Effects of anterior tibial displacement on the proprioceptive functions of soccer players' knee joints

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ABSTRACT

Objective: To investigate the effects of joint laxity on proprioceptive functions of the knee joints of soccer players.

Methods: In this study, we measured anterior tibial displacements and thresholds to detect the knee joint passive motion of 20 healthy soccer players (18.1±1.6 years of age). We performed all the measurements in Autumn 2000 in Ankara, Turkey. We applied the Mann-Whitney U test to analyze the relationship between the anterior tibial displacement and threshold to detect the passive motion (TDPM).

Results: There were no statistically significant differences between the TDPM values of the knee joints with insignificant laxity differences at different angles, and direction of motion ($p>0.05$). At 45° of knee flexion and externally directed motion, we found the TDPM of looser knees to be significantly greater ($p<0.05$).

Conclusion: Increased knee laxities without any clinical pathologic findings, have negative effects on knee joint proprioception. This is possibly due to the overuse degeneration of the anterior cruciate ligaments, which are the main stabilizers of knee joints.

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Many authors define proprioception as a specialized variation of the sensory modality of touch that encompasses the sensation of joint movement and joint position sense.[1-3] The sensory receptors for proprioception that are found in the skin, muscles, joints, ligaments and tendons all provide input to the central nervous system (CNS) regarding tissue deformation.[1-5] Mechanoreceptors are of importance for proprioception, which plays a major role in muscular control and influences both the precision of movement and the stability of the joint. Proprioceptive deficits have been described, in selected populations, at different times after a knee injury with a rupture of the anterior cruciate ligament (ACL).[1,3,6-10] Proprioceptive deficits resulting in motor reflex insufficiencies, possibly secondary to excessive joint laxity, may render a joint unable to sense and respond to joint stress, thereby resulting in connective tissue and ligament injury.[11] In this study, we investigated the anterior tibial displacements and threshold to detect the passive motion (TDPM) values at 2 different directions, and sought for a difference in proprioceptive ability of knees with greater laxity without any clinically pathologic findings.

Methods. Twenty healthy, male, athletes who played soccer, in the under 21 team of a professional soccer team (Gençlerbirligi SK, Ankara, Turkey) participated in this study during Autumn 2000, in Ankara, Turkey. No subject, enrolled in the study, had

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a significant history of ligament trauma to either knee joint. In addition, no subject reported suffering from any systemic or vestibular-system disorders known to impair cutaneous sensation or balance. Detailed information has been given to all the subjects, and those that signed the consent form were included in the study. In this study, 2 devices were utilized to detect the knee ligament laxities and proprioceptive status. Both measurements were performed bilaterally. Order of the tests and extremities were randomly determined. To familiarize with the proprioception assessment, subjects were tested 2 times with open eyes and ears. To quantify knee joint laxity, arthrometric measurements were performed on both knees of each subject using the KT-1000 arthrometer (MEDmetric, San Diego, California). Subjects were tested in the supine position with legs placed on thigh supports, and the feet secured with VELCRO (VELCRO USA, Inc., Manchester, New Hampshire) straps to the footrest. An experienced examiner performed all tests. Two measurements were recorded for each leg; 30 lb anterior displacement and the manual maximum anterior displacement. Three test trials were performed, and a mean test value was calculated. Prosport 1000 PMS (Tümer Engineering Co. Ltd., Ankara, Turkey) was designed to measure the threshold to detect the passive motion and the passive repositioning of the shoulder and knee joints. The device had a moving arm on which the forearm holder and pneumatic cuff had been adapted firmly. The motor of the instrument produced a rotational movement with a pre-adjusted angular velocity between 0.2°/sec-20°/sec. The time, angular displacement, and velocity were displayed digitally. Subjects were told to stop the motion of the moving arm by the hand-held disengage switch when they detected the motion and prepositioned angle. For safety, there was an emergency stop button under the control of the tester. Test-retest reliability on the proprioception testing device had been established at intraclass correlation coefficients.[12,13] Proprioception was measured using a proprioception testing device that measured the subject's threshold to detect passive motion. The proprioception testing device moved the knee at a slow, constant angular velocity (0.5°/sec). A rotational transducer interfaced with a digital microprocessor counter provided angular displacement values to the nearest tenth of degree. The testing order was randomized and counterbalanced relative to the lax knee and control limbs, starting position, and direction of movement. The subjects were seated in a neutral angle of lumbar flexion with the popliteal fossa situated 4-6 cm from the edge of the seat to prevent any cutaneous stimulation of the joint. Both feet were placed in pneumatic compression sleeves inflated to 40 mm Hg. Subjects manipulated an on-off switch to start and stop angular rotation. Also, each subject was blindfolded and wore headphones with 'white noise' to eliminate any audiovisual cues. Threshold to detect passive motion for flexion and extension was randomly tested from starting positions of 15º (near the end of extension) and 45º of flexion (midrange of motion) on both the lax and control limbs. At the beginning of the test, subjects were alerted with a tap on the shoulder. The subjects responded with a 'thumbs-up' sign to signal their readiness before engaging the motor. At some random time after the thumbs-up signal (between 1-10 seconds), the motor was engaged and moved slowly into flexion or extension. The subject pressed the on-off switch as soon as motion was perceived. Angular displacement values were recorded from the digital microprocessor counter to the nearest tenth of a degree. At either position, 3 measurements were performed and the average values were evaluated in statistical analysis. All statistical analyses were performed using the Statistical Package for Social Sciences, Personal Computer version 9. (SPSS Inc., 1998, Chicago, IL). The 0.05 level was used to denote statistical significance throughout testing. Mann-Whitney U test was used to determine whether there was a statistically significant proprioceptive difference between the normal and lax knees.

Results. The average age of the athletes participating in the study was 18.1 ± 1.6 years, and the regular training period was 8.7 ± 1.8 years. There were no significant differences between the laxities obtained by 30 lb anterior displacement and manual maximum tests of the right and left knees of the 15 athletes. The TDPM scores measured with the starting position of 15° and 45° flexion revealed no significant differences either to flexion and extension direction in these 15 subjects. The TDPM scores and the anterior displacement (laxity) values of the athletes with no laxity differences between the knees are presented in **Table 1**. There were significant differences between the laxities obtained by 30 lb anterior displacement and manual maximum tests of the right and left knees of 5 athletes. At the starting position of 45° flexion, the TDPM scores of the knees with significant laxities were found to be significantly higher than the other knees while the knees were moving to the extension direction (p=0.028). At the starting position of 15° flexion, there were no significant differences between the TDPM scores of the knees with significant laxities than the other knees while the knees were moving to the extension direction (p=0.071). At a starting position of 15° and 45° flexion, there were

Table 1 - Anterior tibial displacement (mm) and TDPM values (degrees) of athletes with no significant knee laxity differences (n=15).

Joint	Anterior tibial displacement with KT-1000		TDPM			
			Extension direction		Flexion direction	
	30 lb	MM	45°	15°	45°	15°
Right knee	6.60±1.64	7.80±1.57	0.625±0.256	0.576±0.264	0.699±0.321	0.618±0.278
Left knee	6.53±1.60	7.47±1.46	0.579±0.228	0.537±0.250	0.605±0.294	0.639±0.289
p-value	0.911	0.551	0.608	0.684	0.407	0.843

MM - manual maximum test, TDPM - Threshold to detect the passive motion

Table 2 - Anterior tibial displacement (mm) and TDPM values (degrees) of athletes with significantly different displacements (n=5).

Joint	Anterior tibial displacement with KT-1000		TDPM			
			Extension direction		Flexion direction	
	30 lb	MM	45°	15°	45°	15°
Normal knee	6.40±1.34	7.40±0.89	0.570±0.158	0.464±0.113	0.556±0.196	0.438±0.128
Knee with laxity	8.80±1.30	10±0.71	0.784±0.107	0.604±0.357	0.682±0.141	0.630±0.161
p-value	0.033*	0.007*	0.028*	0.071	0.401	0.141

MM - manual maximum test, TDPM - Threshold to detect the passive motion, *significant value

no significant differences between the TDPM scores of the knees with significant laxities than the other knees while the knees were moving to the flexion direction (*p*=0.401). The TDPM scores and the anterior displacement (laxity) values of the athletes with significant laxity differences between the knees are presented at **Table 2**.

Discussion. Soccer players participated in this study, and the values obtained by KT 1000 arthrometer did not revealed any obscure results consistent with ACL rupture. The KT 1000 arthrometer has been extensively studied,[14-16] and is widely used to test ACL deficient knees before and after reconstruction.[17] Daniel et al[14] tested 128 normal subjects to create baseline data for uninjured knees. In predominantly adult subjects, the mean anterior tibial excursion at 20 lb was 7.2±1.9 mm (range, 3-13). Eighty-eight percent of the normals had a right-left difference of less than 2 mm. In another study by Daniel et al,[15] they emphasized the importance of side-to-side difference when using the arthrometer to diagnose ACL insuffiency.[15] Normal ranges were established for a control group of 48 normal subjects in another study.[18] With the KT 1000 device at 89 N of applied tibial force, 95% of normal knees have an anterior laxity less than 9 mm, and a side-to-side difference less than 2 mm. Daniel et al[15] stated that displacement over 14 mm at 20 lb, 3 mm displacement differences between the knees, and 15 mm displacement at manual maximum test were strong indicators of ACL rupture.

Fifteen athletes were found to have anterior tibial displacement values with side-to-side differences under 2 mm. These had revealed no significant differences in TDPM results. Five athletes with side-to-side anterior tibial differences between 2-3 mm had significant differences in TDPM values at 45° flexion position (*p*=0.028) when tested at extension direction, whereas at 15° flexion position, they had near significant changes in TDPM values (*p*=0.071).

Most investigators consider ligaments to be passive stabilizers of the joints. However, more than 100 years ago, clinicians and investigators recognized the presence and potential roles of mechanoreceptors in the function of joints.[3,19,20] Joint mechanoreceptors have been most often studied in the knee, with most investigations focusing on the ACL.[3,4,6,11,19,21-25] The presence of mechanoreceptors in the ACL has led several authors to suppose that these receptors influence motor function and, conversely, that their loss leads to disfunction.[6,21,24,25] Loss of the ACL alters the kinematics of the knee and probably induces a change in the stimulation and the afferent signals or output of the remaining mechanoreceptors – for example, those in the joint capsule. Therefore, the function of the receptors of the ACL per se, must be distinguished from that of the remaining receptors in the knee.[19,26] Beynnon et al[23] studied joint position sense in knees within the first few months after ACL disruption, and found no differences in comparison with the contralateral uninjured knee.[23] Others have studied joint position sense in knees with chronic ACL tears and determined that there are differences in comparison with the contralateral knee.[4,21] Similarly, the threshold to detection of passive knee motion is altered in subjects with chronic ACL tears.[3,4,23,27] In a previous study of subjects with uninjured knees, Beynnon et al[23] determined that measurement of the threshold to detection of passive knee motion was more accurate, precise, and reliable in comparison with joint position sense. Threshold to detect the passive motion was preferred to evaluate the proprioceptive ability of the knee joints of the soccer players in our study.

Borsa et al,[24] demonstrated TDPM deficits at the ranges of knee extension (15°). For the ACL-deficient limb, TDPM at 15° moving into extension was significantly lower than moving into flexion. Similarly, in the ACL-deficient limb, TDPM at 15° moving into extension was significantly lower than at 45° Mechanoreceptors of the ACL are stimulated primarily by hyperextension. Krauspe et al,[20] in single-fiber studies, identified 26 mechanoreceptors of the cruciate ligament among 13 animals. No activity was seen with the knee in the resting position of 30° of flexion. All fibers responded to movement, primarily extension, with a marked increase in activity if internal or external rotation was added in extension. Our study revealed similar results. The knee joints with greater displacement values had lesser ACL tension, which might have led to increased thresholds to detect the passive motion.

In one study,[11] female athletes were compared with their male counterparts, and results revealed that women inherently possess significantly greater knee joint laxity values, and demonstrate a significantly longer time to detect the knee joint motion moving into extension. In this study, all tests were performed at the starting position of 15° of knee flexion. This starting position is near the end range of the joint's motion. As the knee further extends from this position, the ACL becomes increasingly taut, which may be why the investigators found differences between men and women in joint kinesthesia. The significantly greater knee joint laxity inherent to the female athletes may have caused them to have less taut, and therefore less sensitive, ligaments at the initiation of testing.

Ligaments and other capsular structures, which surround the knee joint, contain collagen as a primary constituent. Because of this, they could be expected to demonstrate viscoelastic behavior. There is clinical evidence that ligaments undergo significant loading with many activities, since the symptomatology from ACL insufficiency and medial collateral ligament insufficiency is a major indication for reconstruction of these ligaments.[28] In our study, 5 athletes with significantly different anterior displacement values between their knee joints did not cope with ACL injury criteria. The displacement values that were under 3 mm were thought to result from continuous mechanical loadings experienced during the training sessions.

In this study, a relationship has been sought between mechanical and neuromuscular functions of knee joints. Mechanically, anterior tibial displacements were tested, TDPM test was performed to evaluate the proprioceptive status of the knee joints of soccer players. The most considerable outcome of this study is that differences under pathologic limits resulted in significant kinesthetic losses.

In conclusion, this study demonstrates the knee joints with greater displacement values had lesser ACL tension, which might have led to increased thresholds to detect the passive motion. Increased knee laxities without any clinical pathologic findings, have negative effects on knee joint proprioception. This is possibly due to the overuse degeneration of the ACLs, which are the main stabilizers of knee joints.

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References

1. Harter RA, Osternig LR, Singer KM, James SL, Larson RL, Jones DC. Long-term evaluation of knee stability and function following surgical reconstruction for anterior cruciate ligament insufficiency. ***Am J Sports Med*** 1988; 16: 434–443.
2. McDonald PB, Hedden D, Pacin O, Sutherland K. Proprioception in anterior cruciate ligament-deficient and reconstructed knees. ***Am J Sports Med*** 1996; 24: 774-778.
3. Miyatsu M, Atsuta Y, Watakabe M. The physiology of mechanoreceptors in the anterior cruciate ligament. ***J Bone Joint Surg Br*** 1993; 75: 653-657.
4. Corrigan JP, Cashman WF, Brady MP. Proprioception in the cruciate deficient knee. ***J Bone Joint Surg Br*** 1992; 74: 247-250.
5. Grigg P. Peripheral neural mechanisms in proprioception. ***Journal of Sport Rehabilitation*** 1994; 3: 2-17.
6. Barrack RL, Skinner HB, Cook SD. Proprioception of the knee joint. ***Am J Sports Med*** 1984; 63: 175-181.
7. Co FH, Skinner HB, Cannon WD. Effect of reconstruction of the anterior cruciate ligament on proprioception of the knee and the heel strike transient. ***J Orthop Res*** 1993; 11: 696-704.

8. Fridèn T, Roberts D, Zätterström R, Lindstrand A, Moritz U. Proprioception in the near extended knee-measurements of position and movement in healthy individuals and in symptomatic ACL injured patients. ***Knee Surg Sports Traumatol Arthros*** 1996; 4: 217–224.
9. Jerosch J, Prymka M. Knee joint proprioception in normal volunteers and patients with anterior cruciate ligament tears, taking special account of the effect of a knee bandage. ***Arch Orthop Trauma Surg*** 1996; 115: 162-166.
10. Lephart SM, Kocher MS, Fu FH, Borsa PA, Harner CD. Proprioception following anterior cruciate ligament reconstruction. ***Journal of Sport Rehabilitation*** 1992; 1: 188-196.
11. Rozzi SL, Lephart SM, Gear WS, Fu FH. Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. ***Am J Sports Med*** 1999; 27: 312-319.
12. Kunduracioglu B, Ulkar B, Cetin C, Guner R, Ergen E. Detection of passive motion threshold in evaluation of shoulder proprioception. ***Journal of Rheumatology & Medical Rehabilitation*** 2001; 12: 51-55.
13. Ulkar B, Kunduracioglu B, Cetin C, Guner RS. Effect of positioning and bracing on passive position sense of shoulder joint. ***Br J Sports Med*** 2004; 38: 549-552.
14. Daniel DM, Malcom LL, Losse G, Stone ML, Sachs R, Burks R. Instrumented measurement of anterior laxity of the knee. ***J Bone Joint Surg Am*** 1985; 67: 720-726.
15. Daniel DM, Stone ML, Sachs R, Malcom L. Instrumented measurement of anterior knee laxity in patients with acute anterior cruciate ligament disruption. ***Am J Sports Med*** 1985; 13: 401-407.
16. Highgenboten CL, Jackson A, Meske NB. Genucom, KT-1000, and Stryker knee laxity measuring device comparisons. ***Am J Sports Med*** 1989; 17: 743-746.
17. Flynn JM, Mackenzie W, Kolstad K, Sandifer E, Jawad AF, Galinat B. Objective evaluation of knee laxity in children. ***J Pediatr Orthop*** 2000; 20: 259-263.
18. Sherman OH, Markolf KL, Ferkel RD. Measurements of anterior laxity in normal and anterior cruciate absent knees with two instrumented test devices. ***Clin Orthop Relat Res*** 1987; 215: 156-161.
19. Hogervorts T, Brand RA. Mechanoreceptors in joint function. ***J Bone Joint Surg Am*** 1998; 80: 1365-1378.
20. Krauspe R, Schmidt M, Schaible HG. Sensory innervation of the anterior cruciate ligament. An electrophysiological study of the response properties of single identified mechanoreceptors in the cat. ***J Bone Joint Surg Am*** 1992; 74: 390-397.
21. Barrett DS. Proprioception and function after anterior cruciate reconstruction. ***J Bone Joint Surg Br*** 1991; 73: 833-837.
22. Beard DJ, Kyberd PJ, Fergusson CM, Dodd CAF. Proprioception after rupture of the anterior cruciate ligament. ***J Bone Joint Surg Br*** 1993; 75: 311-315.
23. Beynnon BD, Ryder SH, Konradsen L, Johnson RJ, Johnson K, Renström PA. The effect of anterior cruciate ligament trauma and bracing on knee proprioception. ***Am J Sports Med*** 1999; 27: 150-155.
24. Borsa PA, Lephart SM, Irrgang JJ, Safran MR, Fu FH. The effects of joint position and direction of joint motion on proprioceptive sensibility in anterior cruciate ligament deficient athletes. ***Am J Sports Med*** 1997; 25: 336-340.
25. Pap G, Machner A, Nebelung W, Awiszus F. Detailed analysis of proprioception in normal and ACL-deficient knees. ***J Bone Joint Surg Br*** 1999; 81: 764-768.
26. Johansson H, Sjölander P, Sojka P. A sensory role for the cruciate ligaments. ***Clin Orthop Relat Res*** 1991; 266: 161-178.
27. Barrack RL, Skinner HB, Buckley SL. Proprioception in the anterior cruciate deficient knee. ***Am J Sports Med*** 1989; 17: 1-6.
28. Skinner HB, Wyatt MP, Stone ML, Hodgdon JA, Barrack RL. Exercise-related knee joint laxity. ***Am J Sports Med*** 1986; 14: 30-34.