

Neuromuscular disorder in response to anterior cruciate ligament creep

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Abstract

Objective. To determine the effect of creep developed in the anterior cruciate ligament and other viscoelastic knee structures on the function of the flexor and extensor muscles of males and females.

Design. Static load applied to the proximal tibia of young healthy male and female subjects in a laboratory setting with maximal voluntary knee flexion and extension performed before and after the load application.

Background. Static loads applied to various joints during occupational and sports activities are epidemiologically linked to higher than normal rates of disability reports. The physiological and biomechanical processes active in the development of such a neuromuscular disorder are not known. We hypothesize that creep developed in the anterior cruciate ligament due to prolonged static load will have pronounced impact on the reflexive activation of the associated musculature in a manner that may increase the risk of injury. Females are expected to be exposed to higher risk than males.

Methods. Male and female groups performed maximal voluntary knee flexion and extension before and after applying 200 and 150 N, respectively, to the proximal tibia for a 10 min period. Flexion and extension forces as well as electromyograph from agonist and antagonist muscles were measured at 35° and 90° knee flexion. Data was analyzed through repeated measures of analysis of variance.

Results. It was found that in extension, quadriceps electromyographic activity increased significantly after anterior cruciate ligament creep while hamstrings co-activation did not change. There was also a trend towards increased extension force after creep was developed, with significant effect of gender (larger increase in females). Similarly, significant increase in hamstrings electromyographic activity and a trend towards increased force during knee flexion was observed but with no effect of gender. Electromyographic spasms from the flexors and extensors were recorded in 30% of the subjects during the 10 min static loading period at 90° angle and from the flexors only at 35°. Creep in the ligament was marginally greater in females than in males with a significant effect of angle, being greater at 35° than in 90° knee flexion.

Conclusions. The results suggest that ligament creep may develop a neuromuscular disorder consisting of spasms, increased electromyography and force in the agonist muscles without compensation from the antagonist. Static loading of a joint, therefore, may develop a neuromuscular disorder compounded with laxity of the ligaments and subject the individual to increased risk of injury.

Relevance

The data provides evidence that prolonged static loads applied to the anterior cruciate ligament and associated viscoelastic structures results in unbalanced muscular activation which puts individuals at increased exposure to injury. Work and sports activities should be scheduled while minimizing periods of static joint loading and emphasizing sufficient rest periods to allow recovery of creep and return to balanced muscular activation and co-activation.

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1. Introduction

In addition to its role as the primary restraint against anterior tibial displacement, the anterior cruciate ligament (ACL) plays a sensory role which has been widely reported (Baratta et al., 1988; Dyhre-Poulsen and Krogsgaard, 2000; Fujita et al., 2000; Raunest et al., 1996; Solomonow et al., 1987a; Solomonow and Krogsgaard, 2001; Tsuda et al., 2001). In earlier reports, this reflexive activity was thought to be purely protective of the ligament (Baratta et al., 1988; Solomonow et al., 1987a), however, the current view is that the ligament contributes to the overall sensorimotor control of the joint, with its neural input integrated with that of other sensory structures to control joint coordination during normal daily motion (Dyhre-Poulsen and Krogsgaard, 2000; Solomonow and Krogsgaard, 2001). Findings of reflex arcs originating in ligaments have been also reported in the elbow (Philips et al., 1997), shoulder (Guanche et al., 1995; Knatt et al., 1995), the ankle (Lewis et al., 1996), and most recently the spine (Solomonow et al., 1998). These reports and others point to the concept that ligamentous tissues possess mechanoreceptors, which when under tensile load, activate some muscles that decrease that tensile load, and simultaneously also inhibit other muscle groups that would increase the stress.

Ligaments and other soft tissues possess classical viscoelastic properties such as creep, hysteresis and time dependent stress–strain relations. If static tension is applied to a ligament over a period of time, the ligament will exhibit creep (stretch) such that when the tension is removed, the ligament will not retract immediately to its original length nor develop the same tension at a given length as before the creep developed. After the load is removed, a recovery period is required to attain the original length and stiffness. This recovery period has been reported to last anywhere from 1 to 24 h, depending on the ligament and the strain it reached at the end of loading (Crisco et al., 1997; Gedalia et al., 1999; McGill and Brown, 1992; Solomonow et al., 2000). Such creep is manifested as temporary laxity between the bony structures attached by the ligament in question (Crisco et al., 1997; Thornton et al., 1997; Ticker et al., 1996; Woo et al., 1981, 1999a). Static load applied to the supraspinous ligament was also shown to elicit spasms in the paraspinal muscles as creep was developing in the ligament (Jackson et al., 2001).

Recent reports have shown that ligamentous creep is also associated with desensitization of the reflex arcs initiated by the mechanoreceptors in the ligament (Jackson et al., 2001; Solomonow et al., 1999), diminishing the reflexive muscle activity and exposing the joint to further instability and potential injury. Clinically, the changes in the function of the muscles and ligaments constitute a neuromuscular disorder. The reflexive

muscular activity recovers in parallel with the recovery of the ligament's original length and tension (Solomonow et al., 2000). Therefore, it can be argued that in the case of ligamentous laxity due to creep, not only is the mechanical stability compromised, but the ability of viscoelastic tissues to signal changes in joint stability is also decreased. With desensitization of the ligament mechanoreceptors, the reflexive muscular response is impaired as well. Since this neuromuscular disorder diminishes with time, as the creep in the ligaments recovers to normal, it is transient in nature. Indeed, a large study in a homogeneous population supports this idea by finding an association between transient laxity and rate of injury (Acasuso-Diaz et al., 1993).

Reports in the literature indicate that women have more inherent joint laxity than men (Acasuso-Diaz et al., 1993; Borsa et al., 2000; Gannon and Bird, 1999; Larsson et al., 1987; Rozzi et al., 1999; Arendt and Dick, 1995), diminished joint proprioception (Rozzi et al., 1999), as well as increased risk of ACL injuries (Arendt and Dick, 1995; de Loes et al., 2000; Gwinn et al., 2000; Wojtys et al., 1998). Acasuso-Diaz et al. (1993) and Larsson et al. (1987) postulate that the increased risk of ligamentous injuries in women is associated with their increased joint laxity. Physical activities that can lead to temporary laxity are various sports and occupational activities. Several reports detail increased laxity following exercise (Crisco et al., 1997; de Loes et al., 2000; Skinner et al., 1986; Marfleet, 1991). Similarly, individuals who spend a majority of time in a partially flexed knee postures such as skaters, skiers, goal keepers, concrete workers, etc., have significantly more antero-posterior knee laxity than do workers who spend most of their time standing such as painters (Kivimaki et al., 1994). Also, ACL injuries are commonly thought to be associated with fatigue (Marfleet, 1991), which occurs with prolonged muscle activity. Given the connection between exercise and laxity, and between laxity and ligament injuries, it is conceivable that rather than muscular fatigue, the underlying mechanism of injury may be joint laxity resulting from ligamentous and capsular creep during exercise and the resulting diminished reflexive muscular activity. Muscle fatigue, if any, probably further compounds the problem by the reduced joint stiffness associated with lower muscle forces.

The relationship between ACL creep and agonist–antagonist muscle coordination is not known. Therefore, the objective of this report is to explore if a neuromuscular disorder of some of the knee musculature is elicited by an experimentally induced creep in the ACL of normal, healthy male and female subjects. We hypothesize that by altering the sensorimotor control of knee stability, creep in the soft tissues including the ACL and knee capsule will induce changes in coordination of the knee musculature.

2. Methods

2.1. Subjects and setup

Twenty healthy young adults (age 24.4 SD 4.5 years), including 10 men and 10 women participated in this study after giving their informed consent as approved by the Institutional Review Board. For electromyographic (EMG) recordings, the skin overlying their right rectus femoris and biceps femoris was cleansed and lightly abraded with rubbing alcohol prep pads, and disposable 1 cm diameter Ag/Ag–Cl electrodes were placed longitudinally over the muscle bellies between the motor point and the distal tendon with a 2.5 cm interelectrode distance. A similar reference electrode was placed over the fibular head. The electrodes were connected to a custom made differential amplifier with gain of 1000, common mode rejection ratio of 90 dB, and frequency pass-band between 6–500 Hz.

The subjects were seated in a specially designed chair, and the knee placed at either 35° or 90° of flexion. A strap across the distal thigh, and another strap across the pelvis held the subject firmly in position. After the knee was strapped in place, the shank was secured to a padded polypropylene cuff. The cuff was connected through a rod to a load cell in order to measure extension and flexion force. A thin rod was affixed with two-sided tape to the anterior tibia, and was used to connect to a low friction linear displacement potentiometer fixed to the thigh in order to estimate ACL creep via anterior tibial displacement. The line of action of the transducer

was parallel to the femur. The experimental setup is shown on Fig. 1. The two EMG signals, as well as the flexion/extension force, and the anterior tibial displacement signal were acquired by a 12 bit A/D board at a rate of 1000 samples per second and stored in the computer for further processing and analysis.

2.2. Protocol

After the subject was seated comfortably, their maximum voluntary contraction (MVC) force in isometric flexion and extension were determined in several serial trials in which the subject attempted to surpass the previous mark as set on an oscilloscope (Solomonow, 1996) with 4 min intertrial rest to prevent fatigue. Through these trials and subsequently, the subject held his/her arms folded across the chest in order to prevent from using their arms to improve their performance. Verbal encouragement was given through these trials. Maximal voluntary contraction force in flexion was assessed in a similar manner. The highest MVC forces in extension and flexion obtained by each subject was selected for the analysis. An anterior load was placed on the tibia via a padded strap attached to a cable guided over a pulley system. The creep in the weight and metal cable system was nil. The loads used were 200 N for males, and 150 N for females, and were held for 10 min while the subject was completely relaxed. Reduced loads were applied to females due to reduced body weight. The EMG and anterior tibial translation were recorded continuously. After the 10 min loading period, the



Fig. 1. A photograph of a subject during the loading period showing the static load applied to the tibia via a weight and a pulley, the load cell attached to the ankle, the displacement transducer attached to the knee and the EMG electrodes over the rectus femoris.

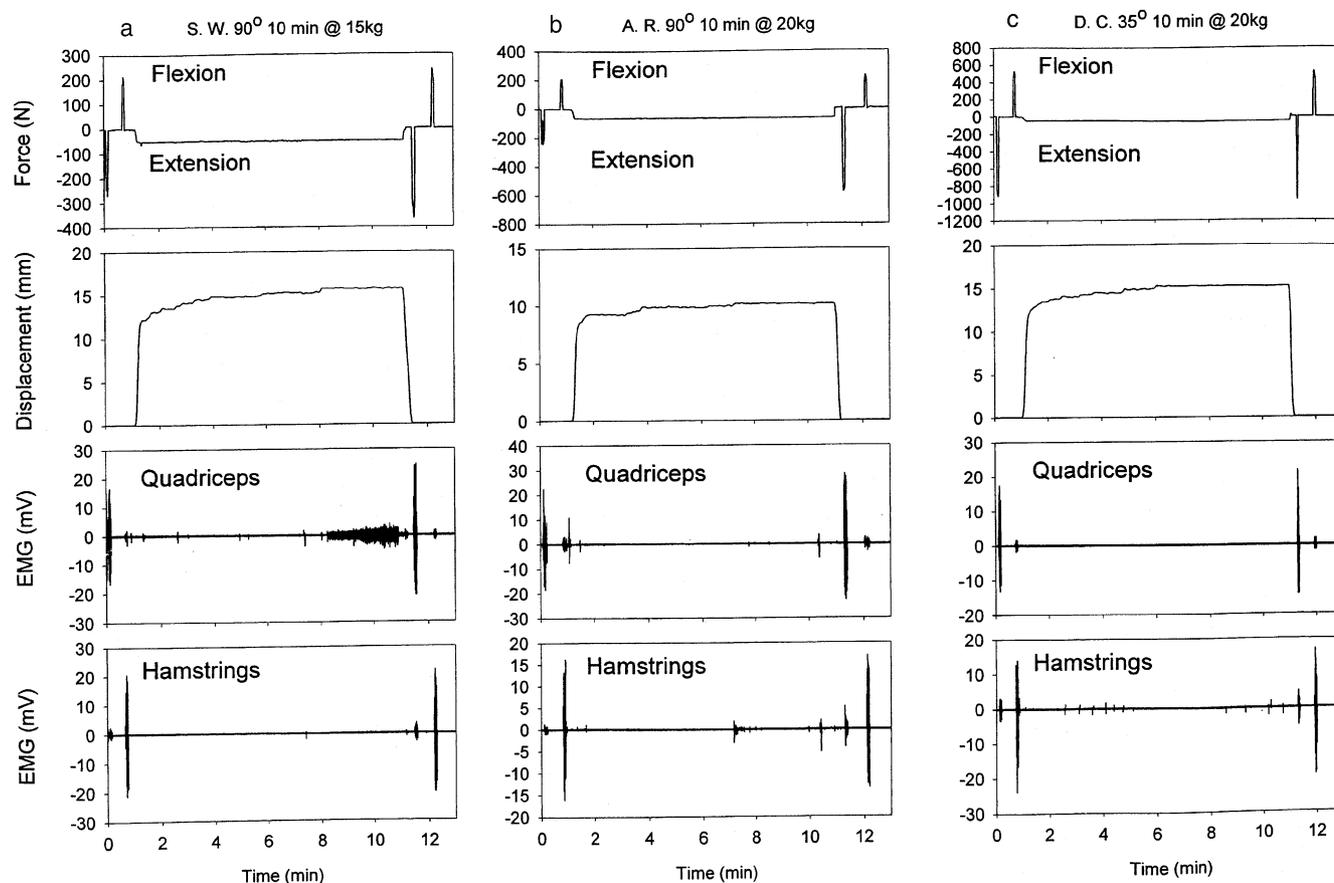


Fig. 2. (a, b, c) Three typical recordings from three different subjects at 90° and 35° knee angle showing the extension and flexion MVC forces before and after the 10 min loading session (top trace), the anterior displacement of the tibia during the 10 min loading period (second trace from top), quadriceps EMG (third trace) and hamstring EMG (bottom trace). Note the strong continuous burst of spasms in the quadriceps EMG trace of (a) from the 8th minute to the 11th minute. Similarly, in (b), two bursts of spasms are seen, one at about the 7th minute and the second just after the 10th minute, with a corresponding spasm in the quadriceps. In (c) short bursts of spasms are seen in the hamstrings EMG throughout the 10 min loading period.

Table 1
Summary of the mean (SD) normalized data of all subjects

	Male		Female	
	35°	90°	35°	90°
<i>Before creep</i>				
Quads agonist* EMG	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)
Quads antagonist EMG	0.14 (0.063)	0.16 (0.06)	0.15 (0.10)	0.17 (0.10)
Extension force*	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)
Hams agonist* EMG	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)
Hams antagonist EMG	0.27 (0.34)	0.33 (0.25)	0.25 (0.23)	0.24 (0.14)
Flexion force*	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)
<i>After creep</i>				
Quads agonist EMG	1.10 (0.29)	1.05 (0.28)	1.27 (0.48)	1.16 (0.32)
Quads antagonist EMG	0.14 (0.06)	0.14 (0.08)	0.16 (0.09)	0.17 (0.08)
Extension force	1.01 (0.14)	0.98 (0.13)	1.19 (0.37)	1.08 (0.18)
Hams agonist EMG	1.05 (0.22)	1.13 (0.32)	1.08 (0.27)	1.13 (0.20)
Hams antagonist EMG	0.29 (0.38)	0.34 (0.26)	0.27 (0.17)	0.29 (0.17)
Flexion force	1.01 (0.05)	1.20 (0.36)	1.05 (0.15)	1.00 (0.15)
Displacement (mm)	4.31 (1.73)	3.53 (1.85)	6.36 (2.67)	4.55 (1.53)

Items marked with * are normalizing factors and hence have a mean of 1.00 and standard deviation of 0.00.

subjects again performed a maximum voluntary isometric extension, waited 30 s and then a maximum isometric flexion. Subjects were requested to walk normally for a 30 m track, after the end of the test, report their impression of knee stability and then rest while supine on a mat for a 20 min period to allow recovery of the majority of the creep developed in the ACL.

The same protocol was repeated for each subject two weeks later at the second knee angle. The angle used initially was randomized amongst the subjects.

2.3. Signal processing and statistical analysis

The acquired EMG signals were processed via a mean absolute value (MAV) algorithm with a time window width of 200 ms. The algorithm consisted of an absolute value function applied to the digitally recorded signals, followed by averaging 100 points before and 100 points after the point in time over which the window was centered. The algorithm was applied from the 101st to the 11899th points in each signal. The peak force and peak MAV for each muscle in each contraction were used, yielding agonist and antagonist activity values for each muscle and peak flexion and extension forces. Then, peak force and MAV levels were normalized by dividing each value by the value obtained from the same muscle during its agonist function prior to the loading period (e.g., before ACL creep). In addition, the anterior tibial displacement from the linear potentiometer was divided into two parts: Initial displacement, calculated as the displacement occurring within 5 s of load application, and displacement due to creep, calculated as the difference in displacement at the end of the 10 min period minus the initial displacement. Analysis of variance with repeated measures was used to discern if there were effects of creep, angle and gender on the post-creep flexion and extension force, quadriceps agonist activity, quadriceps antagonist activity, hamstrings agonist activity and hamstrings antagonist activity.

3. Results

Typical recordings of quadriceps and hamstrings EMG signals, force and anterior displacement of the tibia from three different subjects at 35° and 90° knee flexion are shown in Fig. 2. In general, 12–15 mm of tibial translation occurred, 3–5 mm of which was due to creep that developed within 5 s after the load was applied. During the 10 min loading period random EMG discharge was evident in 6 (4 males, 2 females) out of the 20 subjects (e.g., 30%). The discharge amplitude, frequency and timing was unpredictable and conforms to the definition of spasms (e.g., non-voluntary, unpredictable muscle activity). The spasms were evident in the

EMG traces of the hamstrings and/or quadriceps at knee angles of 90°, but always in the hamstrings at knee angles of 35°. In the rest of the subjects the EMG traces during the loading period were quiet.

After the test, all subjects reported various levels of knee instability while walking over a 30 m track. In some subjects (1 male, 2 females) sensation of instability was accompanied with discomfort which disappeared after the 20 min rest period. In one female subject the sensation of instability lasted for 2 days after the test.

Table 1 shows the mean and standard deviations of the normalized data pooled from all the subjects. Since the trials at 35° and 90° of flexion were performed on different sessions, the data for each subject was normalized within session, and the statistical analysis refers to the *changes* from before to after creep, and not to absolute values. Hence, the MVC flexion and extension forces before ACL creep, as well as the corresponding agonist quadriceps and hamstrings EMG activities before ACL creep are set as normalizing factors, with mean value of 1.00, and standard deviation of 0.00. The EMG activity of the quadriceps (agonist) in MVC extension increased significantly following creep ($p \leq 0.01$), with no significant effect of angle and gender, indicating that the increase was similar at both angles in men and women. There were no changes noted in the hamstrings (antagonist) EMG co-activation due to ACL creep, gender or angle. There was an increase in force after ACL creep at both 90° and 35°, although the increases were trends ($p \leq 0.09$) with a significant effect of gender, indicating that women had greater increases than men. Increases were found for hamstrings (agonist) EMG activity during flexion in all groups post-creep, yielding a significant effect of creep ($p \leq 0.02$) with no effect of gender or angle. There was a trend to increased flexion force ($p \leq 0.06$), with no significant effect of gender or angle. The antagonist quadriceps co-activation EMG did not show any difference due to ACL creep, gender or angle (Table 2).

The results of the anterior displacement reflecting ACL creep demonstrate a significant effect of angle, with

Table 2
Results of the statistical analysis (p values)

Parameter	Effect of creep	Effect of angle	Effect of gender
Quads agonist activity	0.01	0.48	0.21
Quads antagonist activity	0.60	0.76	0.46
Extension force	0.09	0.34	0.04
Hamstrings agonist activity	0.02	0.89	0.41
Hamstrings antagonist activity	0.35	0.78	0.49
Flexion force	0.06	0.26	0.27
Anterior displacement	–	0.045	0.09

Results of ANOVA on the change from before creep to after creep values for each parameter.

Bold figures represent significance.

anterior displacement at 35° being greater than at 90° ($p \leq 0.05$), and a trend of women to have greater anterior displacement than men ($p \leq 0.09$).

4. Discussion

The most important finding in this study is that creep developed in the ACL due to static anterior load on the proximal tibia develops a neuromuscular disorder consisting of spasms and increased contractile ability of the flexors and extensors and in trends of a corresponding increase in their torques. Increased EMG activity and corresponding force in the flexors and extensors is not accompanied by compensation from the corresponding antagonist co-activation. The increased force and EMG activity is also more prominent in women than in men. It was also found that the anterior displacement of the tibia after the 10 min interval of loading was greater at 35° of flexion than at 90°, with trends of greater creep in women than in men.

The reader should note that the load applied to the knee via the tibia was limited to 150–200 N for safety and ethical reasons such that the subjects will not be exposed to high risk of knee injury yet the effect of the load will elicit some response. Under normal daily, sports and occupational activities much larger loads are sustained by the knee joint. An individual weighing 60 kg transmits his full weight through one knee when going up or down a staircase. Landing from a jump can result in up to 6 times body weight impact forces through the knee. Similarly, when loading/unloading cargo, worker's are subjected to more than body weight loads through the knee. The loads selected in this study, therefore, are light to mild in comparison to loads developed in sports and occupational activities and may have not generated the optimal responses with respect to creep magnitude or neuromuscular changes. Also, in the design and testing stages of this study, the first women tested complained of discomfort and feeling of instability caused by a 200 N load. Subsequently, we used a 150 N load in women. Previous studies comparing the laxity of men and women (Acasuso-Diaz et al., 1993; Borsa et al., 2000; Gannon and Bird, 1999; Larsson et al., 1987; Rozzi et al., 1999) demonstrated greater laxity in the females when compared with similar anterior loads; our study showed gender-related trends, which would have been significant had we used the same load for both males and females. For safety reasons, reduced loads were applied to females, yet the results confirm their greater susceptibility to the effects of creep.

A finding that appeared in both flexion and extension was that the agonist muscles increased their activity levels significantly, with no matching changes in antagonist co-activation. The end results were increases in maximal flexion and extension forces. One could assert

that the creep in both the ACL and the capsular structures induced by the 10 min loading period reduced the neural inhibition of the agonist muscle, allowing greater maximum contractile activity, and hence a greater force output. This explanation supports the concept that ligaments and capsular viscoelastic structures that have undergone creep are neurologically desensitized. Earlier studies focused on reflexive activation of muscles primarily responsible for reducing ligamentous or capsular loads (Baratta et al., 1988; Dyhre-Poulsen and Krogsgaard, 2000; Raunest et al., 1996; Solomonow et al., 1987a; Solomonow and Krogsgaard, 2001). The present study provides evidence that changes in the mechanical properties of ligaments can also have significant neurophysiological effect manifested by increased contraction intensity of the agonist musculature as well, primarily by reducing its neural inhibition. Recent work with the cruciate ligaments of humans supports the assertion that ligaments may have inhibitory effect on muscles under conditions which do not challenge joint or ligament stability (Dyhre-Poulsen and Krogsgaard, 2000; Solomonow and Krogsgaard, 2001; Fischer-Rasmussen et al., 2001). The reduced inhibitory effect of a ligament subjected to creep, however, should be considered as a neuromuscular disorder since it is likely to create a risk of injury. Increase quadriceps force, for example, in the range of 0–60° of flexion will increase the loads on the already lax ACL and may result in its rupture.

EMG spasms were observed in 30% of the subjects participating in this study. Spasms are defined as a non-volitional random and unpredictable muscular activity which indicate tissue damage (Pederson et al., 1956). The spasms appeared in the hamstring and/or quadriceps when the ACL was loaded at a knee angle of 90°. At this angle, the ACL can benefit from reduction in stress if the hamstrings or quadriceps contract (Hirokawa et al., 1991, 1992; Renstrom et al., 1986). At knee angles of 35°, however, spasms appeared only in the hamstrings. Knee angles near 35° were shown to induce maximal strain in the ACL, with additional increase in strain if the quadriceps contract Hirokawa et al., 1991, 1992; Renstrom et al., 1986). Contraction of the hamstrings at this angle has a direct effect on reducing the anterior displacement of the tibia and therefore, the strain in the ACL (Hirokawa et al., 1991; Renstrom et al., 1986). Hamstrings spasms at 35° of knee flexion, therefore, reduce the stress/strain in the ACL whereas spasms in the quadriceps and/or hamstrings will have the same effect in a more flexed position, mostly at angles larger than 60–70° flexion (Hirokawa et al., 1991; Renstrom et al., 1986). The fact that spasms were apparent in 30% of the subjects further emphasizes the issue of necessary and sufficient stimulus. The loads applied, as indicated before, were mild due to safety and ethical considerations. With larger loads, one could anticipate spasms in most subjects.

The fact that spasm appeared in some of the subjects points out that some type of injury or damage was elicited in the ACL due to the creep developed within. Most likely the damage was in the bonding of the collagen molecules (Thornton et al., 1997; Woo et al., 1981) which in turn stimulated the pain receptors in the ligament and in turn triggered the spasms. The fact that spasms are triggered by damage to ligaments was experimentally documented (Pederson et al., 1956) and well known clinically. Creep of ligamentous tissues, therefore, may be associated with a transient damage.

The results we obtained have significant implications. Athletes or workers who spend considerable time in positions in which the ACL is under significant static tensile load may be at increased risk of ligament injury. This increased risk is not simply due to the mechanical aspect of ACL creep, but because of the decreased sensory ability of the ligaments to signal their stress state. This phenomenon is analogous to the desensitization of spinal ligaments after prolonged cyclical (Solomonow et al., 1999) or static (Jackson et al., 2001) lumbar flexion. The viscoelastic nature of the ligaments also mean that given a reasonable recovery time where the ligament is free from a tensile load, it will regain its original length and tautness. Crisco et al. (1997) found that after cyclic loads, 2 h were not sufficient to recover the ligamentous stiffness following exercise. Measurements taken 24 h later, however, showed complete recovery. Similarly, models of creep and reflexive activity recovery in the spinal ligament–muscle complex suggest that the majority of the creep recovers in the first 30 min of rest (McGill and Brown, 1992) but 24 h are necessary to recover completely (Crisco et al., 1997; Solomonow et al., 2000). It can hence be speculated that 24 h may be needed in order to allow complete recovery from ligamentous creep in the knee as well. Rest periods, therefore, are crucial for the safety of joints.

There are several reports of a relationship between muscle fatigue and ligament injuries, sometimes attributed to overuse. Without direct links, several of these reports cite fatigue as a causal agent of injuries (Marfleet, 1991; Pettrone and Ricciardelli, 1987; Troup, 1984). Other reports cite increases in ligament laxity following exercise (Crisco et al., 1997; Larsson et al., 1987; Skinner et al., 1986) or other prolonged occupational activities (Kivimaki et al., 1994). It may be suggested that the perceived and reported increase in susceptibility to injury attributed to fatigue may in fact be due to increased ligamentous laxity, and the resulting decreased sensitivity of sensory tissue signaling. Upon impending instability, the musculature is slow or unable to react reflexively—not due to fatigue, but because of lack of afferent input signaling the instability.

Among the notable findings in this study is the trend towards increased anterior displacement in women, and exacerbated increase in anterior force in women fol-

lowing ACL creep. These findings may help explain the reported higher incidence of injuries in females, since they are more likely to increase their agonist muscle activities as a response to ligamentous laxity, and are more prone to ligamentous sensory desensitization; their knees are rendered more likely to be injured during exercise.

Statistically, the increases in maximal flexion and extension force after the ACL was subjected to creep were trends ($p < 0.09$). Simultaneously, the respective agonist muscles, the quadriceps and hamstrings, demonstrated significant increase ($p < 0.05$) in EMG level during maximal extension and flexion, respectively. Since the EMG vs. force relationship may be non-linear, the increases in force were not as pronounced as that of the EMG (Solomonow et al., 1987b). Again, with larger loads on the knee, one could anticipate a more significant increase in force.

The results suggest that relatively short static loading of the ACL may cause a transient neuromuscular disorder that may increase the risk of knee injury. The data available from recovery studies (Crisco et al., 1997; Gedalia et al., 1999; McGill and Brown, 1992; Solomonow et al., 2000) strongly supports the concept that athletes or workers should allow at least a 24-h of rest between intense periods of activities that exposes the ligaments to creep. If repetition of such activities does not include sufficient rest time to allow the ligament to restore itself to its normal resting length, periods of activity will begin with residual creep that may compound the problem. Frequent repetitions of activity without allowing ligaments to recover to their resting length may develop a cumulative trauma disorder (Kumar, 1990), chronic inflammation and long term disability (Safran, 1985; Woo et al., 1999b).

In summary, the results of this study suggest that ligament creep has a significant effect on neuromuscular functions, moreover, that this effect is a decrease of agonist inhibition, as evidenced in increased agonist muscle activities and resultant forces without change in antagonist muscle co-activation. It is suggested that prolonged ligament tension subjects joints to increased risk of instability and potential injury due to its own laxity, and by increased agonist activity without compensatory antagonist co-activation.

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References

- Acasuso-Diaz, M., Collantes-Estevez, E., Sanchez-Guijo, P., 1993. Joint hyperlaxity and musculoligamentous lesions: study of a population of homogeneous age, sex and physical exertion. *British Journal of Rheumatology* 32, 120–122.
- Arendt, E., Dick, R., 1995. Knee injury patterns among men and women in collegiate basketball and soccer: NCAA data and review of literature. *American Journal of Sports Medicine* 23, 694–701.
- Baratta, R.V., Solomonow, M., Zhou, B., Letson, D., Chuinard, R., D'Ambrosia, R., 1988. Muscular coactivation: the role of the antagonist musculature in maintaining joint stability. *American Journal of Sports Medicine* 16, 113–122.
- Borsa, P.A., Sauer, E.L., Herling, D.E., 2000. Patterns of glenohumeral joint laxity and stiffness in healthy men and women. *Medicine and Science in Sports and Exercise* 32, 1685–1690.
- Crisco, J.J., Chelikani, S., Brown, R.K., Wolfe, S.W., 1997. The effects of exercise on ligamentous stiffness in the wrist. *Journal of Hand Surgery* 22A, 44–48.
- de Loe, M., Dahlstedt, L.J., Thomee, R., 2000. A 7-year study on risks and costs of knee injuries in male and female youth participants in 12 sports. *Scandinavian Journal of Medicine and Science in Sports* 10, 90–97.
- Dyhre-Poulsen, P., Krogsgaard, M.R., 2000. Muscular reflexes elicited by electrical stimulation of the anterior cruciate ligament in humans. *Journal of Applied Physiology* 89, 2191–2195.
- Fischer-Rasmussen, T., Krogsgaard, M., Jensen, D., Dyhre-Poulsen, P., 2001. Inhibition of dynamic thigh muscles contraction by electrical stimulation of the posterior cruciate ligament in humans. *Muscle and Nerve* 24, 1482–1488.
- Fujita, I., Nishikawa, T., Kambic, H.E., Andrish, J.T., Grabner, M.D., 2000. Characterization of hamstring reflexes during anterior cruciate ligament disruption: in vivo results from a goat model. *Journal of Orthopaedic Research* 18, 183–189.
- Gannon, L.M., Bird, H.A., 1999. The quantification of joint laxity in dancers and gymnasts. *Journal of Sports Sciences* 17, 743–750.
- Gedalia, U., Solomonow, M., Zhou, B., Baratta, R.V., Lu, Y., Harris, M., 1999. Biomechanics of increased exposure to lumbar injury due to cyclic loading: II. Recovery of reflexive muscular stability with rest. *Spine* 24, 2461–2467.
- Guanche, C., Knatt, T., Solomonow, M., Lu, Y., Baratta, R., 1995. The synergistic action of the capsule and shoulder muscles. *American Journal of Sports Medicine* 23, 301–306.
- Gwinn, D.E., Wilkens, J.H., McDevitt, E.R., Ross, G., Kao, T.C., 2000. The relative incidence of anterior cruciate ligament injury in men and women at the United States Naval Academy. *American Journal of Sports Medicine* 28, 98–102.
- Hirokawa, S., Solomonow, M., Luo, Z., Lu, Y., D'Ambrosia, R., 1991. Muscular co-contraction and control of knee stability. *Journal of Electromyography and Kinesiology* 1, 199–208.
- Hirokawa, S., Solomonow, M., Lu, Y., Lou, Z., D'Ambrosia, R., 1992. Anterior-posterior and rotational displacement of the tibia elicited by quadriceps contraction. *American Journal of Sports Medicine* 20, 299–306.
- Jackson, M., Solomonow, M., Zhou, B.-H., Baratta, R.V., Harris, M., 2001. Multifidus EMG and tension-relaxation recovery after prolonged static flexion. *Spine* 26, 715–723.
- Kivimaki, J., Hanninen, K., Kujala, U.M., Osterman, K., Riihimaki, H., 1994. Knee laxity in carpet and floor layers and painters. *Annales Chirurgiae et Gynaecologiae* 83, 229–233.
- Knatt, T., Guanche, C., Solomonow, M., Lu, Y., Baratta, R., Zhou, B.-H., 1995. The glenohumeral-biceps reflex. *Clinical Orthopaedics and Related Research* 314, 247–252.
- Kumar, S., 1990. Cumulative loads as a risk factor for back pain. *Spine* 12, 1311–1316.
- Larsson, L.G., Baum, J., Mudholkar, G.S., 1987. Hypermobility: features and differential incidence between the sexes. *Arthritis and Rheumatism* 30, 1426–1430.
- Lewis, J., Donatto, K., Solomonow, M., Zhou, B., Lu, Y., Baratta, R., Zhu, M., 1996. Ligamento-muscular protective reflex in the feline ankle. *Orthopaedics International* 4, 359–365.
- Marfleet, P., 1991. Ultimate injuries: a survey. *British Journal of Sports Medicine* 25, 235–240.
- McGill, S., Brown, S., 1992. Creep response of the lumbar spine to prolonged full flexion. *Clinical Biomechanics* 7, 43–46.
- Pederson, H., Blunk, C., Gardner, E., 1956. The anatomy of lumbosacral posterior rami and meningeal branches of spinal nerves. *Journal of Bone and Joint Surgery* 38A, 377–391.
- Pettrone, F.A., Ricciardelli, E., 1987. Gymnastic injuries: the Virginia experience 1982–1983. *American Journal of Sports Medicine* 15, 59–62.
- Philips, D., Petrie, S., Solomonow, M., Zhou, B.-H., Guanche, C., D'Ambrosia, R., 1997. Ligamento-muscular protective reflex in the elbow. *Journal of Hand Surgery* 22A, 473–478.
- Raunest, J., Sager, M., Burgener, E., 1996. Proprioceptive mechanisms in the cruciate ligaments: an electromyographic study on reflex activity in the thigh muscles. *Journal of Trauma-Injury Infection and Critical Care* 41, 488–493.
- Renstrom, P., Arms, S., Stanwyck, T., et al., 1986. Strain within the ACL during hamstrings and quadriceps activity. *American Journal of Sports Medicine* 14, 83–87.
- Rozzi, S.L., Lephart, S.M., Gear, W.S., Fu, F.H., 1999. Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. *American Journal of Sports Medicine* 27, 312–319.
- Safran, M., 1985. Elbow injuries in athletes: a review. *Clinical Orthopaedics and Related Research* 310, 257–277.
- Skinner, H.B., Wyatt, M.P., Stone, M.L., Hodgdon, J.A., Barrack, R.L., 1986. Exercise-related knee joint laxity. *American Journal of Sports Medicine* 14, 30–34.
- Solomonow, M., 1996. Standards for reporting EMG. *Journal of Electromyography and Kinesiology* 6 (III–IV).
- Solomonow, M., Krogsgaard, M., 2001. Sensorimotor control of knee stability. A review. *Scandinavian Journal of Medicine and Science in Sports* 11, 64–80.
- Solomonow, M., Baratta, R.V., Zhou, B., Shoji, H., Bose, W., Beck, C., D'Ambrosia, R., 1987a. The synergistic action of the ACL and knee muscles in maintaining joint stability. *American Journal of Sports Medicine* 15, 207–218.
- Solomonow, M., Baratta, R.V., Zhou, B., Shoji, H., D'Ambrosia, R., 1987b. The EMG-force model of electrically stimulated muscle: dependence on control strategy and fiber type. *IEEE Transactions on Biomedical Engineering* 34, 592–703.
- Solomonow, M., Zhou, B.-H., Harris, M., Lu, Y., Baratta, R.V., 1998. The ligamento-muscular stabilizing system of the spine. *Spine* 23, 2552–2561.
- Solomonow, M., Zhou, B.-H., Harris, M., Lu, Y., Baratta, R.V., 1999. Biomechanics of increased exposure to lumbar injury due to cyclic loading I: Loss of reflexive muscular stabilization. *Spine* 24, 2426–2434.
- Solomonow, M., Zhou, B.-H., Baratta, R., Lu, Y., Zhu, M., Harris, M., 2000. Biexponential recovery model of lumbar viscoelastic creep and reflexive muscular activity after prolonged cyclic loading. *Clinical Biomechanics* 15, 167–175.
- Thornton, G.M., Oliynyk, A., Frank, C.B., Shrive, N.G., 1997. Ligament creep cannot be predicted from stress relaxation at low

- stress: a biomechanical study of the rabbit medial collateral ligament. *Journal of Orthopaedic Research* 15, 652–656.
- Ticker, J.B., Bigliani, L.U., Soslowky, L.J., Pawluk, R.J., Flatow, E.L., Mow, V.C., 1996. Inferior glenohumeral ligament: geometric and strain-rate dependent properties. *Journal of Shoulder and Elbow Surgery* 5, 269–279.
- Troup, J.D., 1984. Causes, prediction and prevention of back pain at work. *Scandinavian Journal of Work, Environment and Health* 10, 419–428.
- Tsuda, E., Okamura, Y., Otsuka, H., Komatsu, T., Tokuya, S., 2001. Direct evidence of the anterior cruciate ligament–hamstring reflex arc in humans. *American Journal of Sports Medicine* 29, 83–87.
- Wojtys, E.M., Huston, L.J., Lindenfeld, T.N., Hewett, T.E., Greenfield, M.L., 1998. Association between the menstrual cycle and anterior cruciate ligament injuries in female athletes. *American Journal of Sports Medicine* 26, 614–619.
- Woo, S.L., Gomez, M.A., Akeson, W.H., 1981. The time and history-dependent viscoelastic properties of the canine medial collateral ligament. *Journal of Biomechanical Engineering* 103, 293–298.
- Woo, S.L., Debski, R.E., Withrow, J.D., Jansushek, M.A., 1999a. Biomechanics of knee ligaments. *American Journal of Sports Medicine* 27, 533–543.
- Woo, S., Aprelena, M., Hoher, J., 1999b. Tissue Mechanics of Ligaments and Tendons. In: Kumar, S. (Ed.), *Biomechanics in Ergonomics*. Taylor & Francis, Philadelphia.