

## A mechanism for altered flexibility in human skeletal muscle

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1. We investigated the effect of a long-term stretching regimen on the tissue properties and stretch tolerance of human skeletal muscle.
2. Resistance to stretch was measured as torque (in N m) offered by the hamstring muscle group during passive knee extension while electromyographic (EMG) activity, knee joint angle and velocity were continuously monitored during a standardized stretch manoeuvre. Seven healthy subjects were tested before and after a 3 week training period using two separate protocols. Protocol 1 consisted of a slow stretch at  $0.087 \text{ rad s}^{-1}$  to a predetermined angle followed by a 90 s holding phase. Subjects were brought to the same angle before and after the training period. Protocol 2 was a similar stretch, but continued to the point of pain.
3. During protocol 1 the torque rose during the stretch and then declined during the holding phase. EMG activity was small and did not change significantly during the protocol. No significant differences in stiffness, energy and peak torque about the knee joint were seen as a result of the training. During protocol 2 the angle to which the knee could be extended was significantly increased as a result of the training. This was accompanied by a comparable increase in peak torque and energy. EMG activity was small and not affected by training.
4. It is concluded that reflex EMG activity does not limit the range of movement during slow stretches and that the increased range of motion achieved from training is a consequence of increased stretch tolerance on the part of the subject rather than a change in the mechanical or viscoelastic properties of the muscle.

The stretching of human skeletal muscle to augment the range of motion of joints is commonly applied in diverse areas of medicine and recreational activities. However, the mechanism for the acute and chronic changes in joint range of motion remains ambiguous. The acute response to stretching has been attributed to both neurophysiological and mechanical factors (Taylor, Dalton, Seaber & Garrett, 1990; Hutton, 1993). The neurophysiological explanation suggests the limiting factor during stretching to be muscular resistance secondary to reflex activity (Hutton, 1993). Accordingly, the aim of stretching is to inhibit the reflex activity, which reduces resistance and thereby improves joint range of motion (Hutton, 1993), but paradoxically, the techniques most effective in acutely increasing joint range of motion have been associated with an elevated EMG response (Moore & Hutton, 1980; Osternig, Robertson, Troxel & Hansen, 1987). Alternatively it has recently been proposed that acute adaptation may be attributed to an amplified stretch tolerance rather than a change in EMG activity (Magnusson, Simonsen, Aagaard, Dhyre-Poulsen, McHugh & Kjær, 1996a).

A third explanation for the acute effects of stretching is a change in the mechanical properties of the muscle (Taylor *et al.* 1990). Biological materials under tension exhibit viscoelastic behaviour and animal data have provided evidence that the muscle-tendon unit displays viscoelastic behaviour both during the loading and holding phases of a stretch (Abbott & Lowy, 1956; Taylor *et al.* 1990). Human models have shown that a chronic regimen of stretching may increase joint range of motion (Sady, Wortman & Blanke, 1982; Gajdosik, 1991; Bandy & Irion, 1994). The suggested mechanism for this augmented joint range of motion is a change in the tissue properties of the muscle (Gajdosik, 1991; Hutton, 1993). The development of a new technique allows for the continuous and synchronous monitoring of joint angle, velocity, muscle EMG response and passive resistance to stretch of the human hamstring muscle during the stretch and holding phase of a stretch (Magnusson, Simonsen, Aagaard, Gleim, McHugh & Kjær, 1995). The purpose of the present study was to investigate if tissue properties and stretch tolerance changed as a result of a long-term stretching regimen in a human model using this measurement technique.

## METHODS

### Subjects

Seven female subjects volunteered to participate. The mean  $\pm$  s.d. for the age, body mass and height was  $26.0 \pm 6.0$  years,  $59.1 \pm 5.1$  kg and  $1.70 \pm 0.10$  m, respectively. The subjects were free of any lower extremity or lower back pathology. The subjects did not participate in any organized sporting activities and only occasionally participated in recreational sports. None of the subjects had performed any specific stretching regimen of the hamstring muscle group prior to entering the study. The study was approved by the local Ethics Committee and informed consent was obtained from the subjects.

### Measurement technique

Resistance to stretch was defined as the passive torque (N m) offered by the hamstring muscle group during passive knee extension using a KinCom dynamometer (Kinetic Communicator, Chattecx Corp., Chattanooga, TN, USA) with a modified thigh pad (Fig. 1). All measurements of passive torque were gravity corrected (Magnusson *et al.* 1995). Subjects were seated with the trunk perpendicular to the seat for the stretch procedure. The thigh rested on a specially constructed thigh pad elevating it to between  $0.524$  and  $0.785$  rad (range) from horizontal. The trunk and thigh position disallowed subjects to reach complete knee extension. Consequently, the position of the subject during the stretch manoeuvre placed tension primarily on the muscle-tendon unit without involvement of posterior capsular constraints about the knee. Passive force (N) was detected by the load cell of the dynamometer, which was calibrated prior to the experiment (10–750 N). The dynamometer and knee joint axis were aligned and the torque about the knee joint was calculated by multiplying the measured force by the lever arm distance. The lever arm attachment was placed 2 cm proximal to the lateral malleolus. The distal thigh and pelvis were firmly secured with straps in an attempt to minimize joint movement during the stretch manoeuvre. The load cell was calibrated with 98 N load to ensure that stress relaxation of the load cell did not occur. No stress relaxation occurred over 90 s and when manually unloaded the load cell returned from 98 N to baseline (0 N) in  $< 0.2$  s. Reliability of the method has been demonstrated; it yields a correlation coefficient of  $r = 0.99$  with a coefficient of variation of 6.5% with respect to passive torque (Magnusson *et al.* 1995).

Gross electrical activity of the human hamstring muscle group was measured with Ag–AgCl surface electrodes (Medicotest, Type N-10-A, Denmark) placed midway between the gluteal fold and the knee joint (McHugh *et al.* 1992), with a 3 cm inter-electrode distance. Custom-made amplifiers with a frequency response of between 20 and 10 kHz and 1:1 preamplifiers were used for EMG signal sampling. The mean EMG signal was full-wave rectified and integrated (time constant, 200 ms) (Basmajian & DeLuca, 1985).

### Protocol 1

Protocol 1 was administered to determine if tissue changes resulted from the stretching regimen. It consisted of a stretch phase to a predetermined final angle followed by a 90 s holding phase. The final angle during the stretch manoeuvre was determined by passively extending the knee to an angle which provoked a sensation of tightness in the posterior thigh similar to a hold stretch manoeuvre. Care was taken to avoid a painful response during determination of the final angle. The leg was then immediately returned to the starting position. The final angle was determined for the left and right side separately on the first test day. The same final angle was used for the pre- and post-training stretch manoeuvre. During protocol 1, the dynamometer was programmed to extend the knee passively at  $0.087$  rad  $s^{-1}$  from the starting point of  $1.222$  rad below horizontal to the final angle (stretch phase) where it remained for 90 s (holding phase) (Fig. 2). Throughout the stretch manoeuvre, subjects were requested to relax completely and not offer any voluntary resistance. After 90 s in the holding phase, subjects were requested to produce a maximal voluntary contraction (MVC) effort with the hamstring muscle to produce a flexion torque. This yielded an EMG and torque response which could be compared with that during the holding phase of the stretch manoeuvre.

### Protocol 2

Protocol 2 was administered to determine the stretch tolerance. For this part of the data collection, subjects were requested to close their eyes. The dynamometer extended the knee passively at  $0.087$  rad  $s^{-1}$  from the starting point of  $1.222$  rad below horizontal to the onset of pain, at which point the subjects were instructed to press a switch which instantaneously stopped the lever arm; the leg was immediately returned the starting position. Subjects were thoroughly instructed in this manoeuvre and were allowed to try the procedure to a point below the pain threshold prior to data collection. In addition, one test trial was performed prior to data collection. Throughout the stretch manoeuvre subjects were requested to relax completely and not offer any voluntary resistance.



Figure 1. Test position for stretch manoeuvre

1, platform for distal thigh; 2, thigh strap; 3, pelvic strap; 4, KinCom ankle attachment with load cell.

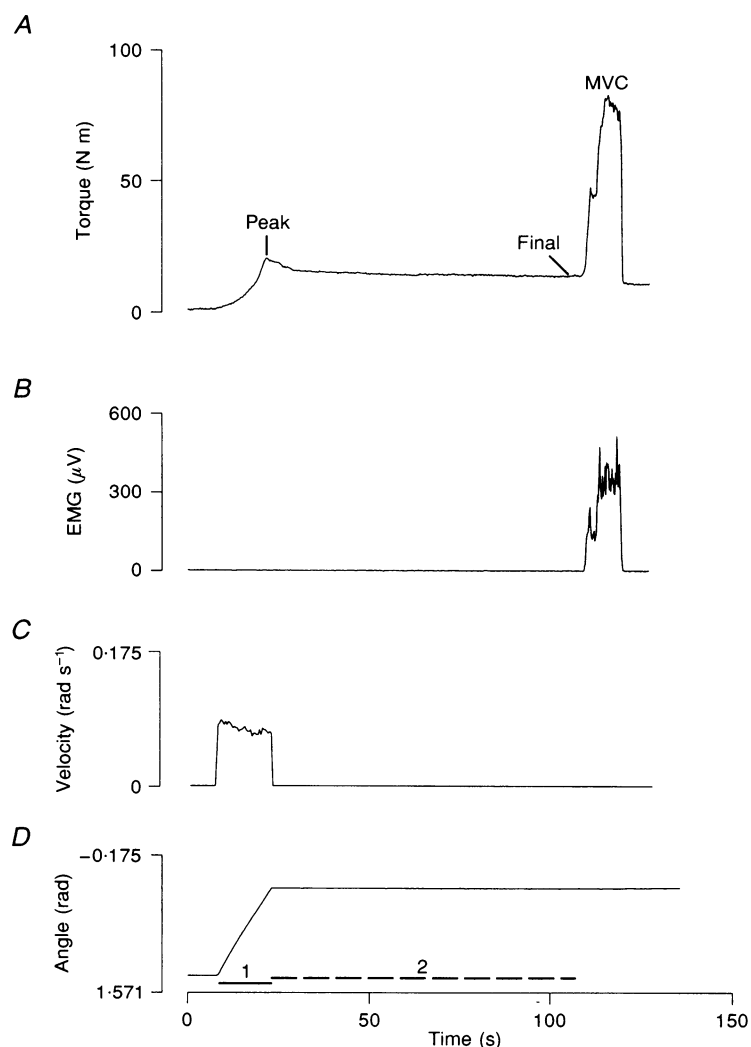
### Training regimen

The subjects were randomly assigned to stretch the hamstring muscle group on one leg while the opposite side served as a control. Two sessions, one in the morning and one in the afternoon, were performed on a daily basis for 20 consecutive days. Each session consisted of five stretches for 45 s with a 15–30 s rest in between. The total stretch stimulus was 9000 s, which to our knowledge exceeded any previously published study. The stretch manoeuvre was performed in the seated position with the stretch leg in a straight position in front of the subject and the hip in neutral rotation. The control leg was flexed at the knee and hip and slightly abducted to ensure that the hamstring muscle group was not under tension. Subjects were requested to lean forward with a straight back to a point where they experienced a 'stretch' sensation which was subsequently maintained for 45 s. Previous research has shown that a 45 s stretch in this position acutely yields a 29% stress relaxation (Magnusson *et al.* 1995). The subjects filled out a form on a daily basis to register compliance. They were instructed not to initiate any new forms of training.

Protocols 1 and 2 were administered to the left and right side of the subjects prior to (test 1) and after (test 2) the 3 week stretching period. The tests were performed at the same time on both test days. The subjects did not perform any stretch training on the day of test 2.

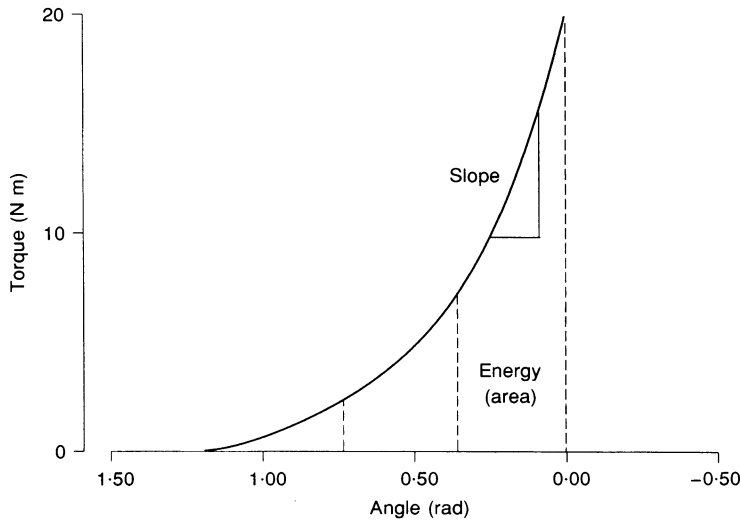
### Data reduction and analysis

Passive torque, joint range of motion, angular velocity and hamstring EMG were continuously recorded over the entire range of stretch manoeuvres. Signals were sampled at 50 Hz, A/D converted (DT 2801A, Data Translation), and stored on a PC for subsequent analysis. In the stretch phase in protocol 1 the total energy (Magnusson, Simonsen, Aagaard & Kjaer, 1996*b*) and the mean EMG amplitude were calculated. Further, the torque–angle curve was considered to contain three approximately even portions; an initial toe region, a transition period and a linear portion (Fig. 3). The slope, i.e. the stiffness, of the linear portion of the torque–angle curve was calculated (Magnusson *et al.* 1996*b*). In the holding phase, peak torque was obtained the instant the lever arm



**Figure 2.** Data for one subject from protocol 1

A shows the passive torque (gravity corrected) recording with the peak torque, final torque and maximal voluntary contraction (MVC). B, the corresponding EMG amplitude. Note the absence of activity despite a decline in torque from peak to final. C, velocity, which during the stretch phase is  $0.0875 \text{ rad s}^{-1}$ . D shows the angle (negative value indicates angles above horizontal) with its stretch phase (1, continuous line) and 90 s holding phase (2, dashed line) prior to MVC.



**Figure 3.** Data for one subject from the stretch phase of protocol 1

Data have been gravity corrected and a 4th order polynomial fit was employed. The area under the curve represents the potential energy return from the tissue. The stiffness, or elastic modulus, was calculated in the linear portion of the curve (labelled Slope on the curve).

reached the final angle, and final torque was obtained after 90 s in the holding phase.  $\Delta$ Torque was the decline in torque expressed as a percentage of peak torque. The mean EMG amplitude was calculated for the initial and final 5 s of the 90 s in the holding phase. Additionally, peak torque and EMG amplitude were calculated for the MVC. For protocol 2, maximal joint angle and corresponding peak torque were obtained at the point of pain. The area under the torque-angle curve (energy) and the mean EMG amplitude of the last 1 s prior to the onset of pain were calculated.

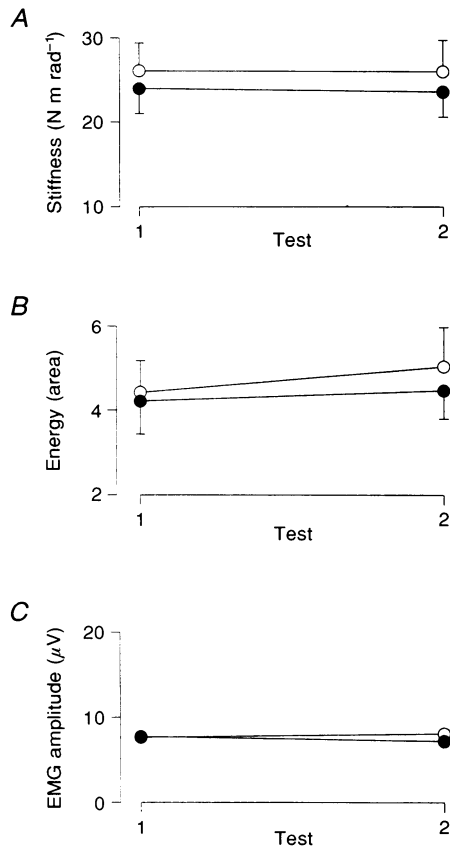
Wilcoxon matched pairs signed rank tests were used to determine whether differences existed between test 1 and test 2 for the

measured variables. An  $\alpha$  level of 0.05 was considered significant. Results are reported as the means  $\pm$  s.e.m.

## RESULTS

### Protocol 1

The results of variables examined in the stretch phase of protocol 1 are shown in Fig. 4. There were no significant differences in stiffness between the control ( $P = 0.86$ ) and stretch side ( $P = 0.86$ ). Similarly, no differences existed in energy (control side,  $P = 0.24$ ; stretch side,  $P = 0.61$ ) or in



**Figure 4.** Results from the stretch phase of protocol 1

No significant differences existed for stiffness (A), energy (B) or EMG (C) before and after the training period on the control (○) and the stretch side (●). Note that the EMG variation was negligible. Here, and in the following figures, vertical lines and bars indicate the s.e.m.

**Table 1. Initial and final 5 s EMG amplitudes ( $\mu\text{V}$ ) in the holding phase of protocol 1**

	Test 1		Test 2	
	Initial ( $\mu\text{V}$ )	Final ( $\mu\text{V}$ )	Initial ( $\mu\text{V}$ )	Final ( $\mu\text{V}$ )
Stretch side	$2.2 \pm 0.2$	$3.1 \pm 1.2$	$2.0 \pm 0.2$	$2.1 \pm 0.3$
Control side	$2.2 \pm 0.2$	$1.8 \pm 0.1$	$3.2 \pm 1.0$	$2.7 \pm 0.7$

mean EMG amplitude (control side,  $P = 0.68$ ; stretch side,  $P = 0.24$ ).

The knee joint torques in the holding phase are shown in Fig. 5. All stretches yielded a significant decline in torque ( $\Delta\text{torque}$ ,  $P = 0.018$ ). However, peak and final torques and  $\Delta\text{torque}$  remained unchanged before and after the training period. The EMG results from the holding phase are shown in Table 1. There were no significant changes in EMG amplitude within a stretch (initial vs. final EMG) or between tests (test 1 vs. test 2). The MVC resulted in a knee joint torque of  $64.7 \pm 5.8$  N m with a corresponding EMG amplitude of  $273.2 \pm 43.2 \mu\text{V}$ . The peak torque in the holding phase of the stretch represented  $23 \pm 4\%$  of the MVC torque. Initial and final EMG amplitude in the holding phase corresponded to  $0.9 \pm 0.2\%$  of the EMG amplitude during MVC.

**Protocol 2**

Figure 6 shows the results from protocol 2. There were no significant changes on the control side. On the other hand,

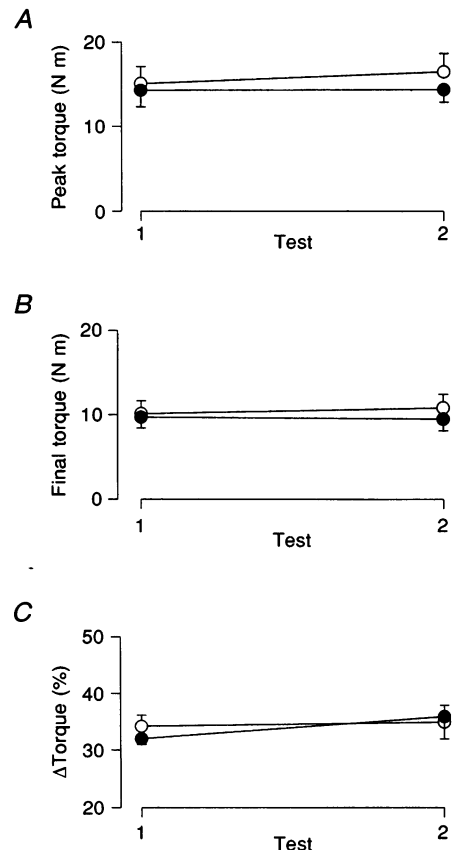
there were significant increases on the stretch side in maximal angle ( $P = 0.018$ ), peak torque ( $P = 0.018$ ) and energy ( $P = 0.018$ ) (Fig. 7). The EMG amplitude prior to pain was unchanged on the control side between test 1 and test 2 ( $2.8 \pm 0.7$  vs.  $3.1 \pm 0.6 \mu\text{V}$ ). Similarly, the EMG amplitude prior to pain did not differ on the stretch side between test 1 and test 2 ( $3.8 \pm 0.6$  vs.  $5.6 \pm 2.0 \mu\text{V}$ ).

The subjects completed  $94 \pm 1\%$  of the stretch sessions, i.e. the hamstring muscle group on the stretch side was subjected to a total of 8460 s of stretch stimulus during the training period. None of the subjects undertook any new forms of training.

**DISCUSSION**

The purpose of the present study was to determine whether tissue properties and stretch tolerance changed as a result of a 3 week regimen. No permanent change was observed in the tissue properties after the stretching period. On the other

**Figure 5. Results from the holding phase of protocol 1**  
No significant differences existed for peak torque (A), final torque (B) or  $\Delta\text{torque}$  (the decline in torque as a percentage of peak torque; %; C) before and after the training period on the control (O) and the stretch side (●).



hand, maximum joint range of motion and corresponding passive torque increased, which suggests that the mechanism for improved joint range of motion is an increased stretch tolerance rather than a viscoelastic accommodation.

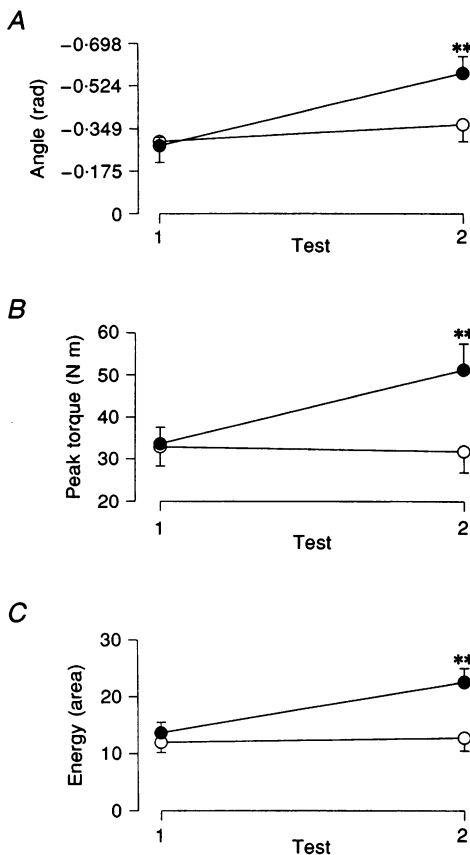
### Protocol 1

In the stretch phase, reduced stiffness and energy would be expected after the training period if the stretching regimen produced an enduring effect on the tissue properties of the hamstring muscle group. In animal models muscle stiffness and energy during stretch can change acutely with temperature increases (Strickler, Malone & Garrett, 1990; Noonan, Best, Seaber & Garrett, 1993) and stimulation (Garrett, Safran, Seaber, Glisson & Ribbeck, 1987) and chronically as a result of age and endurance training (Kovanen *et al.* 1984; Kovanen & Suominen, 1988). However, it has never been confirmed that tissue properties are affected by stretch training. In the present study, both stiffness in the final portion of the torque–angle curve and energy, which accounts for the entire torque–angle curve, were unaffected by the stretch training. In addition, in the holding phase peak, final and  $\Delta$ torque remained constant over the training period on both the control and training sides. Again, the low-level EMG response did not influence the results since it was constant over the training period on both sides.

The holding phase of protocol 1 confirms earlier observations of the acute effects of a single stretch in animal (Abbott & Lowy, 1956; Taylor *et al.* 1990) and human *in*

*vivo* models (McHugh *et al.* 1992; Magnusson *et al.* 1995). In the present study the low-level EMG response remained unchanged in the holding phase, which suggests that the 33–35% decline in torque about the knee joint was a tissue response. Further, since during the holding phase the EMG amplitude was below 1% and peak torque was 23% of that during MVC, it is unlikely that muscle activity contributed significantly to passive peak torque and its subsequent stress relaxation response. In contrast to the neurophysiological explanation (Hutton, 1993), the present data demonstrate that the acute effects of stretching in the holding phase is a viscoelastic accommodation rather than a decline in EMG activity.

Human models have shown that habitual stretching produces chronic increases in joint range of motion (Sady *et al.* 1982; Gajdosik, 1991; Bandy & Irion, 1994). Although unconfirmed, such increases are frequently attributed to a change in the tissue properties of the muscle (Gajdosik, 1991; Hutton, 1993). Tissue properties may be affected by repeated stretches (Magnusson *et al.* 1995; Magnusson *et al.* 1996*b*), but the change is transient in nature and the viscoelastic properties return to baseline values within 1 h (Magnusson *et al.* 1996*b*). In the present study the total stretching stimulus far exceeded that provided by previous studies but produced no lasting change in the viscoelastic properties of the muscle–tendon unit. It cannot be excluded that rigorous forms of stretching regimens, such as in ballet or gymnastics, over several years is a sufficient stimulus to yield a lasting change in the tissue properties.



**Figure 6. Results from protocol 2**

The stretching regimen resulted in significant increases on the stretch side (●) in maximal angle (A), peak torque (B) and energy (C),  $P = 0.018$ , while the control side (○) remained unchanged.

**Protocol 2**

After the training period the subjects reached a greater maximal joint angle on the stretch side while the control side was unchanged. Accompanying this increased angle was an increased peak torque, while the low-level EMG remained unchanged (Figs 6 and 7). Consequently, the present data suggest that the mechanism for increased joint range of motion as a result of habitual stretching is an altered stretch tolerance, while the tissue properties remain unaffected. It has been demonstrated that stretch tolerance may be altered acutely as a result of an isometric contraction, as evident by a 5 deg increase in joint angle and an accompanying increase in passive torque (Magnusson *et al.* 1996*a*). However, there have been no reports of such chronic changes in flexibility. In the present study the magnitude of the joint angle increase appears to have been greater (approximately 10 deg) than the acute increase (5 deg) previously reported. However, since the subjects did not perform any stretching on the test day after the training period it appears that the change was enduring in nature.

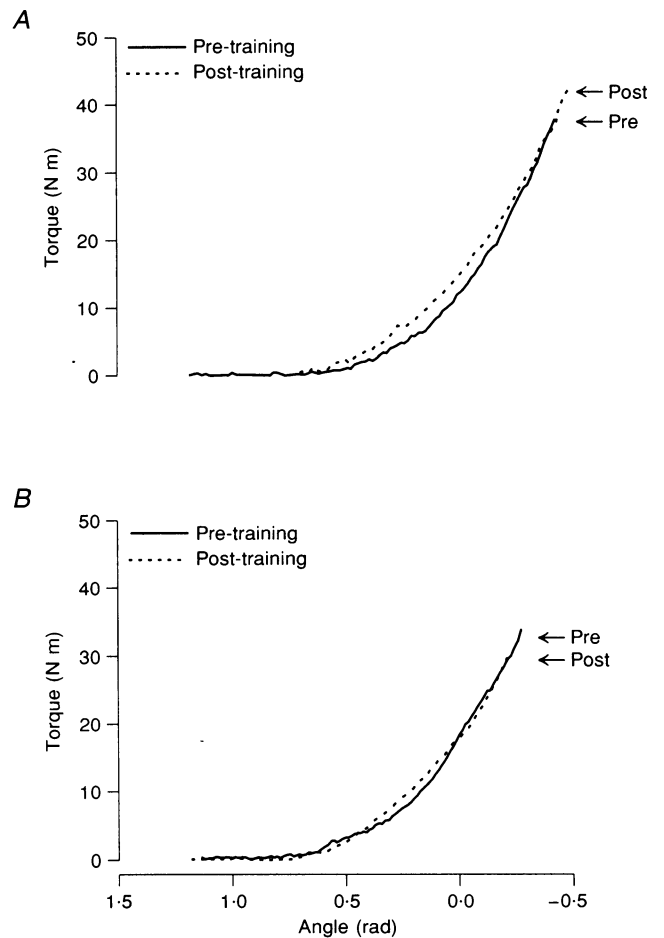
An increased joint range of motion with a concomitant increase in torque about the knee joint has been shown when the knee was brought to an angle which yielded  $> 50 \mu V$  before and after a 3 week training period

(Gajdosik, 1991). The results were attributed to an increase in muscle length. However, a change in the tissue property may only be concluded if a decrease in force was observed at the same joint angle, or if a greater joint angle was achieved with the same load. Therefore, the reported change in joint angle cannot be a change in length, but may be explained by an altered stretch tolerance. The present study demonstrates that increases in joint range of motion (protocol 2) can clearly be achieved without an accompanying change in the passive length–tension relationship (protocol 1, stretch phase) or viscoelastic stress relaxation (protocol 1, holding phase). Therefore, muscular flexibility is best defined as joint range of motion rather than stiffness, or compliance. The mechanism for an altered stretch tolerance is unknown. However, it is possible that nociceptive nerve endings in the joint and muscle play a role (Netter, 1983; Marchettini, 1993).

Although the total stretch stimulus in the present study far exceeded that of previous studies (Gajdosik, 1991; Bandy & Irion, 1994), the magnitude of the increased joint range of motion appears to have been similar. It has been shown that stretch durations of 30 and 60 s yield similar results over a 6 week training period (Bandy & Irion, 1994). Others (Hardy, 1985) have shown comparable increases in joint

**Figure 7.** Torque–angle data for 1 subject from protocol 2

*A*, the training side: note that after training the subject reached a greater knee joint angle prior to the onset of pain with an accompanying increase in torque. Further, it should be noted that the pre-training and post-training are similar with respect to the slope. *B*, the control side: the slope and the end point do not differ over the training period. The arrows indicate the end points for pre- and post-training.



range of motion over a 1 week period with a total stretch stimulus of merely 360 s. Consequently, it is possible that a stretch stimulus of considerably smaller duration could have produced a similar change in stretch tolerance, or that the observed changes occurred early in the training period.

### Conclusion

Protocols 1 and 2 were used to examine whether tissue properties and stretch tolerance, respectively, changed as a result of a 3 week stretching regimen in a human model. The result showed that the tissue properties were unchanged. On the other hand, stretch tolerance was augmented, as evidenced by the increased maximal joint angle and the accompanying increased knee joint torque. Therefore improved joint range of motion appears to be due to an increased stretch tolerance.

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