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ACUTE EFFECTS OF ANTAGONIST STRETCHING ON JUMP HEIGHT, TORQUE, AND ELECTROMYOGRAPHY OF AGONIST MUSCULATURE

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Abstract

Although there has been substantial research on the acute effects of static stretching on subsequent force and power development, the outcome after stretching of the antagonist musculature has not been examined. The purpose of this study was to investigate the effects of static stretching of antagonist musculature on multiple strength and power measures. Sixteen trained men were tested for vertical jump height and isokinetic peak torque production during knee extension at 60°.s (SlowKE) and 300°.s (FastKE). Electromyography was recorded for the vastus lateralis and the biceps femoris muscles during isokinetic knee extension. Subjects performed these tests in a randomized counterbalanced order with and without prior stretching of the antagonist musculature. Paired samples t-tests indicated significantly greater torque production during the FastKE when preceded by stretching of the antagonist musculature vs. the nonstretch trial (102.2 vs. 93.5 N.m; p = 0.032). For SlowKE, torque production was not significantly different between the trials (176.7 vs. 162.9 N.m; p = 0.086). Vertical jump height (59.8 vs. 58.6 cm; p = 0.011) and power (8571 vs. 8487 W; p = 0.005) were significantly higher after the stretching trial vs. the nonstretching trial. Electromyography responses were similar between the trials. These results suggest that static stretching of the antagonist hamstrings before high-speed isokinetic knee extension increases the torque production. Furthermore, stretching the hip flexors (emphasis on single-joint hip flexors) and dorsiflexors, the antagonists of the hip extensors and plantarflexors, may enhance jump height and power, although the effect sizes were small.

INTRODUCTION

Stretching has traditionally been a part of thepreexercise and competition warm-up to increasejoint range of motion (1), which potentially reduceinjury risk (21,28) and improve performance (36).In recent years, however, the practice of static stretchingbefore exercise has been questioned. A review conducted byShrier (30) concluded that it is unlikely that preactivity staticstretching prevents injury. There is also a great deal ofevidence indicating that preexercise static stretching has anegative impact on strength and power performances(7,9,16,22,25,26,37).

Despite the vast amount of research that has examined theeffects associated with static stretching of the agonist, we areunaware of the published research investigating the effects ofstretching the antagonist musculature on subsequent expressionof strength and power. Concurrent neural adaptationsto both the agonist and antagonist muscles are important tofacilitate greater torque and power output

(5,17). The netexternal force applied during a movement is proportional tothe force produced by the agonist and inversely proportionalto the force produced by the antagonist muscles (2,14).Therefore, inhibiting the force produced by the antagonistmuscles, via static stretching, may allow the agonist musclesto apply greater external force and power output.

Gains in strength might be accompanied with an increasein neural activity of the agonist and neurological inhibitionof the antagonist (5,17). It has already been postulated thatstretching the agonist before a given movement maydecrease the agonist muscle strength and power perhaps,through decreased neural drive (9,16,33). Conversely,stretching the antagonist may result in its inhibition andreciprocally facilitate increased activity of the agonist, withsubsequent improvements in strength and power–relatedperformance. Therefore, the purpose of this study was toinvestigate the effects of static stretching of the antagonist muscles on peak torque of the knee extensors recorded at60_.s**2**1 (SlowKE) and 300_.s**2**1 (FastKE), and vertical jumpheight (VJheight) and power (VJpower). This study alsosought to determine whether antagonist stretching wouldaffect the neural activity in the agonist and antagonistmusculature.

METHODS

Experimental Approach to the Problem

To determine if stretching the antagonist musculature affectsperformance, subjects were tested for peak knee extensiontorque at 60_.s**2**1 and 300_.s**2**1 and for VJheight. All tests wereperformed with and without preceding the antagoniststretching for each subject. The study used a within-groupdesign, and the treatment was provided in a randomizedorder. Subjects underwent the SlowKE and FastKE on thesame day with or without the stretching treatment; theyreceived the opposite treatment 1–3 days later. The SlowKEand FastKE were performed in a randomized order on thefirst testing day. The opposite order was repeated with theopposite treatment on the second testing day. The VJheightwas tested by itself on 2 separate days with or withoutstretching treatment with 1–3 days between each session.

Subjects

Of the 18 people who volunteered for the study, 2 droppedout because of scheduling issues. The remaining 16 men(22.5 **6** 4.9 years) finished all stretching and nonstretchingtrials for SlowKE, FastKE, and VJheight. The average heightand mass of the study sample was 180.3**6**10.1 cm and 84.9**6**19.5 kg, respectively. Subjects had engaged in resistancetraining a minimum of 2 times a week, for the past 6 months.All subjects were free from musculoskeletal, cardiovascular,and metabolic disorders at the time of the study. Subjects gavewritten informed consent. The study was approved by theInstitutional Review Board of Utah State University.

Procedures

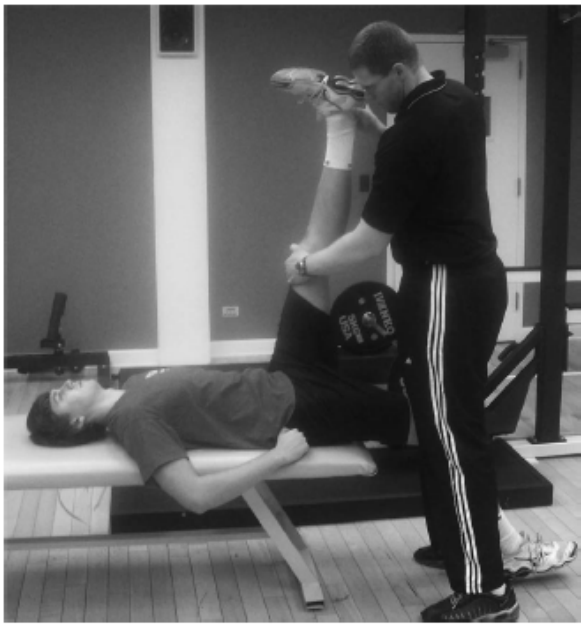

Figure 1. Hamstring stretch performed before knee extension.

Figure 2. Hip flexors stretch performed before vertical jump.

Figure 3. Dorsiflexor stretch performed before vertical jump.

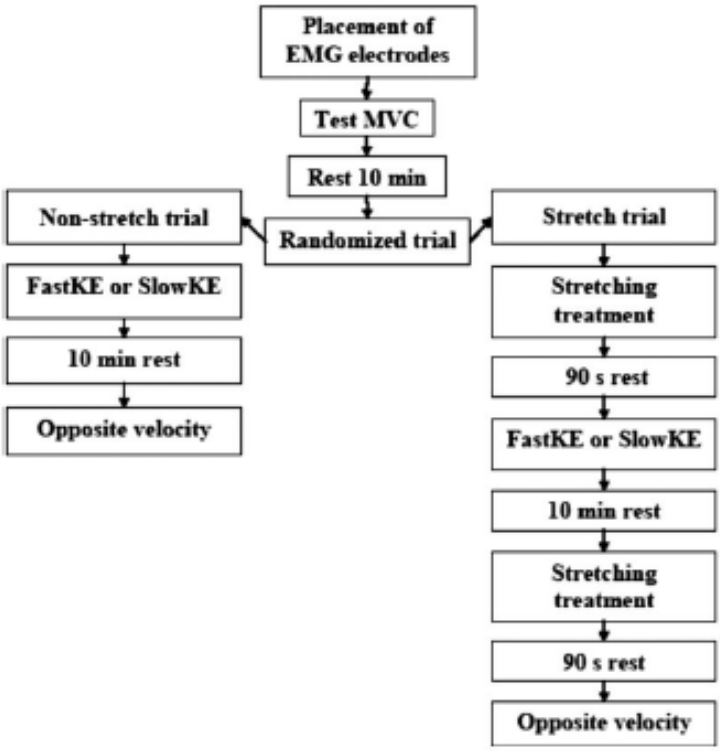

Figure 4. Summary for isokinetic knee extension trials.

Stretching Treatments. All stretches for the SlowKE, FastKE,and VJheight, were held for 30 seconds and repeated 3 times with 20-second rest between the stretches. Previous research has recommended holding static stretches for a duration of30 seconds (1,6). A 90-second rest period was provided between stretching and knee extension and VJ tests.

Stretches before the knee extension tests emphasized the hamstring group. With a subject supine on a training table, the investigator stabilized the opposing limb and put one hand on the subject's heel and the other hand just above the knee. The investigator then pushed the subject's heel and took him into knee extension and hip flexion (see Figure 1).

TABLE 1. Mean (±*SD*) for knee extension isokinetic peak torque and vertical jump (*N* = 16).*

Variable	Stretch	Nonstretch	% change	*P*	*ES*
FastKE (N.m)	102.2 ± 26.8	93.5 ± 33.4	9.3	0.032	0.29
SlowKE (N.m)	176.7 ± 52.1	162.9 ± 46.3	7.8	0.086	0.29
VJheight (cm)	59.8 ± 13.3	58.6 ± 13.3	2.0	0.011	0.09
VJpower (W)	8571 ± 597	8487 ± 615	1.0	0.005	0.14

*FastKE = knee extension fast speed (300°.s^{-1}); SlowKE = knee extension slow speed (60°.s^{-1}); VJheight = vertical jump height; VJpower = vertical jump power; ES = effect size.

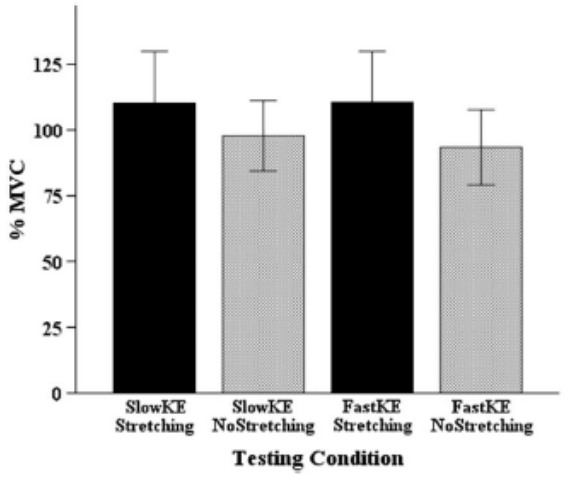

Figure 5. Mean percentage of maximal voluntary contraction (**6**SEM) for all conditions of the vastus lateralis.

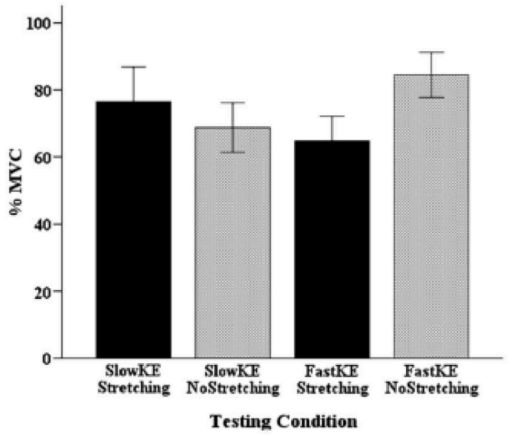

Figure 6. Mean percentage of maximal voluntary contraction (**6**SEM) for all conditions of the biceps femoris.

The stretching treatment before the vertical jump emphasized the stretching of the hip flexors and dorsiflexors. To stretch the hip flexors, the subject was positioned in a half-kneel position. For comfort, a foam pad was placed under the knee of the kneeling limb. The subject was instructed to keep an erect torso. The hip that was posterior was then extended by contracting the gluteals. The subject was then instructed to internally rotate the leg or turn his foot out (see Figure 2).Internal rotation of the hip joint effectively stretches the hip flexors (iliopsoas group) because of their insertion point on the lesser trochanter of the femur. Because the knee was placed only in partial flexion, a greater emphasis of stretch was placedon the single-joint hip flexors (iliopsoas group) vs. the 2-jointhip flexors that also act at the knee joint. For example, therectus femoris, which has a dual role in hip flexion and knee extension, was lengthened more at the origin (anterior inferioriliac spine and acetabulum) than at the insertion (tibial tuberosity via the quadriceps tendon).

To stretch the dorsiflexors, subjects began in a supine position on a training table with their feet hanging freely off the edge of the table. An investigator put the foot into plantar flexion by pulling on the toes and pushing on the heel(see Figure 3). The

dorsiflexors were stretched to a point of mild discomfort. The dorsiflexors were stretched first followed by the hip flexors.

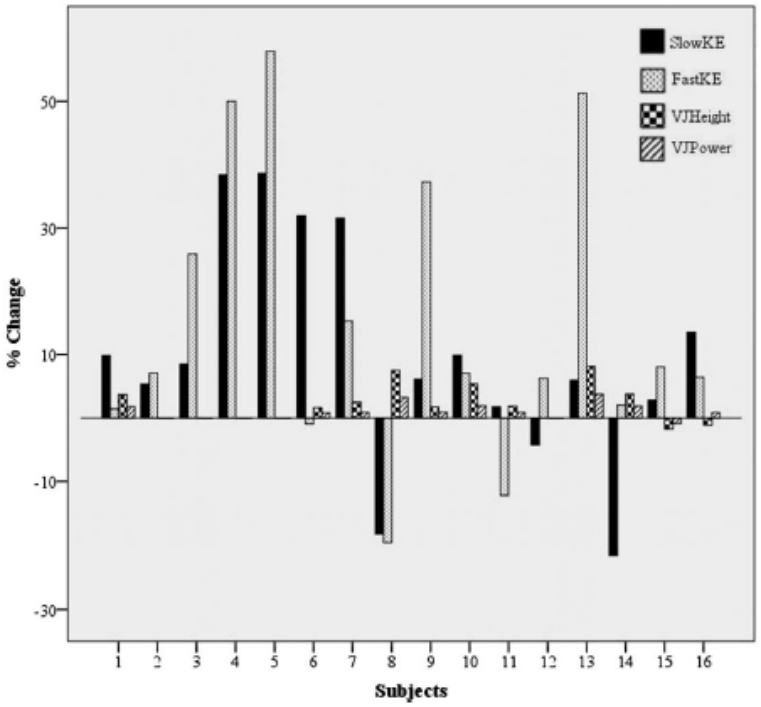

Figure 7. Percent change from nonstretching to stretching trials for knee extension torque at 60_.s**2**1 (SlowKE), knee extension torque at 300_.s**2**1 (FastKE), vertical jump height (VJHeight), and vertical jump power (VJPower) for each subject (N = 16).

Isokinetic Testing. Knee extensors peak torque was measured on a Biodex isokinetic dynamometer (Biodex, Shirley, NY,USA). Calibration was performed before testing. Subjects were tested in a seated position with straps placed over their waist and distal thigh for stabilization. The tibial pad was placed and secured approximately 2 finger widths proximal tothe medial malleolus on the dominant leg. The axis of rotation of the dynamometer was aligned with the medial epicondyle of the knee. Concentric peak torque of the knee extensors was recorded at 60_.s-1 and 300_.s-1. Each testing velocity was performed in a randomized order with 10-minute rest between maximal tests. For the stretching treatment, the stretching protocol was repeated before each maximal attempt at each testing velocity. Five maximal attempts were made, and the highest value was used for data analysis. A similar 5 repetition isokinetic protocol has been shown to be reliable (r = 0.95) for 60_.s-1 and 300_.s-1 (15).

Electromyography. Electromyography(EMG) was collected from the vastus lateralis and the long head of the biceps femoris muscles during knee extension tests using Biopac Systems instrumentation (MP150; Goleta, CA, USA). Positioning and placement of the electrodes was determined using procedures described byHerda et al. (20). Before applying the EMG electrodes, the skin at the placement sight was shaved, rubbed with alcohol, and slightly abraded to ensure good surface contact and to reduce skin resistance. Bipolar surface electrodes (2.5 cm interelectrode distance) were placed at the approximate center of each muscle belly. Aground

electrode was applied to the tibial tuberosity. For the biceps femoris, the electrodes were placed at 50% of the distance from the ischial tuberosity to the lateral epicondyle of the tibia. For the vastus lateralis, the electrodes were placed at 50% of the distance between the greater trochanter and the lateral epicondyle of the femur. The positions of the electrodes were marked with a small ink mark on the skin. The precise distance was also recorded and used for electrode placement in all conditions. The electrodes were placed before commencing the stretching treatments.

The raw EMG signals were preamplified 100 times at the electrode site, then further amplified for a total gain of 5,000with a bandwidth of 10–500 Hz. A low-pass filter was usedwith a cutoff frequency of 250 Hz. A high-pass filter was used with a cutoff frequency of 25 Hz. The EMG signal wassmoothed by integration. As recommended by Basmajian and DeLuca (3), the root mean square was calculated for the signal after it was filtered and smoothed.

Normalization of EMG signal characteristics is typically performed when comparisons are to be made, which involve reapplication of electrodes or between individuals (4,13). In this study, normalization was done by taking the EMG signal from each treatment and comparing it with a reference contraction of the same muscle with the same electrode placements. The reference was a maximal voluntary contraction(MVC) with the dynamometer set at 45_ of knee flexion for terminal point. Subjects were tested in a seated position described previously for torque testing. Velocity was set at30_.s-1 and subjects performed 3 maximal knee extensions with a 5-second isometric hold at the terminal range of motion. Subjects were instructed to exert maximal effort against the tibial pad. The MVC was performed, and then subjects rested 10 minutes before performing stretching and randomized isokinetic testing. The peak EMG voltage for the vastus lateralis and biceps femoris for SlowKE stretch, SlowKE nonstretch, FastKE stretch, and FastKE nonstretch was divided by the peak EMG voltage for the MVC. The EMG results are represented as a percentage of MVC. A summary of the time line for isokinetic knee extension and EMG testing is shown in Figure 4.

Vertical Jump Testing. The vertical jump test was performed using a Vertec device (Sports Imports, Columbus, OH, USA)using a protocol described by Harman et al. (18). The investigator adjusted a vertical column with vanes low enough that the subject could register a standing reach measurement. The subject then stood so that the dominant hand reached straight upward and directly below the center of the vanes. The highest vane that could be pushed forward while standing flat-footed determined the standing reach height. The same reach height was used for both trials. The vertical column was then raised to accommodate the jumping abilityof the subject. The subject was allowed a countermovement with no approach step and then jumped to the highest vane possible. Subjects were allowed to jump until they were unable to touch a higher vane on 2 consecutive trials. Jump height was determined by subtracting the distance betweenthe highest vane touched and the standing reach. Jump height and body mass were used to calculate absolute VJpower

using the Harman equation (19). This test waschosen because it is a commonly used field test to measure power. The test has been found to have high reliability (r =0.94) (38).

Statistical Analyses

Statistical comparisons were made for the 2 conditions of the independent variable of the study (stretch and nonstretch).Differences of the mean values of the dependent variables for the 2 conditions were evaluated using paired t-tests. The dependent variables were SlowKE, FastKE, VJheight, VJpower,and normalized EMG activity for the vastus lateralis and the long head of the biceps femoris during knee extension tests. Significant differences were assumed with p # 0.05. Effect sizes were also calculated using Cohen's d for each dependent variable. All analyses were executed using Statistical Package for Social Sciences (SPSS 18.0; IBM, Somers, NY, USA).

RESULTS

Torque

The results for knee extension torque are summarized in Table 1.Stretching the antagonist elicited significantly greater torque production for the FastKE but not the SlowKE. According to Cohen (8), the effect sizes for both trials were moderate.

Vertical Jump Height and Power

Both VJheight and VJpower were significantly higher after the stretching protocol (Table 1). The effect sizes were small (8)for both the variables.

Electromyography

Paired samples t-test indicated no significant (p . 0.05)differences between the trials for EMG, represented as a percentage of the MVC. The results for all conditions of the vastus lateralis are summarized in Figure 5. The results for all conditions of the biceps femoris are summarized in Figure 6.

Subject Responsiveness

There was considerable variability among subjects as several had dramatic improvements, particularly in SlowKE and FastKE, following the stretching treatment while others had minimal or even negative responses. The percent change from nonstretching to stretching trials for the 4dependent variables for each subject is illustrated in Figure 7.

DISCUSSION

Despite a large amount of research on the effects of statically stretching the agonist musculature before rapid force production, this is the first published study to our knowledge that has examined the effects of statically stretching the antagonist musculature before a power movement. We hypothesized that a static stretch of the antagonists could inhibit this muscle group, allowing for greater expression of strength and power production of the agonists. Indeed, the primary finding of this study was that there were statistically significant increases in isokinetic knee extension torque at 300_.s21 as well as VJheight and VJpowerfollowing static stretches of the antagonist musculature. However, these results are tempered by the fact that the magnitudes of the increases were small (8). Furthermore, there was no significant difference in EMG between stretching and nonstretching trials.

The increase in isokinetic knee extension torque occurred only at 300_.s21 and not 60_.s21, suggesting that the effects of antagonist stretching on torque production could be velocity specific. Nelson et al. (24) found an opposite and negative effect for agonist stretching on peak knee extension torque. These researchers reported that knee extension torque was reduced at the slowest velocities after static stretching of the quadriceps. In the current study, antagonist stretching exhibited the opposite effect vs. the agonist stretching inthe study by Nelson et al. (24); in the current study, torque increased 9.3% at the fastest velocity after antagonist stretching. However, the majority of other studies that have examined torque at different speeds after agonist stretching have found no velocity-specific effect (10–12,23,32,39).

Decrements in torque production of 2–3% after static stretching of the agonists have been reported in several studies (10–12,23), whereas others have reported slightly larger drops in isokinetic peak torque of 5–12% (24,31,32,39).However, as Sobolewski et al. (32) observed, the magnitudes of these decrements were often within the standard error of measurement of isokinetic testing, and the effect sizes were small. Similarly, we observed an 8% increase for SlowKE and a 9% increase for FastKE after stretching of the antagonist, but the magnitudes of these increases exhibited small effect sizes (8). Thus, although there is a trend (and often a statistically significant difference) for static stretching to impact torque output, whether these differences between stretching and nonstretching conditions have practical meaning is not clear. Nevertheless, the magnitude of torque increase observed in the present study after static stretching of the antagonist was remarkably similar to the magnitude of decrease in torque output that has been reported after static stretching of the agonist.

Similar to the change in isokinetic peak torque after static stretching of the antagonist, there was a statistically significant increase in VJheight and VJpower after static stretching of the hip flexors and dorsiflexors, the antagonists of the hip extensors and plantar flexors, but the effect sizes associated with these changes were small. The magnitude of change in VJheight observed in the present study is similar to, but in the opposite direction of, that reported by other researchers who have examined the effects of agonist stretching on VJheight; we observed an

increase in VJheight after static stretching of the antagonists, whereas other researchers observed a decrease in VJheight after static stretching of the agonists.

Church et al. (7) reported a decrease of 1.5 cm in VJheight following proprioceptive neuromuscular facilitation (PNF)stretching of the hamstrings and quadriceps. This is similar to the increase of 1.2 cm (2%) in VJheight seen in the current study. Robbins and Scheuermann (27) investigated the effects of 3 different volumes of static agonist stretching on vertical jump height. The greatest stretching volume of 6 sets of15 seconds decreased VJheight by an average of 1.9 cm. Cornwell et al. (9) also found a significant decrease in VJheight after acute static stretching of the plantar flexors.

Two hypotheses have been proposed for agonist stretch–induced force deficits (9,16,20,33). One proposed mechanism for decreased strength and power after agonist preactivity static stretching involved mechanical adaptations, namely, reduction in stiffness and increase in length between resting sarcomeres that alters the length-tension relationship of the muscle. Thesecond proposed mechanism involved neural factors such as decreased recruitment or reflex sensitivity or both. It was hypothesized in the current study that stretching the antagonist musculature would result in an increased performance by increasing the neural drive to the agonist, decreasing neural drive to the antagonist, reducing stiffness of the antagonist and braking forces to the agonist, or a combination of these factors. Despite an increase in isokinetic peak torque for FastKE after hamstring stretching and a 9.7% increase in EMG activity of the vastus lateralis following the stretching trial compared with the nonstretching trial, the differences of EMG activity between test conditions were not statistically significant. Similarly, the EMG activity of the antagonist biceps femoris during FastKE was 16% lower after the stretching trial compared with the nonstretching trial, but again this difference was not statistically significant. The lack of a statistically significant finding for EMG activity suggests that the difference in torque observed was not related to increased activation of the prime movers.

Herda et al. (20) found no change in EMG after agoniststatic stretching. They hypothesized that decrements in force following stretching were related to mechanical factors. Cornwell et al. (9) found a decrease in EMG activity and stiffness after agonist static stretching. However, these authors hypothesized that reductions in stiffness were insufficient to cause a decrease in force production. Fowles et al. (16) found that EMG activity was significantly decreased for the first15 minutes following static stretching, and force decrements were greatest during this time. However, electrical activity did return to normal after 15 minutes while force decrements remained for 60 minutes. These authors theorized that neural factors played a bigger role in strength decreases early, but astime passed, the reduction in maximum voluntary contractions originated peripherally in the muscle. It is possible that the improvement in knee extension torque after antagonist stretching at 300_.s**2**1 in a mechanically mediated response. If the length-tension relationship of the hamstrings was disrupted, this could lead to a reduction in braking forces,

which would allow the quadriceps to produce more torque. This is speculation because no measure of mechanical adaptation was taken.

With each measured variable, there was a large amount of subject-to-subject variability, as shown by the relatively large SDs (Table 1). The differences of interindividual strength and power responses to antagonist stretching may have been due to initial levels of flexibility. One of the limitations of this study is that there was no initial flexibility assessment taken. There is evidence that tight or short antagonist musculature may result in decreased function of the agonist musculature (29,34,35). It is possible that individuals with lower initial levels of flexibility in antagonist musculature experienced a greater training effect with stretching than those with higher initial levels. The researchers are not aware of any studies that have investigated whether initial levels of flexibility affect the magnitude of treatment effect from stretching, and this is a potential avenue of investigation.

PRACTICAL APPLICATIONS

Antagonist stretching of the hamstrings resulted in significantly greater torque during a high-velocity (300_.s**2**1)isokinetic knee extension. The take home message for the practitioner is that stretching the antagonist to the hip extensors (hip flexors) and plantar flexors (dorsi flexors) before jumping resulted in significantly greater VJheight andVJpower. These findings suggest that stretching the antagonist musculature immediately before a high-velocity activity may enhance the performance of that activity. Practitioners could apply the results of the current study in designing the warm-up procedures before plyometric training sessions orother training sessions that require high-velocity movements.

However, the findings of the current study are mitigated by the fact that the magnitude of improvement was small, and there was substantial interindividual variability in the response to antagonistic stretching. Furthermore, the difference in EMG activity after antagonistic stretching compared with a nonstretching trial was not significant, leaving the mechanism of improvement in doubt. Nevertheless, there is justification for practitioners to experiment with stretching the antagonist musculature to improve performance in high-velocity activity.

For the researcher, there is ample opportunity for further investigation on this topic. Future research should investigate other muscle groups and movement patterns. It should also be determined if initial levels of flexibility affect responses to antagonist stretching before strength and power–related performance. The effects of antagonist stretching using other stretching techniques (e.g., PNF, dynamic) should be investigated, as well as gender effects to antagonist stretching. Future research should also attempt to determine possible mechanisms, whether mechanical, neural, or a combination of both.

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