions would exert their greatest effects during the stretch period with minimal continuance 5–10 min into recovery.

Finally, Fowles et al. (2000) indicated that a transient increase in muscle length due to stretching might negatively impact the excitatory stretch reflex originating from the muscle spindles. However, this excitation is more prevalent during the stretch and recovers immediately after the stretch (Guissard et al., 1988). Thus the origin of the post-stretch inactivation has yet to be established and should provide impetus for further research.

Changes to the visco-elastic properties of the muscle after stretching have been suggested to affect force output. A recent study by Kokkonen et al. (1998) reported a decrease in 1 RM for the knee extensors and flexors after an acute bout of passive stretching of both muscle groups for 20 min. They suggested that the stretching treatment might have influenced maximal strength through a reduction in either the passive or active stiffness of the musculotendinous unit. Rosenbaum and Hennig (1995) investigated the acute effects of prior exercise (warm-up and stretching) on Achilles tendon reflex activity. They found a decrease in the reflexive peak force and myoelectrical activity of the triceps surae. Additionally, they also found the passive peak force caused by a tendon tap to be significantly reduced following the stretching treatment. This increased compliance may relate to the significant 11.7% decrease in peak twitch force as seen in this study. A similar 10–19% decrease in peak twitch torque (post stretch, 15-min recovery) following prolonged stretching was implicated as evidence of impaired muscle contractile force by Fowles et al. (2000). However, an evoked twitch involves an incomplete saturation of the myofilaments with Ca^{++} (Binder-Macleod et al., 1996), resulting in significantly less force than an MVC or a tetanus. The dramatically smaller force of a twitch would be more sensitive to changes in muscle stiffness. Indeed, if increased compliance was a dominant factor in MVC force reduction, then tetanic force should also have been reduced. A non-significant reduction of 1.9 N indicated that the summated contractions of the 300 ms high frequency tetanic stimulation were sufficient to overcome the increased laxity of the musculotendinous unit and provide an efficient transfer of force from muscle to bone. The greater force and duration of the MVC would also be expected to be sufficiently efficient in overcoming the tendon laxity.

However, unlike the Fowles et al. (2000) study, the present study did not measure force output at a variety of angles. Fowles et al. (2000) indicated that the optimal force that was achieved at 10° dorsiflexion pre-stretch was shifted to 20° dorsiflexion post-stretch. Perhaps alterations in tetanic force in the present study could have been discovered at other angles.

Conclusion

The data indicated that a given regimen of prolonged, static, passive stretching can inhibit MVC force and activation of the knee extensors. A loss of maximal force due to the inefficient transfer of force with a more compliant musculotendinous unit was not substantiated at the angle measured, since tetanic force was not diminished.

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