“Comparison in Joint Position Sense and Muscle Coactivation Between ACL Deficient and Healthy Individuals”
by Suarez T et al.
Journal of Sport Rehabilitation
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Section: Original Research Report

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Journal: Journal of Sport Rehabilitation

Acceptance Date: April 27, 2015

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DOI: http://dx.doi.org/10.1123/jsr.2014-0267
TITLE: Comparison in joint position sense and muscle coactivation between ACL deficient and healthy individuals

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ABSTRACT

Context: Tearing of the anterior cruciate ligament (ACL) may disrupt the ability to recognise the knee position in space during limb repositioning tasks, which is referred to as joint position sense (JPS). Impairments in JPS have been shown to be lower during active than passive repositioning tasks, thus suggesting that coactivation patterns of the muscles surrounding the knee might compensate for the disrupted JPS and ensure accurate limb repositioning in ACL deficient individuals. Objective: To investigate muscle coactivation patterns during JPS repositioning tasks in ACL deficient and healthy individuals. Design: Prospective observational study. Setting: Functional assessment laboratory. Participants: Eight males aged 25±8 with isolated ACL rupture and 10 males aged 30±4 with no history of knee injury. Intervention: JPS was evaluated by means of an electrogoniometer in a sitting position during either passive or active joint positioning and repositioning tasks with a 40-degree target knee angle. Main outcome measures: Root-mean-square (RMS) of the surface electromyogram from the vastus lateralis and biceps femoris muscles was measured during active joint positioning and repositioning. Results: Healthy participants showed a significant decrease in the vastus lateralis RMS (-19%) and an increase in the biceps femoris RMS (+26%) during joint repositioning compared to positioning. In contrast, ACL deficient patients showed no modulation in muscle coactivation between joint positioning and repositioning, although they exhibited significantly lower RMS of the vastus lateralis (injured limb:-28%; uninjured limb:-21%) and higher RMS of the biceps femoris (injured limb:+19%; uninjured limb:+30%) than the healthy participants during joint positioning. Conclusions: The lack of modulation in muscle coactivation patterns between joint positioning and repositioning in ACL deficient patients might be attributed to the disrupted neural control following the injury-related loss of proprioceptive information. These results should be taken into account in the design of rehabilitation protocols with emphasis on muscle coactivation and JPS.
INTRODUCTION

The anterior cruciate ligament (ACL) is a major restraint of the knee joint, which prevents anterior translation of the tibia and hyperextension of the knee. Beside its mechanical role, the ACL is also recognized as a major sensory structure, which monitors relevant afferent information through mechanoreceptors and mediates the ability to recognize the knee position in space, referred to as joint position sense (JPS). This is indirectly supported by a number of previous studies reporting impaired JPS in individuals with ACL deficient knees. For this reason, over the last 15 years, rehabilitation programs following ligament injuries of the knee have been focusing not only on recovering mechanical stability, but also on improving sensory function of ligament and capsules in order to maintain the joint functional stability.

Mechanoreceptors in the ACL have been shown to contribute to muscle co-ordination and functional joint stability through feedback mechanisms, i.e. initiating a reflex arc leading to stabilizing muscle contractions, and feed-forward mechanisms, i.e. pre-programming the muscle stiffness by a continuous reflex modulation of the γ-muscle spindle system. Significant alterations in neural activation of muscles acting on the knee joint following ACL rupture may involve a reduced drive to the knee extensors, which is referred to as arthrogenic muscle inhibition (AMI), and an increased drive to the knee flexors, which is referred to as antagonist coactivation. Such alterations in coactivation of muscles surrounding the knee may act as a compensatory mechanism to protect against anterior translation of the tibia and ensure joint stability during functional activities in ACL injured patients.

The assessment of JPS following ACL injury has been performed over the last decades by measuring the ability to reproduce a pre-established position of the knee joint either passively or actively. During active repositioning tasks, the accuracy of reproducing
pre-established joint positions has been shown to be greater than during passive repositioning tasks, i.e. when relying exclusively on passive joint structures, in both healthy\textsuperscript{16,17} and ACL reconstructed individuals.\textsuperscript{18,19} This has been attributed to the adjunctive contribution of muscle spindles during active with respect to passive repositioning tasks.\textsuperscript{20} In addition, it may be hypothesised that coactivation patterns of the muscles surrounding the knee joint adapt to compensate for the disrupted JPS and ensure accurate joint repositioning in ACL deficient individuals. To the best of the authors’ knowledge, however, there are no studies investigating muscle coactivation patterns underpinning JPS during active repositioning tasks in ACL deficient individuals.

This study, therefore, aimed at comparing coactivation patterns of the knee extensor and flexor muscles during JPS tasks between ACL deficient and healthy individuals. It was hypothesised that JPS tasks would have been accompanied by a decrease in the activation of the knee extensor muscles and an increase in the activation of the knee flexor muscles as a compensatory mechanism in ACL deficient patients with respect to healthy individuals.

**MATERIALS AND METHODS**

**Participants**

Eight male patients (age: 25 ± 8 years; stature: 1.79 ± 0.2 m; body mass: 74.6 ± 5.1 kg) with unilateral isolated ACL rupture in the dominant limb were recruited to participate in the study. Inclusion criteria were occurrence of ligament rupture from 30 to 60 days before testing, full range of motion at the knee joint and lack of pain, locking and swelling of the knee joint as evaluated by the Lysholm Knee Scoring Scale.\textsuperscript{21} Physical activity levels were evaluated in all patients by means of the Tegner Activity Score\textsuperscript{21} and only patients with activity level between 3 and 5 were included in the study, while sedentary individuals and competitive sport athletes were excluded. Exclusion criteria were concomitant injury to any
other knee ligament or any previous surgery on either knee, and adherence to any rehabilitation program after the injury. Ten healthy and physically active male volunteers (age: 30 ± 4 years; stature: 1.73 ± 0.3 m; mass: 75.8 ± 15.3 kg), with no disorder or history of knee injury, served as the control group. Individuals were not included in the study if they were engaged in regular training or sport practice more than 3 times a week or in professional sport activities. This study was approved by the local Ethics Committee and carried out in accordance with the Declaration of Helsinki. Informed consent was obtained from all participants.

Experimental procedures and data analysis

All experimental procedures were completed in an isolated room and subjects wore loose-fitting shorts. Participants sat on a raised plinth with their trunk inclined approximately of 90° from the horizontal and were blindfolded to eliminate visual input during testing (Figure 1). The knee angle was measured by an electrogoniometer (Penny & Giles, Santa Monica, CA), of which adhesive end blocks were placed in line with the vertical axis of the leg on the sagittal plane, connected to a computerized system unit (MuscleLab, Bosco-System Technologies, Rieti, Italy). By using visual feedback from the electrogoniometer, the experimenter asked each participant to perform either active or passive JPS trials with a target knee angle of 40°. The 40° target angle was chosen as it corresponds to the position in which there is the highest moment arm of the hamstrings muscles. During each active JPS trial, the participant was asked to extend the knee from a 90-degree starting position to the target angle and hold it for 5 s (i.e. positioning task). After moving back to the starting position, the participant was asked to actively reproduce the target angle and hold it for 5 s (i.e. repositioning task). During each passive JPS trial, the experimenter, by supporting with his palm open the heel of the subject, moved the participant’s limb from the starting position to
the target angle and held it for 5 s (i.e. positioning task). After moving back to the starting position, the experimenter extended the participant’s limb, stopped it when the participant communicated verbally that the target angle was reached, and held it for 5 s (i.e. repositioning task). Each active and passive JPS trial was repeated 3 times in a random order. Prior to the experimental measurements, all participants were familiarised with the procedure by explanation, demonstration and 3 practice repetitions for each condition. Both limbs of ACL deficient patients and the dominant limb of controls were tested. JPS trials were performed at slow speed by only one investigator, who had been trained carefully for the testing procedure.

During active JPS trials, surface electromyography (sEMG) signals from the vastus lateralis (VL) and biceps femoris (BF) muscles were recorded by means of silver/silver chloride, pre-gelled, self-adhesive, and 4 mm in diameter electrodes (Blue Sensor Ag/AgCl type NF-00-S/12, Ambu A/S, Ballerup, Denmark). For each muscle, after shaving and gentle skin abrasion with abrasive paste (Meditec-Every, Parma, Italy), two electrodes were attached 20 mm apart (centre-to-centre) on the skin half way between the centre of the muscle belly and the distal myotendinous junction, in accordance with SENIAM recommendations.23 A ground electrode was placed around the ankle of the contra lateral limb. The VL and BF muscles were considered to be representative of the knee extensors and flexors muscle groups, respectively, as in previous studies.24-26 Medical adhesive tape and an elastic band were used to fix the sEMG cables on the skin in order to minimize any motion artefacts that could be encountered during the JPS trial. The sEMG cables included a pre-amplifier (gain: 1k) and a Butterworth band-pass filter (cut-off frequencies: 8-600 Hz). Signals were then full-wave root mean square (RMS) converted with an averaging time constant of 100 ms and then sampled at 100 Hz by a portable EMG system (MuscleLab 4020e, Ergotest Technology AS, Langesund, Norway), as previously described.27,28
Data analysis and statistics

For both the active and passive JPS trials, the repositioning error was calculated as the difference between the average angle over the last 2 s of the positioning and repositioning tasks of each trial. For the active JPS trials, the average RMS in both muscles was computed off-line over the last 2 s of the positioning and repositioning phases of each trial. The average RMS of the VL was normalized to the average RMS obtained over the last 2 s while maintaining for 5 s a 0-degree knee angle position (= full extension), while the average RMS of the BF was normalized to the average RMS over the last 2 s while maintaining for 5 s a 90-degree knee angle position during standing on the contralateral limb.

All data were normally distributed in terms of skewness and kurtosis (all values < |2|). A two-way ANOVA was used to compare the repositioning error between conditions (active and passive) and groups’ limbs (injured and uninjured for ACL deficient patients, dominant for healthy controls). A one-way repeated-measures was performed to compare both VL and BF muscles activity between joint positioning and repositioning tasks, and different groups’ limbs (injured and uninjured for ACL deficient patients, dominant for healthy controls). When a main effect F-value was significant, paired-samples t-test was used to locate the significant differences. All statistical analyses were performed using SPSS statistical software package (version 20.0, SPSS, Inc., Chicago, IL, USA). A significance level of P < 0.05 was adopted.

RESULTS

The two-way ANOVA showed that both condition and limb had a significant main effect on the repositioning error (F = 9.63, p < 0.01 and F = 7.44, p < 0.01, respectively), while there was no interaction between condition and limb. As shown in Figure 2, follow-up analysis revealed that the repositioning error was higher in the passive condition than in the
active condition for both limbs of ACL deficient patients (p < 0.05). In the passive condition, there was a significant main effect of limb on the repositioning error (F = 5.25; p < 0.01), as it was higher in both the injured and the uninjured limbs of ACL deficient patients than in the dominant limb of healthy participants (average increase of 1.3 and 1.6°, respectively; p < 0.05). In the active condition, there was a significant main effect of limb on the repositioning error (F = 4.17; p < 0.05) although differences in repositioning error reached significance only between the uninjured limb of ACL deficient patients and the dominant limb of the healthy participants (average increase of 0.3°; p < 0.05).

For the active condition, the repeated-measures showed no significant main effect of both task and limb on the vastus lateralis RMS, although there was a significant interaction between task and limb (F = 8.35; p < 0.01). Follow-up analysis revealed that there was a main effect of task only for the control group, in that vastus lateralis RMS of healthy participants was 19% lower during joint repositioning than during joint positioning (F = 19.15; p < 0.01). As shown in Figure 3, during joint positioning the vastus lateralis RMS was lower in both the injured and the uninjured limb of ACL deficient patients than in healthy participants (average decrease of 28% and 21%, respectively; p < 0.01 and p < 0.05, respectively), while during joint repositioning there were no differences between ACL deficient patients and healthy participants.

The repeated measures showed that there was a main effect of task on the biceps femoris RMS (F = 5.53; p < 0.05) and a significant interaction between task and limb (F = 5.19; p < 0.05). Follow-up analysis revealed that differences between positioning and repositioning were significant only for the control group, in that the biceps femoris RMS of healthy participants was 26% higher during joint repositioning than during joint positioning (F = 8.6; p < 0.05). Figure 4 shows the biceps femoris RMS in ACL deficient patients and healthy participants during both joint positioning and repositioning tasks. Visual inspection
shows that during joint positioning the biceps femoris RMS was higher in both the injured and the uninjured limbs of ACL deficient patients than in the dominant limb of healthy participants (average increase of 19% and 30%, respectively; \( p < 0.01 \) and \( p < 0.05 \), respectively), while during repositioning there were no differences between limbs of ACL deficient patients and healthy participants.

**DISCUSSION**

For the first time, to the best of the authors’ knowledge, muscle coactivation patterns during JPS tasks have been compared between ACL deficient and healthy male individuals. The results of the present study revealed peculiar differences in the coactivation of the knee extensor and flexor muscles between ACL deficient and healthy participants during joint positioning and repositioning tasks. Healthy participants showed a significant task-related modulation of muscle coactivation, which consisted of an increase in the BF muscle activity and a decrease in the VL muscle activity between joint positioning and repositioning. ACL deficient patients exhibited higher BF and lower VL muscle activity than healthy participants during joint positioning, but no modulation of muscle coactivation between joint positioning and repositioning.

A greater accuracy in active repositioning of a pre-established knee angle with respect to passive repositioning is consistent with the finding of others studying JPS on ACL deficient patients under similar experimental conditions.\(^{18,19}\) This has been attributed to the adjunctive contribution of muscle spindles during active with respect to passive JPS tasks.\(^{20}\)

In the present study, ACL deficient patients exhibited lower agonist and higher antagonist activity than healthy participants during the joint positioning task of active JPS trials. The decrease in activation of the knee extensor muscles may be due to AMI, which is referred to as a failure in voluntary activation and is thought to be a consequence of the
altered afferent input originating from knee mechanoreceptors and of pain and disuse following injury.\textsuperscript{11,12} On the other hand, the increase in activation of antagonist muscles is of similar magnitude to that observed during other dynamic functional activities, such as walking,\textsuperscript{15} and may act to prevent anterior translation of the tibia and ensure knee stability.\textsuperscript{2}

In healthy participants, during the repositioning task of active JPS trials, there was a lower activity of the antagonist muscles and a higher activity of the agonist muscles with respect to the positioning task. Conversely, ACL deficient patients maintained similar levels of muscle activity during both positioning and repositioning tasks. In other words, a modulation of muscle coactivation patterns was found between positioning and repositioning tasks in healthy participants, while there was no task-related modulation in ACL deficient patients. This would indicate that ACL deficient patients decreased agonist activity and increased antagonist muscle activity regardless of the task that was executed. Interestingly, the lack of task-related modulation in muscle coactivation patterns was found in both the injured and the uninjured limb of ACL deficient patients. Furthermore, both the injured and the uninjured limb of ACL deficient patients showed significantly impaired JPS with respect to healthy individuals, which is in line with previous findings.\textsuperscript{30,7} Even though the anatomical structures are intact in the uninjured limb, previous authors have speculated that the injury-related disruption of afferent information from sensory receptors of the injured joint would affect the functioning of muscle spindles in the uninjured limb,\textsuperscript{30,31} thus leading to impaired JPS in both limbs of ACL deficient patients. This may have important implications on the rehabilitation process following the ligament injury, especially considering neuromuscular exercises to improve and preserve the knee JPS.\textsuperscript{9}

The results of the present study show that the assessment of muscle coactivation patterns during active JPS tasks is effective for identifying and quantifying deficits of both limbs after ACL injury. The earliest goals after ACL injury, therefore, should be to target the
coactivation patterns of the knee extensor and flexor muscles during active joint positioning/repositioning tasks to progress to further stages of the rehabilitation program. With this regard, it is paramount to include joint positioning and repositioning exercises in “active” form of both limbs during the rehabilitation with the aim of moving as close as possible to the muscle coactivation patterns of healthy participants.

The present study has, however, some limitations that need to be addressed. First, there was a limited number of patients, although the selection of a homogenous sample with a recent unilateral ACL rupture is one of the major strengths of the study. Second, as only the target joint angle of 40° knee flexion was evaluated during JPS trials, our results cannot be applied to other knee joint angles. Another limitation of the present study is the exclusion of female subjects, which limits the generalizability of the results. Last, testing was carried out in a seated position, whereas in the most recent studies JPS was assessed in a standing weight-bearing position, which has the advantage of stimulating proprioceptors that are involved in functional actions of daily living activities. On the other hand, the adopted sitting position allowed to investigate exclusively the muscles acting on the knee joint, thus ruling out other confounding factors.

In conclusion, the present study showed that ACL deficient male patients exhibited higher antagonist and lower agonist activity than healthy male participants during the joint positioning task, but no modulation of muscle coactivation between joint positioning and repositioning tasks. Since these muscle coactivation patterns were found to be similar in both the injured and the uninjured limb of ACL deficient patients, it is recommended to clinicians that neuromuscular rehabilitation should be addressed to both limbs of injured patients. Further studies are needed to evaluate whether muscle coactivation patterns may be restored after appropriate rehabilitation or ACL reconstruction.
CONFLICT OF INTEREST

The authors have no conflict of interest.
REFERENCES


Figure 1. Experimental setup and starting position of participants prior to the joint positioning/repositioning tasks.
Figure 2. Active and passive joint repositioning error in the dominant limb of healthy control participants and in both the injured and uninjured limb of ACL deficient patients. * = Significantly different from Control; † = Significantly different from Active.
**Figure 3.** RMS of sEMG from the vastus lateralis muscle during active positioning and repositioning tasks in the dominant limb of healthy control participants and in both the injured and uninjured limb of ACL deficient patients. Data (mean ± standard deviation) are reported as percentage of RMS at a reference position of 0-degree angle. * = Significantly different from Healthy control; † = Significantly different from Positioning.
Figure 4. RMS of sEMG from the biceps femoris muscle during active positioning and repositioning tasks in the dominant limb of healthy control participants and in both the injured and uninjured limb of ACL deficient patients. Data (mean ± standard deviation) are reported as percentage of RMS at a reference position of 90-degree angle during standing on the contralateral limb. * = significantly different from Healthy control; † = significantly different from Positioning.