Effect of Stretching on Strength Loss and Pain after Eccentric Exercise

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ABSTRACT

MCHUGH, M. P., and M. NESSE. Effect of Stretching on Strength Loss and Pain after Eccentric Exercise. *Med. Sci. Sports Exerc.*, Vol. 40, No. 3, pp. 1–8, 2008. **Purpose:** The purposes of the this study were to determine whether stretch-induced strength loss was muscle length dependent (study 1) and whether passive stretching prior to eccentric exercise affected strength loss and pain on subsequent days (study 2). **Methods:** For study 1, knee flexion strength was measured isometrically (six angles) and isokinetically (eccentric and concentric) in 10 men (33 ± 9 yr). The subjects then performed six 90-s static hamstring stretches, after which isometric and isokinetic strength were retested. For study 2, the dominant and nondominant legs of eight men (34 ± 9 yr) were assigned to a stretch (six 60-s stretches) or control condition prior to eccentric hamstring exercise. Isometric strength and pain were assessed prior to, immediately after, and on the 3 d after exercise. **Results:** After stretching, strength was decreased by 17% at 80°, 11% at 65°, 5% at 50°, 7% at 35°, and 8% at 20°, and it was increased by 6% at 5° (angle effect $P < 0.01$). Strength loss following eccentric exercise was less on the stretched versus the unstretched control limb at 37° ($P < 0.05$), but not at other angles (stretch by time by angle $P < 0.01$). Pain was not different between the stretched and the unstretched control limb ($P = 0.94$). **Conclusion:** Stretch-induced strength loss was dependent on muscle length, such that strength was decreased with the muscle group in a shortened position, but not with the muscle group in a lengthened position. Strength loss and pain after eccentric exercise were generally unaffected by prior stretching, with the exception that stretching prevented strength loss when assessed with the muscle in a lengthened position. **Key Words:** ANGLE–TORQUE RELATIONSHIP, MUSCLE DAMAGE, HAMSTRING, KNEE FLEXION

Passive muscle stretching has repeatedly been shown to result in an acute loss of strength (1,7,8,9,10,15, 23). A poststretching decrease in the amplitude of the surface EMG signal during maximum voluntary contractions (1,8,23) and poststretching strength loss in the contralateral unstretched limb (8) indicate that stretch-induced strength loss has a neural component. However, there is also evidence of a mechanical effect of stretching, with stretch-induced strength loss more apparent at short versus long muscle lengths (12,27). This muscle length effect was attributed to stretch-induced increases in tendon compliance, allowing greater muscle fiber shortening for a given muscle length. In support of this explanation, static stretching of the plantarflexors has been shown to increase tendon extensibility (16). An increase in tendon and/or aponeurosis extensibility will allow greater muscle fiber shortening during muscle contraction and, therefore, would shift the angle–torque curve to the right (reduced torque at short muscle lengths increased torque at long muscle lengths). If a stretching intervention is sufficient to increase tendon and/or aponeurosis compliance, both neural and mechanical effects might affect subsequent maximal voluntary torque production. Stretching would decrease torque production at muscle lengths shorter than optimum, where the mechanical and neural effects would be additive. At muscle lengths longer than optimum, the mechanical and neural effects of stretching would be opposing. Therefore, stretch-induced strength loss may be diminished or absent at longer muscle lengths. Nelson et al. (27) and Herda et al. (12) have demonstrated stretch-induced strength loss at short muscle lengths, but not at long muscle lengths. However, in neither study was the change in passive resistance to stretch after the stretching intervention reported.

A stretch-induced increase in tendon and aponeurosis extensibility would reduce muscle fiber strain, and, more specifically, sarcomere strain, during eccentric contractions. A reduction in sarcomere strain will limit myofibrillar disruption and minimize the subsequent markers of muscle damage associated with eccentric contractions. In this regard, protection from eccentric contraction–induced muscle damage with a repeated bout has been attributed to a rightward shift in the angle–torque curve for knee extension (26) and knee flexion contractions (4). Additionally, susceptibility to muscle damage from an initial bout of eccentric contractions was shown to be related to interindividual differences in the angle–torque relationship (25,26). Greater relative force production at longer muscle lengths prior to eccentric exercise was associated with less strength loss on the days after eccentric

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exercise. Theoretically a stretch-induced rightward shift in the angle–torque relationship (decreased force at short muscle lengths, increased force at long muscle lengths) would be expected to decrease strength loss on the days after a bout of eccentric exercise.

Numerous studies have previously examined the effects of preexercise stretching on the subsequent symptoms of muscle damage. A single preexercise bout of stretching in humans (11,13,14,18) and in an animal model (3) has been shown to have no effect on the symptoms of muscle damage after eccentric exercise. Similarly, a period of stretching has also been shown to have no effect on the symptoms of muscle damage in humans (17) and in an animal model (2). However, the preexercise combination of stretching and warm-up was shown to reduce symptoms of muscle damage in some studies (29,30), but not in another (13). In none of these studies were changes in passive resistance to stretch reported. It is possible that the preexercise stretching protocols were inadequate to affect passive resistance to stretch. For example, four 20-s static hamstring stretches (14) or three 30-s quadriceps stretches (18) would not be expected to alter passive resistance to stretch. Magnusson et al. (21) report that three 45-s hamstring stretches did not reduce passive resistance to stretch but that five 90-s stretches were sufficient to reduce passive resistance to stretch by approximately 15% (22). In determining the effect of preexercise passive stretching on the subsequent symptoms of muscle damage, it is important to document the mechanical effects of the stretching intervention.

Therefore, the purpose of this study was twofold. The purpose of the initial part of this study (study 1) was to measure the change in passive resistance to stretch after a stretching intervention, and to determine whether any subsequent stretch-induced strength loss was muscle-length dependent. The purpose of the second part of the study (study 2) was to determine whether a bout of passive muscle stretching, sufficient to decrease passive resistance to stretch, performed before eccentric exercise, would affect strength loss and pain on subsequent days. For study 1, it was hypothesized that stretching would reduce passive resistance to stretch and that subsequent stretch-induced strength loss would be greater at short versus long muscle lengths. For study 2, the hypothesis was that stretching before eccentric exercise would reduce the strength loss and pain on subsequent days.

METHODS

Methods Study 1: The Effect of Passive Stretching on the Angle–Torque Relationship

Protocol. Knee flexion strength was measured isometrically (six angles) and isokinetically (eccentric and concentric) in 10 men (33 ± 9 yr). The subjects then performed six 90-s static hamstring stretches during which passive resistive torque was measured. After the stretching, isometric and isokinetic strength were retested. The delay from the last stretch to the initiation of poststretching isometric contractions was less than 1 min in all cases (switch program from passive to isometric protocol). Only one leg was tested for each subject (arbitrarily chosen). The protocol was approved by institutional review board, and all subjects gave written informed consent.

Isometric and isokinetic knee flexion strength. Subjects were seated with the test thigh flexed 20° higher than the contralateral thigh and the trunk flexed to 90°. This created a trunk-to-thigh angle of 70°, which was sufficient to place significant stretch on the hamstring muscle group as the knee was moved into full extension (Fig. 1). In this position, subjects performed two maximum isometric knee flexion contractions (Biodex System 2, Shirley, NY) 30 s apart, at each of six knee flexion angles (80, 65, 50, 35, 20, and 5°). The sequence of contractions was always from short to long muscle length (80° to 5°), with 1 min between test angles. Torque attributable to limb mass and passive hamstring stiffness was recorded at each angle and subtracted from the average of the two peak torques during maximum contractions at each angle. F1

After isometric contractions, subjects performed four maximum reciprocal eccentric and concentric knee flexion contractions (80° to 0° for eccentric and 0° to 80° for concentric) at $60°\cdot s^{-1}$, with a 1-s pause between eccentric and concentric phases.

This sequence of isometric and isokinetic strength testing was repeated immediately after the stretching protocol was completed.

Static stretching protocol. Subjects remained in the same seated position after the isokinetic contractions.

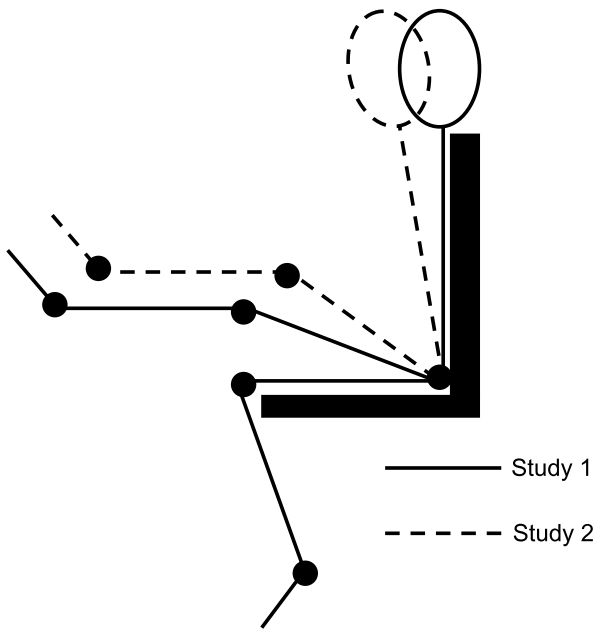

FIGURE 1—Test position for isometric contractions, isokinetic contractions, and passive hamstring stretch in study 1 (*solid lines*) and study 2 (*dashed lines*). The combination of thigh flexion and trunk flexion increased the stretch on the hamstrings in study 2 versus study 1 for any given knee flexion angle.

Immediately after the last contraction, the leg was passively stretched to full extension at $5°\cdot s^{-1}$ and held in that stretched position for 90 s. After 90 s, the leg was returned to 80° knee flexion at $5°\cdot s^{-1}$ and immediately brought back to full extension at the same speed. This sequence was repeated for six 90-s stretches. Passive torque at full extension was recorded at the start of the first and sixth stretches.

Methods Study 2: The Effect of Passive Stretching on the Strength Loss and Pain after Eccentric Exercise

Protocol. The dominant and nondominant legs of eight men (34 ± 9 yr) were alternately assigned to either a stretch or control condition for the experimental protocol. Six of the eight subjects were also subjects in study 1, with more than 6 months between studies. For the stretch condition, isometric knee flexion strength was measured at five angles prior to six 60-s static hamstring stretches. After the stretches, subjects performed six sets of 10 unilateral isokinetic eccentric hamstring contractions at 100% of isometric MVC. The delay from the last stretch to the initiation of eccentric contractions was approximately 1 min and not more than 2 min in any case. For the control condition, the contralateral limb performed the isokinetic eccentric hamstring contractions 8 min after the isometric contractions. The subjects sat quietly during the 8-min period prior to eccentric contractions, to simulate the time required for the stretch condition. For both limbs, isometric strength was retested immediately after the eccentric contractions and on each of the next 3 d. Additionally hamstring pain was assessed on each of these days. In addition to alternately assigning the dominant or nondominant leg to the stretch or control condition, the first leg to be tested (stretch or control) was alternated for each subject. Thus, with eight subjects, all four possible sequences were repeated twice. The protocol was approved by institutional review board, and all subjects gave written informed consent.

Isometric knee flexion strength. Based on the shape of the angle–torque curve for isometric contractions in study 1, it was determined that the hamstring muscle group needed to be placed in a more stretched position for testing in study 2, to have more than one test angle on the descending limb of the angle–torque curve. Therefore, subjects were seated with the test thigh flexed to 40° higher than the contralateral thigh (previously 20)° and the trunk flexed to 80° (previously 90°). This created a trunk–test thigh angle of 40°, which was sufficient to place significant stretch on the hamstring muscle group such that subjects were not able to reach full extension (Fig. 1). In this position, subjects performed two maximum isometric knee flexion contractions (Biodex System 2, Shirley, NY) at each of five knee flexion angles (89, 76, 63, 50, and 37°). As described above, the sequence of contractions was always from short to long muscle length. Torque from limb mass and passive hamstring stiffness was recorded at each angle and subtracted from the average of the two peak torques during maximum contractions at each angle.

Static stretching protocol. Subjects remained in the same seated position after the isometric contractions. Immediately after the last contraction, the limb assigned to the stretch condition was passively stretched to maximum tolerable range of motion at $5°\cdot s^{-1}$ and held in that stretched position for 60 s. After 60 s, the leg was returned to 89° knee flexion at $5°\cdot s^{-1}$ and immediately brought back to the previous angle at the same speed. This sequence was repeated for six 60-s stretches. Passive torque at the end of range of motion was recorded at the start of each stretch. The stretch duration was decreased from 90 s (study 1) to 60 s (study 2) because of the discomfort of the test position and the magnitude of the stretch (significantly more discomfort than the stretch in study 1).

Isokinetic eccentric exercise. The subjects remained in the same seated position for the isokinetic eccentric exercise. For both the stretch and control conditions, six sets of 10 unilateral isokinetic eccentric hamstring contractions were performed at $60°\cdot s^{-1}$ from 89° to 32° of knee flexion (all subjects could passively reach 32° knee flexion in this test position). Target torque was set at 100% of isometric MVC (peak torque at each subject's optimal angle).

Symptoms of muscle damage. Isometric strength loss and pain were recorded as indirect indicators of muscle damage. Isometric strength was assessed at five angles, as described above. Measurements were made prior to eccentric exercise, immediately after eccentric exercise, and on each of the next 3 d. Strength changes at each test angle are expressed as a percentage of the baseline strength prior to eccentric exercise at each test angle. Pain in the hamstring region was expressed on a 0- to 10-point scale, where zero was "no hamstring discomfort" and 10 was "walking with a limp due to hamstring discomfort or pain." Pain assessments were

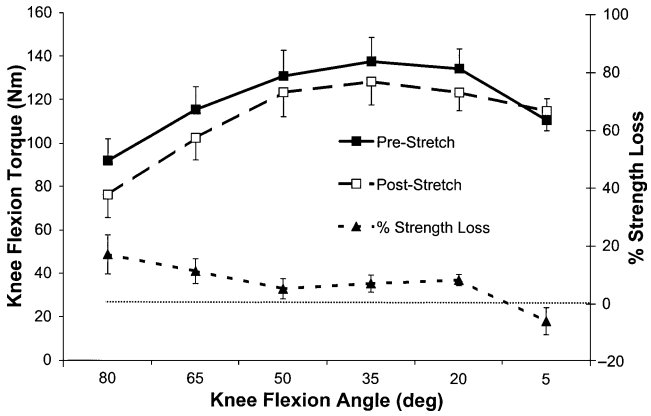

FIGURE 2—Isometric knee flexion torque prior to (*solid line*, *filled boxes*) and after (*dashed line*, *open boxes*) six passive hamstring stretches. Torque was measured at six different knee flexion angles from short muscle length (80°) to long muscle length (5°). Stretch × angle $P < 0.01$ (mean ± SE displayed). Percent strength loss at each angle is also displayed (*dashed line*, *closed triangles*) and graphed on the right *y*-axis (*dotted line* indicates zero strength loss); angle effect $P < 0.01$.

made prior to the experimental protocol and on each of the 3 d after the eccentric exercise. Pain values were recorded for both the stretched limb and unstretched control limb.

Statistics. The effect of stretching on passive resistance to stretch (study 1 and study 2) was assessed using paired *t*-tests to compare passive torque at the end of range of motion between stretch 1 and stretch 6. The effect of stretching on the angle–torque relationship for isometric contractions (study 1) was assessed using a 2 × 6 repeated-measures analysis of variance (ANOVA), where the factor with two levels was stretch (pre to post) and the factor with six levels was angle (80, 65, 50, 35, 20, and 5°). Paired *t*-tests were used to compare peak isokinetic torque and angle of peak isokinetic torque prior to and following passive stretch.

The hypothesis for study 1 was that there would be a muscle length–dependent effect on stretch-induced strength

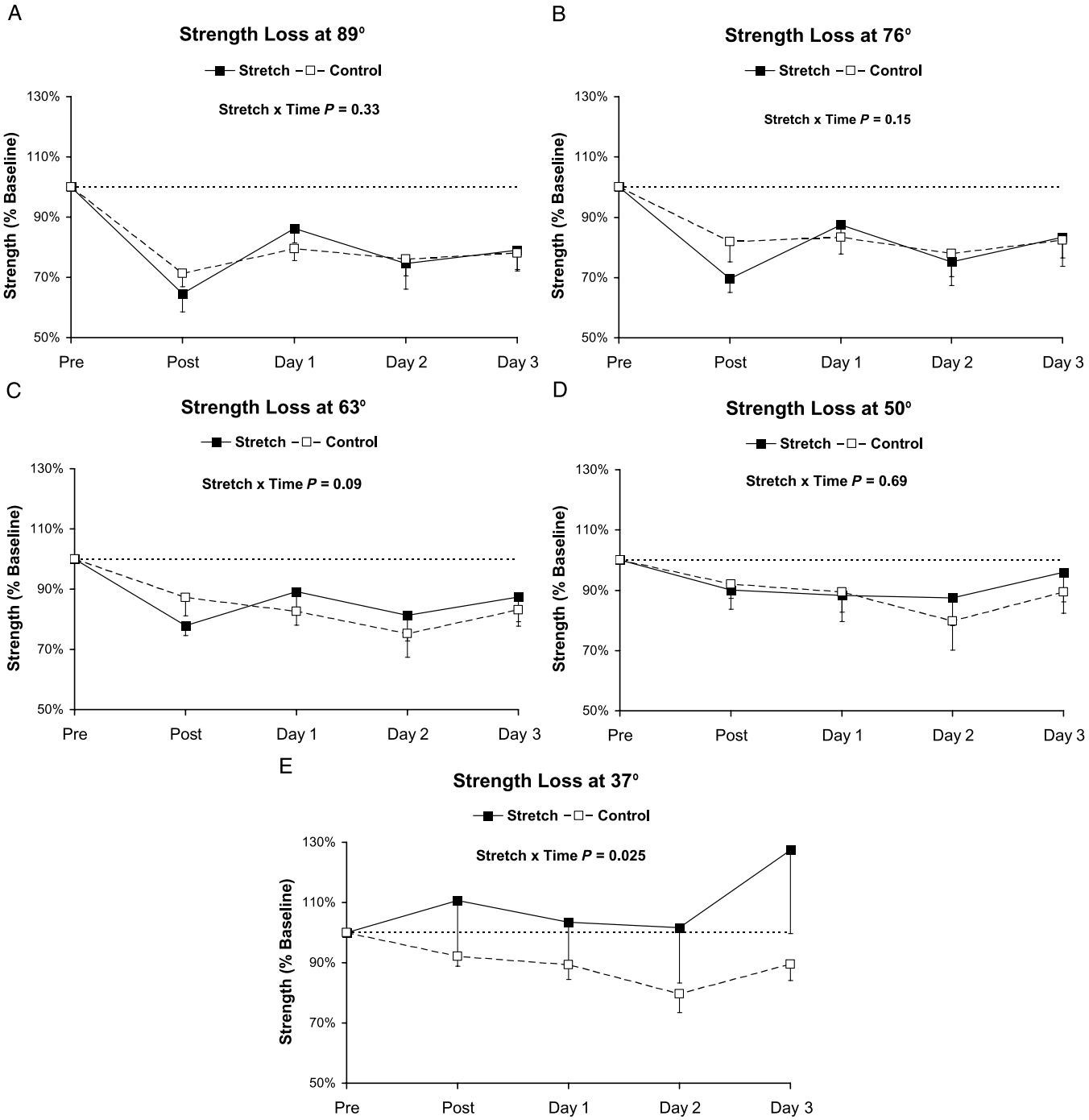

FIGURE 3—Isometric knee flexion strength loss at each test angle (*A–E*) after eccentric contractions in the stretched limb (*closed squares* and *solid line*) and unstretched control limb (*open squares*, *dashed line*). Stretch by angle by time interaction $P < 0.01$. The *dotted line* on each graph indicates baseline strength. The hamstring muscle group was in the most shortened position at 89° (*A*) and in the most lengthened position at 37° (*E*).

loss. Therefore, the study was powered to detect a difference in stretch-induced strength loss at short versus long muscle lengths. Based on previously published variability in isometric hamstring strength (24), it was estimated that there would be 80% power to detect a 13% change in strength at $P < 0.05$ with 10 subjects. For the purposes of this study, a difference in stretch-induced strength loss of 13% could be detected between short and long muscle lengths, such as a 7% decrease at 80° versus a 6% increase at 5°.

The effect of stretching on isometric strength loss after eccentric exercise (study 2) was assessed using a 5 × 5 × 2 repeated-measures ANOVA, where the factors with five levels were time (pre, post, day 1, day 2, or day 3) and angle (89, 76, 63, 50, or 37°), and the factor with two levels was stretch (stretched limb vs unstretched control limb). Similarly, the effect of stretching on pain after eccentric exercise was assessed using a 4 × 2 repeated-measures ANOVA, where the factor with four levels was time (pre, day 1, day 2, or day 3) and the factor with two levels was stretch (stretched limb vs unstretched control limb).

The hypothesis for study 2 was that there would be less strength loss on the stretched limb on the days after eccentric exercise. Therefore, the study was powered to detect a difference in isometric knee flexion strength loss between the stretched and unstretched control limb. Based on previous data examining differences in posteccentric exercise isometric knee extension strength loss between limbs (25), it was estimated that there would be 80% power to detect an 8% difference in strength loss (averaged for the four postexercise measurements) between the stretched and control limbs at $P < 0.05$. It was presumed that the interlimb variability in strength loss after eccentric exercise would be similar for knee flexion and knee extension, given that similar measurement techniques were adopted. Data are reported as means ± SE in the text and in the figures.

RESULTS

Study 1: the effect of passive stretching on the angle–torque relationship. Passive resistance to stretch decreased from 59.5 ± 4.5 N·m at full extension at the beginning of the first stretch to 54.6 ± 4.1 N·m at the beginning of the sixth stretch ($P < 0.05$). This was an 8.3% decrease in resistance to stretch.

There was a significant stretch by angle interaction for isometric knee flexion torque ($P < 0.01$), such that strength was decreased poststretch with the muscle group in a shortened position, but not with the muscle group in a lengthened position (Fig. 2). After stretching, strength was decreased by 17% at 80°, 11% at 65°, 5% at 50°, 7% at 35°, and 8% at 20°, and increased by 6% at 5° (angle effect on percent strength loss $P < 0.01$; Fig. 2).

Isokinetic eccentric and concentric peak knee flexion torques were unaffected by stretching ($P = 0.83$ and 0.81, respectively). Eccentric knee flexion torque (prestretch 140 ± 10 N·m, poststretch 138 ± 11 N·m) was significantly higher ($P < 0.01$) than concentric torque (prestretch 93 ± 8 N·m, 94 ± 9 N·m). The angle at peak torque for eccentric and concentric knee flexion contractions was also unaffected by stretching ($P = 0.73$ and 0.33, respectively). The angle of peak torque for eccentric contractions (11 ± 2° pre- and poststretch) was significantly lower (longer muscle length; $P < 0.01$) than for concentric contractions (prestretch 22 ± 3°, poststretch 24 ± 1°).

Study 2: the effect of passive stretching on the strength loss and pain after eccentric exercise. Passive resistance to stretch decreased from 53.7 ± 4.6 N·m at full extension at the beginning of the first stretch to 48.9 ± 3.4 N·m at the beginning of the sixth stretch ($P < 0.05$). This was a 9% decrease in resistance to stretch.

Overall strength loss after eccentric exercise was not different between the stretched and unstretched control limbs (time × stretch $P = 0.09$). However, there was a significant time × angle × stretch interaction ($P < 0.01$) on strength loss after eccentric exercise, indicating that the effect of stretching on strength loss was muscle length dependent. Further analysis showed that the strength loss following eccentric exercise was less on the stretched limb versus the unstretched control limb at 37° (time × stretch $P < 0.05$; Fig. 3E), but not at other angles (Fig. 3A–D). At 37° strength on the unstretched control limb was 82% of baseline (averaged across the four postexercise time points) compared with 111% of baseline on the stretched limb ($P < 0.05$).

On the stretched limb, there was greater strength loss at short versus long muscle lengths (angle effect $P < 0.05$), but this effect was not apparent on the unstretched control limb ($P = 0.21$). On the stretched limb, strength (averaged across postexercise, day 1, day 2, and day 3 measures) was 76% of baseline at 89° and 111% of baseline at 37°, whereas these values were 76% and 82% on the unstretched control limb.

Pain peaked at 2 d after eccentric exercise (time effect $P < 0.001$), with no difference ($P = 0.94$) between the stretched limb (peak 7.1/10) and the unstretched control limb (6.9/10).

DISCUSSION

Whereas numerous studies have examined stretch-induced strength loss (1,7,8,9,10,15,23) and the effect of preeccentric exercise stretching on the subsequent symptoms of muscle damage (11,13,14,18,29,30), none of these studies concurrently examined the change in passive resistance to stretch. In the present study, six 90-s stretches resulted in an 8% decrease in resistance to stretch (study 1), and six 60-s stretches (in a position of grater hamstring stretch) resulted in a 9% reduction in resistance to stretch.

Effect of stretching on the angle–torque relationship. With respect to study 1, it was hypothesized that passive stretching would increase muscle extensibility and allow greater muscle fiber shortening during isometric contraction at any given muscle length. It was theorized that

this effect would minimize stretch-induced strength loss with the muscle in a lengthened position. The results support this theory. Following hamstring stretching, knee flexion torque was clearly reduced at knee flexion angles on the ascending limb of the angle–torque curve (17% at 80° and 11% at 65°) but slightly increased at the only knee flexion angle on the descending limb of the angle–torque curve (6% at 5°). These results are in agreement with previous work showing a muscle length–dependent effect on stretch-induced strength loss (12,27). The finding that concentric and eccentric strength were unaffected by stretching (peak torque and angle of peak torque) can be explained by the fact that peak torque occurred close to the end of the range of motion (11° for eccentric, 22° for concentric). Isokinetic torque curves were not further examined to assess stretching effects at angles other than the angle at which peak torque occurred. The isometric angle–torque curves provided this information.

Difference in experimental set-up between study 1 and 2. Having established in study 1 that there was a muscle length component to stretch-induced strength loss, the purpose of study 2 was to examine whether stretching before eccentric exercise reduced the subsequent symptoms of muscle damage. The hypothesis was that a stretch-induced shift in the angle–torque relationship would limit sarcomere strain during eccentric contractions, thereby reducing the subsequent symptoms of muscle damage. Previous work has established that sarcomere strain is the initial step in the damage process and that decreased sarcomere strain (e.g., addition of sarcomeres) will reduce damage (19). Based on the shape of the angle–torque curve for study 1 (Fig. 3), it was apparent that the hamstring muscle group would need to be placed in a position of greater stretch for study 2. This would ensure that the eccentric contractions would be performed at muscle lengths where torque production would be compromised by sarcomere strain and, therefore, the stretching intervention would have a greater potential effect on reducing sarcomere strain during eccentric contractions. The test position for study 2 likely resulted in greater sarcomere strain, given that only one isometric test angle was on the ascending limb of the angle–torque curve (it was two in study 1), and two isometric test angles were on the descending limb of the angle–torque curve (it was one in study 1).

The effect of passive stretching on strength loss and pain after eccentric exercise. The hypothesis that stretching prior to eccentric exercise would reduce the subsequent symptoms of muscle damage was not supported by the data. The pain response was very similar between the stretched and unstretched control limb (stretch × time $P = 0.94$), and there was no clear effect of stretching on strength loss (stretch × time $P = 0.09$). However, it was apparent that stretching prevented strength loss with the muscle in a lengthened position (Fig. 3E). An 18% loss of strength at 37° on the unstretched control limb (averaged for the four postexercise measurements) contrasts with an 11% increase in torque on the stretched limb. The mechanism of this angle-specific stretching effect is not immediately apparent, but it might be related to the pattern of strength loss when eccentric contractions are performed with the muscle in a lengthened position. It is well established that strength loss after eccentric contractions is greater at short versus long muscle lengths (5,26,31). However, this effect was not evident in the present study. On the unstretched control limb, there was no muscle length–dependent effect: strength loss at each angle averaged across all four time points was 24% at 89°, 19% at 76°, 18% at 63°, 12% at 50°, and 18% at 37° (angle effect $P = 0.2$). By contrast, a muscle length–dependent pattern was evident on the stretched limb: strength loss at each angle averaged across all four time points was 24% at 89°, 21% at 76°, 16% at 63°, 10% at 50°, and −11% at 37° (angle effect $P < 0.05$).

The data for the unstretched control limb are consistent with the data from McHugh and Pasiakos (25) indicating that greater strength loss is not apparent at short muscle lengths when the damaging eccentric exercise is performed with the muscle in a lengthened position. When eccentric contractions are performed with muscles in a lengthened position, sarcomere disruption may be combined with disruption of other structures affecting contractile force production. For example, disruption of parallel connective tissue structures such as the endomysium and perimysium may affect contractile force production. This effect would only be apparent when these structures are under tension, such as when the muscle is in a lengthened position. In support of this theory, Mackey et al. (20) has demonstrated endomysial disruption with eccentric contractions of the knee extensors performed through a range of 130°. Considering that peak knee extension torque occurs around 70°, a significant amount of eccentric work would have been performed with the knee extensors in a relatively lengthened position. Previously, Street (32) demonstrated that the endomysium is responsible for transmitting contractile forces laterally to adjacent fibers. The endomysium is more effective at transmitting contractile forces when the muscle fibers are in a lengthened position (28). Therefore, it is possible that the difference in strength loss between the stretched limb and unstretched control limb at 37° reflected a difference in endomysium disruption. The endomysium may have experienced less stress during the eccentric contractions in the stretched versus unstretched control limb. Such an effect could have been attributable to 1) decreased endomysial strain during eccentric contractions in the stretched limb secondary to a stretch-induced increase in tendon and aponeurosis compliance, 2) decreased endomysial stress for a given strain secondary to a stretch-induced increase in endomysial compliance, or 3) a combination of these effects. This theory could be tested in future research by examining the effect of stretching prior to eccentric exercise on subsequent connective tissue breakdown.

Any explanation of the effect of stretching on strength loss observed at 37° is purely speculative, because this was an unexpected finding based on the hypothesis and rationale for the study (it was hypothesized that strength loss would

be less at all angles and that pain would also be less on the stretched limb). It is apparent in Figure 3 that intersubject variability in strength loss was greater at 37° compared with other angles on the stretched limb, and greater at 37° on the stretched limb versus 37° on the unstretched control limb. This probably reflects the fact that the effect of stretching on strength loss at 37° was not uniform across subjects, whereas the lack of effect of stretching on strength loss at other angles was more uniform across subjects. Clearly, further study is needed to examine possible stretching effects on muscle function at long muscle lengths and what, if any, the practical implications are.

Limitations. It is important to appreciate methodological limitations when interpreting the results of this study. With respect to study 1, no attempt was made to measure the neural effect of stretching. It is possible that the length-dependent effect on strength loss was neural and not mechanical. Whereas neural inhibition may have been greater at short versus long muscle lengths, there is no direct evidence or theory to support such a conclusion. On the other hand, there is direct evidence to support the theory that stretching would have a length-dependent mechanical effect on muscle strength. Greater experimental control would have been achieved by having a control session (repeated isometric contractions at each angle without intervening stretching). It is possible that the isometric contractions resulted in muscle damage (6,34) and that the poststretching changes in isometric torque were attributable to the length-dependent muscle damage effects rather than static stretching. However, recent findings by Herda et al. (12) indicate that this may not have been the case. In that study, static stretching resulted in the same length-dependent strength loss as in the current study and that by Nelson et al. (27), but dynamic stretching did not affect isometric strength (12). If the isometric testing protocol had caused muscle damage and a length-dependent effect on strength loss, this should have been apparent after dynamic and static stretching. Thus, the isometric strength testing protocol did not seem to result in strength loss.

With respect to study 2, the effect of stretching on the angle–torque relationship was not examined immediately after stretching, and, therefore, based on the results of study 1 and the findings of Nelson et al. (27) and Herda et al. (12), it was presumed that there was a length-dependent effect for muscle contractions. Poststretching isometric contractions were not performed, because the testing protocol would have delayed the time from the last stretch to the beginning of the eccentric contractions. It has been shown that the stretch-induced decrease in passive resistance lasts less than 1 h (22), but more precise data are not available. Additionally, isometric contractions might have had an additive effect on reducing passive muscle tension. Previous work in an animal model has shown that passive muscle tension is similarly reduced with static stretching versus isometric contractions (33). Therefore, it would not have been possible to differentiate between effects of stretching versus isometric contractions. An interesting future study would be to examine the effect of contract–relax stretching prior to eccentric exercise. Isometric contractions during passive stretch (as employed in contract–relax stretching) may be more effective in increasing tendon and aponeurosis extensibility than static stretching, and, therefore, they would have a greater potential effect on muscle fiber mechanics during contraction. An additional limitation pertinent to both study 1 and 2 is that no attempt was made to examine tendon and aponeurosis extensibility during either isometric or eccentric contractions. Although this is possible using ultrasonic imaging, this technology was not available to the authors.

In conclusion, stretch-induced strength loss was dependent on muscle length, such that strength was decreased with the muscle group in a shortened position, but not with the muscle group in a lengthened position. This effect was attributed to greater muscle fiber shortening during eccentric contractions after stretching. Based on these observations, it was hypothesized that stretching prior to eccentric exercise would limit sarcomere strain during the eccentric contractions, resulting in reduced symptoms of muscle damage. However, strength loss and pain were generally unaffected by stretching prior to eccentric exercise, with the exception that stretching prevented strength loss when assessed with the muscle in a lengthened position. There may be practical implications of the observed effect of stretching prior to eccentric exercise on strength loss with the muscle in a lengthened position. Athletes often participate in sports or training with significant symptoms of muscle damage such as during preseason training. A stretch-induced preservation of strength with the muscle in a lengthened position following eccentric exercise may be functionally important in resisting potentially injurious muscle elongation during continued sport performance.

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