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Passive muscle tension augments the anterior cruciate ligament

An in vivo study in the rat

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The contribution by passive muscle tension to the structural load-bearing capacity of the anterior (cruciate) ligament in the anesthetized rat was investigated. Using a stereomicroscope, the joint capsule and ligaments of the right knee, except the anterior ligament, were cut and the menisci removed leaving the anterior ligament and the tendons of the denervated muscle to constrain the knee. The ligament was tested in tension until failure, using a loading rate of 2.5 mm s^{-1} (~ 0.6 s^{-1}). As a control, the femur-anterior ligament-tibia complex of the left knee was tested. The mean ultimate tensile load on the anterior ligament augmented by muscle tendons was 48 percent higher. The energy absorption at failure was 84 percent higher; and the stiffness 26 percent higher. The deformation remained unchanged. This investigation suggests that, when the strain rate is high, muscle may be passively stretched and thus absorb energy and increase the force needed to rupture the anterior ligament.

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The knee must rely on protective mechanisms in addition to its ligaments because the load during vigorous activities substantially exceeds ligament strength (Kuo et al. 1983, Woo et al. 1991). Hamstrings and gastrocnemius co-contraction have been shown to protect the anterior cruciate ligament against rupture in an in vivo rat model (Aune et al. 1994). But the effect of muscle protection in a trauma situation depends upon the magnitude of muscle contraction at the instant of injury (Pope et al. 1979). In some injury circumstances muscles are already contracting at a significant level. In other circumstances they are minimally contracted and, despite the existence of a reflex arc between ligament strain and hamstring contraction (Solomonow et al. 1987, Miyatsu et al. 1993), the reaction time from loading until the hamstring contract may be too long to produce protective muscle contraction (Pope et al. 1979, Solomonow et al. 1987). However, the passive, noncontractile properties of muscles are always present.

The aim of this investigation was to study whether passive muscle is tensioned sufficiently to augment the anterior ligament loaded in tension to failure.

Animals and methods

10 male Wistar/Han/Mol SPF rats (Møllegård, Copenhagen) with a mean weight of 423 (410–436) g were used. The animals were anesthetized with a combination of Hypnorm (fluanisone 5 mg/mL-fentanyl citrate 0.1575 mg/mL, Jansen Pharmaceutica BV, Beerse, Belgium) and Dormicum (midazolam 2.5 mg/mL, Hoffmann-La Roche, Basel, Switzerland). The dose was 0.2 mL/100 g body weight injected subcutaneously. The experiment conformed to the Norwegian Council's Research Code for the Care and Use of Animals for Experimental Purposes.

An arthrotomy of the right knee was performed through a medial parapatellar incision, using a stereomicroscope (Nikon SMZ-2T, Japan). The patella was dislocated laterally, the medial collateral ligament divided and the medial meniscus removed. The cruciate ligaments were identified, and the posterior cruciate was cut at its femoral insertion. Then the posterior capsule and the lateral collateral ligament and capsule were divided and the lateral meniscus was removed, leaving only the anterior ligament, hamstring, gastrocnemius and patellar tendons intact. The patella was reduced and a 2 mm pin was drilled from the point of insertion of the medial ligament transversely through the lateral femoral con-

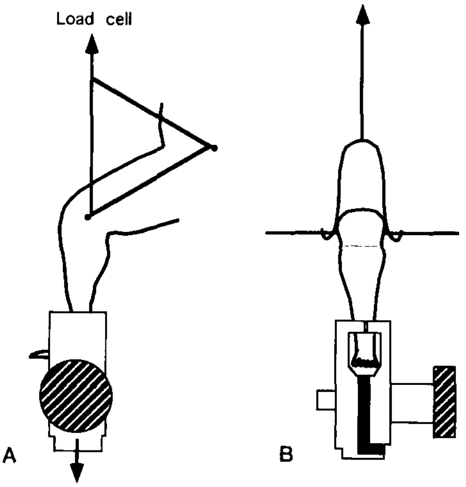

Figure 1. The experimental test used to evaluate the in vivo load-bearing capacity of the anterior cruciate ligament augmented by passive tension from knee flexor and extensor tendons. A. Lateral view. B. Frontal view.

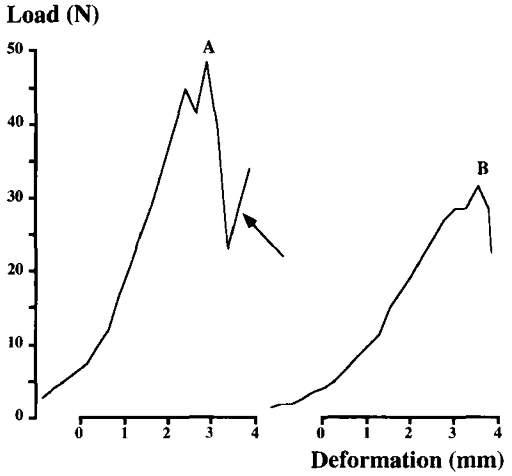

Figure 2. Typical load-deformation curves demonstrating the differences between the structural capacity of the rat anterior cruciate ligament tested with the muscles intact (A), and with tendons cut in a femur-tibia complex (B). The arrow indicates the load elongation curve for the passive muscles after ligament failure.

Table 1. Results for the right anterior cruciate ligament tested in tension in the intact rat hindlimb augmented by passive muscle tendons and for the left ligament tested in a femur-ligament-tibia complex. Mean, *SD*

Muscle augmented	Ultimate tensile load N	Energy absorption at failure J×10^{-2}	Linear stiffness N/mm	Ultimate deformation mm
Yes	45.8 *7.7*[a]	9.4 *2.4*[b]	19.8 *4.0*[c]	3.2 *0.6*
No	31.0 *3.8*	5.1 *1.0*	15.2 *2.2*	2.7 *0.4*

[a] *P* 0.001, [b] *P* 0.004, [c] *P* 0.03

dyle. Another pin was drilled into the pelvis just above the hip joint. To ensure muscle relaxation, the ischiatic nerve was cut behind the hip joint and the femoral nerve was cut in the groin. The rat was suspended in a coat, leaving the right hindlimb free. A clamp was fixed to the lower leg with the ankle in its neutral position of 90°. A triangle to fix the hip and keep the knee flexed at 60° connected the transcondylar and the pelvic pin to a modular test apparatus (Figure 1). The ligament was tested in tension at a loading rate of 2.5 mm s^{-1} (~ 0.6 s^{-1}). The left knee was used as a control and loaded correspondingly with the muscle tendons cut, leaving only the anterior ligament in a femur-tibia complex.

The load in the test apparatus was measured with a load cell connected to a microcomputer via an amplifier (Nordsletten and Ekeland 1993). The load-deformation curves were recorded on-line in WorkBenchMac (Strawberry Tree Incorporated, Sunnyvale, CA, U.S.A.).The failure mode was classified using the stereomicroscope.

Student's paired *t*-test (two-tailed) was used for statistical evaluation. $P < 0.05$ was considered significant.

Results

Passive muscles had a pronounced effect on the structural capacity of the anterior ligament. The deformation to ligament failure elongated the muscles sufficiently to create tension, and at the moment of failure, the load decreased rapidly to represent that of muscle tension (Figure 2). All ligaments failed in their substance. The mean ultimate tensile load for the ligament augmented by muscle tendons was 48 percent higher than for the femur-ligament-tibia complex. The energy absorption at failure was 84 percent higher and linear stiffness 26 percent higher. The deformation remained unchanged (Table 1).

Discussion

The method gave reproducible results with a ligament mode of failure in all knees tested. The strength of the anterior cruciate depends on the orientation of the specimen during testing. The knee flexion angle was therefore kept constant at 60°. At this flexion angle, the ligament was loaded axially as observed after resecting the medial femoral condyle in a pilot study, thus testing in the orientation of maximum strength (Woo et al. 1991).

The strength of ligaments and tendons depends on the loading rate (Noyes et al. 1974). Crowninshield and Pope (1976), calculating the strain rates during a skiing injury mechanism on the basis of a rat model, found that strain rates beyond 0.5 s^{-1} probably occurred during ligament injuries. Our loading rate of 2.5 mm s^{-1} was equivalent to a strain rate of about 60 percent per sec for the anterior cruciate ligament of 20-week-old male Wistar rats.

The knee and hip flexion angle during testing conformed to the habitual flexed position of the rat hindlimb (Evans et al. 1960), keeping the muscles at their resting length. At their resting length, muscles do not produce any passive tension (Crawford and James 1980). When the muscle is stretched beyond its resting length, passive tension develops in the parallel and serial elastic components (Keele et al. 1982). Our experiment showed that, during deformation to cruciate failure, the muscles are elongated sufficiently to elicit passive tension. Monoarticular muscles are not stretched enough for the passive tension to play any important role (Crawford and James 1980). For biarticular muscles the extremes of the length-tension relationship may be reached (Crawford and James 1980). For example, when a skier falls backwards from loss of balance, the knee is flexed and the hip extended elongating the rectus femoris. Trauma situations with hip and dorsal foot flexion and knee extension may tension the gastrocnemius and hamstrings substantially and thus increase the muscular augmentation of the knee.

On the basis of in vitro studies of the anterior cruciate ligament, an inherent biological safety margin of a 1:5 ratio of normal activity force to ultimate load has been proposed (Noyes et al. 1974). In vitro studies are incomplete as this biological safety margin is higher in vivo due to contracting muscles (Aune et al. 1994). The results of our experiment imply that muscle protection should be considered, even in the absence of contraction at the instant of injury.

References

Aune A K, Nordsletten L, Skjeldal S, Madsen S E, Ekeland A. Hamstrings and gastrocnemius co-contraction protect the anterior cruciate ligament against failure. J Orthop Res 1994, *in press*.

Crawford G N C, James N T. The design of muscles. In: Scientific Foundations of Orthopaedics and Traumatology (Eds. Owen R, Goodfellow J, Bullough P) Heinemann, London 1980: 67–74.

Crowninshield R D, Pope M H. The strength and failure characteristics of rat medial collateral ligaments. J Trauma 1976; 16 (2): 99–105.

Evans E B, Eggers G W N, Butler J K, Blumel J. Experimental immobilization and remobilization of rat knee joints. J Bone Joint Surg (Am) 1960; 42: 737–58.

Keele C A, Neil E, Joels N. Muscle and the nervous system. In: Samson Wright's Applied Physiology. 13th ed, Oxford University Press, Oxford 1982: 248–59.

Kuo C Y, Louie J K, Mote C D Jr. Field measurements in snow skiing injury research. J Biomech 1983; 16 (8): 609–24.

Miyatsu M, Atsuta Y, Watakabe M. The physiology of mechanoreceptors in the anterior cruciate ligament. An experimental study in decerebrate-spinalised animals. J Bone Joint Surg Br 1993; 75 (4): 653–7.

Nordsletten L, Ekeland A. Muscle contraction increases the structural capacity of the lower leg: an in vivo study in the rat. J Orthop Res 1993; 11 (2): 299–304.

Noyes F R, DeLucas J L, Torvik P J. Biomechanics of anterior cruciate ligament failure: an analysis of strain-rate sensitivity and mechanisms of failure in primates. J Bone Joint Surg (Am) 1974; 56 (2): 236–53.

Pope M H, Johnson R J, Brown D W, Tighe C. The role of the musculature in injuries to the medial collateral ligament. J Bone Joint Surg (Am) 1979; 61 (3): 398–402.

Solomonow M, Baratta R, Zhou B H, Shoji H, Bose W, Beck C, D Ambrosia R. The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. Am J Sports Med 1987; 15 (3): 207–13.

Woo S L, Hollis J M, Adams D J, Lyon R M, Takai S. Tensile properties of the human femur-anterior cruciate ligament- tibia complex. The effects of specimen age and orientation. Am J Sports Med 1991; 19 (3): 217–25.