The Knee 26 (2019) 194–200

Contents lists available at ScienceDirect

The Knee

Hypoesthesia after anterior cruciate ligament reconstruction: The relationship between proprioception and vibration perception deficits in individuals greater than one year post-surgery

Carol A. Courtney [a,*], Pranoti Atre [a], Kharma C. Foucher [b], Ali M. Alsouhibani [a]

[a] Department of Physical Therapy, University of Illinois at Chicago, Chicago, IL, USA
[b] Department of Kinesiology and Nutrition, University of Illinois at Chicago, Chicago, IL, USA

ARTICLE INFO

Article history:
Received 17 April 2018
Received in revised form 18 September 2018
Accepted 27 October 2018

Keywords:
Quantitative sensory testing
Knee
Surgery
Neurophysiology
Postural control

ABSTRACT

Background: While surgical reconstruction restores mechanical stability following anterior cruciate ligament (ACL) rupture, many experience early-onset osteoarthritis despite surgery. Neurophysiological changes are hypothesized to contribute to knee osteoarthritis progression. Proprioceptive deficits have been reported following ACL injury/reconstruction; however, vibration perception threshold (VPT) has been less studied. This study explored relationships between pain, VPT, proprioception, function, and strength following ACL-reconstruction.
Methods: Twenty individuals (27 ± 6 years; 10 males) (standard deviation) status-post ACL-reconstruction were compared with a control group. Measurements included VPT, proprioception (threshold to detect passive movement), pain, function (Knee Outcome Survey (KOS)) and isometric quadriceps strength. Group differences were assessed using Mann-Whitney U tests, side-to-side differences with Wilcoxon Signed Rank tests, and associations evaluated using Spearman correlations.
Results: The ACL-reconstruction group had minor functional deficits (15 ± 11%) and resting pain (1.8 ± 1.7). Impaired VPT and proprioception (hypoesthesia) were demonstrated on surgical compared to contralateral and control limbs ($p \leq 0.008$). Proprioception was significantly different between contralateral and control knees, but not VPT. Surgical knee proprioceptive deficits and VPT deficits were positively correlated ($\rho = 0.462$, $p = 0.047$) but not in controls ($\rho = -0.042$, $p = 0.862$). Strength was negatively correlated to pain ($\rho = -0.589$; $p = 0.006$), but not to KOS scores, proprioception or VPT ($p \geq 0.099$).
Conclusion: Proprioceptive deficits following ACL injury have been ascribed to loss of afferent input from the torn ligament. Alternatively, multi-modality as well as contralateral sensory deficits suggest a spinal/supraspinal source of neurophysiological findings which may predispose to early osteoarthritis.
Level of evidence: III.

1. Introduction

Anterior cruciate ligament (ACL) rupture is a common athletic injury that occurs most frequently in the young and physically active. These injuries often result in functional deficits and complaints of knee instability [1]. Regardless of treatment, post-traumatic osteoarthritis (OA) may develop over time [2]. The mechanisms behind this accelerated joint degeneration remain

* Corresponding author.
E-mail address: cacourt@uic.edu (C.A. Courtney).

https://doi.org/10.1016/j.knee.2018.10.014
0968-0160/

obscure; however, altered joint loading and/or neurophysiologic processing may be contributing factors [2]. Proprioceptive loss, or hypoesthesia, may contribute to aberrant postural control, which can limit full functional return [3] and allow aberrant stresses at the joint which promote degenerative changes [4,5]. Furthermore, bilateral changes in postural control have been reported in individuals with unilateral ACL deficiency [6] and ACL reconstruction [7]. Both meniscal damage and quadriceps weakness have been identified as factors related to development of OA [8]; however other researchers failed to find quadriceps muscle weakness associated with knee OA at a 10–15 year follow-up [9].

Proprioception involves both the sense of location within space, and the ability to detect how far or how rapidly a joint has moved [10]. Deficits in both modalities have been reported in ACL deficient [11,12] and ACL reconstructed [13] knees. In contrast, other researchers found that proprioception normalized following ACL reconstructive surgery [14]. Proprioceptive loss after ACL rupture has been attributed to sensory receptor loss [4,11], meaning that sensory fibers located in the ligament are destroyed after ACL injury, leaving a void in normal afferent input from the knee to the central nervous system. This equates to a peripheral neurologic lesion as the cause of proprioception deficits. However, proprioceptive loss has been found bilaterally in individuals with unilateral ACL injury [15], suggesting that more complex neurophysiological changes may occur. Proprioception is mediated via input from the joint, muscle and skin; however the major contribution comes from the muscle spindle [10,16]. This generates uncertainty about the role of afferent input from the ACL in proprioceptive acuity. Furthermore, sensory impairments in proprioception, cutaneous mechanical detection threshold and vibration perception threshold (VPT) [17–20] have been demonstrated in knee disorders without ligamentous damage, such as knee OA and patellofemoral dysfunction. Considering this diverse sensory loss in an array of knee disorders, the underlying mechanisms of this phenomenon may be debated.

Vibration perception, especially at threshold (VPT), is mediated via specialized cutaneous mechanosensory receptors [21]. Hence, deficits in this sensory modality would not typically be attributed to joint trauma such as ligamentous injury. If hypoesthesia of multiple sensory modalities was present in this population, it is possible that a distinct central mechanism is responsible for this phenomenon. However, VPT has been less studied in the ACL deficient or ACL reconstructed population. Therefore, the purpose of this study was to examine VPT and proprioception, measured via threshold to detection of passive motion (TDPM), in individuals, status-post ACL reconstruction, and compare their sensory findings to an age and sex matched control group. A secondary purpose was to determine if VPT, proprioception, function, isometric quadriceps strength and pain measures were associated. We hypothesized that if hypoesthesia in individuals with ACL reconstruction is centrally mediated, a relationship may exist between deficits in proprioception and deficits in VPT.

2. Methods

2.1. Patients

Twenty individuals (mean age 27 ± 6 years; range 20–47 years; 10 males) with history of ACL reconstructive surgery and 20 age and sex-matched healthy controls were included in the study. Participants were recruited through use of recruitment flyers on the university campus and outpatient clinics of the University of Illinois at Chicago. ACL reconstruction group participants were excluded if they were >50 years or had history of bilateral ACL reconstruction, sensory and motor impairments, or previous lower extremity injury. Subjects were instructed to avoid anti-inflammatory/pain medications and exercise 24 h prior to testing. Participants were blinded to the results during testing with no information provided on the postulated hypotheses of the study.

The study was approved by the Institutional Review Board of the University of Illinois at Chicago. Each participant signed a written informed consent and provided a brief medical history. The Activities of Daily Living Scale of the Knee Outcome Survey (KOS) was completed prior to testing [22]. This survey is a 14-item scale assessing the effect of knee symptoms on function. Scores are presented as percentages of maximal score where 100% represents full perceived knee function. Resting pain intensity was determined using the self-reported Numeric Pain Rating Scale (NPRS). The NPRS is an 11-point scale where patients rate pain from 0 (no pain) to 10 (worst pain imaginable). Passive range of motion (ROM) was tested in supine using a standard goniometer to ascertain full knee extension and functional knee flexion. Prior to testing, subjects were acclimated to a room temperature of approximately 70 °F for 10–15 min [23]. Sensory testing was performed followed by isometric strength testing.

2.2. Vibratory acuity

Prior to testing, all subjects were educated on VPT procedures at a proximal site until capable of responding reliably [24]. Assessment of VPT was assessed using a Bioesthesiometer (Bio-Medical, OH) at the medial femoral condyle, lateral femoral condyle and tibial tuberosity. The medial and lateral malleoli at the ankle were also tested.

The 13 mm vibratory tip, oscillating 100 Hz, was placed at the site of application. The amplitude of vibration (expressed in "bioesthesiometer units" [25]) was increased at one volt/second until the participant reported an initial sensation of vibration [20]. Three measurements were taken at each location with mean of results used for data analysis. Previous researchers have found assessment using the bioesthesiometer to be highly reproducible and reliable [20]. Shakoor et al. [20] demonstrated an intraclass correlation coefficient of 0.96–0.99 between initial and repeat testing on separate days.

2.3. Proprioceptive acuity

Proprioception was measured by examining TDPM. This method has been shown to be reliable [26,27] and has been used in previous studies [11,12,27]. Subjects were tested in a standardized seated position (hip flexion 70°, knee flexion 45°, ankle neutral). An air splint (20 mm Hg) was placed on the foot to minimize cutaneous input. Subjects were blindfolded and listened to white noise to minimize visual and auditory input, respectively. Before each trial, subjects were asked to perform a 10% co-contraction of the test-limb in the test position for three seconds [28]. After the contraction, subjects were instructed to relax completely. A conditioning muscle co-contraction in the test position has been demonstrated to limit confounding thixotrophic effects on TDPM measures by removing slack in intrafusal fibers and raising spindle resting activity [29]. The purpose was to standardize the starting point for all proprioceptive testing.

Following a random delay, passive, slow (0.5°/s) flexion or extension of the knee was achieved by a motor and pulley attached to the air splint (Figure 1). Subjects indicated when they detected movement or change in position of the knee by pressing a switch, and were required to note the direction of movement. The trial duration ranged from one to three minutes. A pretest trial was followed by three actual trials on each limb, with order of testing randomly assigned. The mean of these trials was used for data analysis. The amount of linear movement of the pulley (X) was recorded to calculate threshold to detection (Y, in degrees) using the following formula:

$$Y = \tan^{-1}\frac{X}{R},$$

where R = shank length (medial joint line of the knee to inferior aspect of the medial malleolus [30].

2.4. Isometric strength

Quadriceps strength was assessed in a standardized sitting position with both the hip and knee maintained in 90° flexion. A MicroFet 2 dynamometer (Hoggan Health Industries Inc., West Jordan, UT, USA) was used to determine isometric muscle strength. Each participant was instructed to extend the knee at maximal intensity against the stationary dynamometer. A mean of three trials was calculated and recorded and a percent score was calculated by dividing the affected limb score by the unaffected limb score in the ACL reconstruction group, and the dominant by the non-dominant in the control group.

2.5. Statistical analysis

Statistical analysis was conducted using SPSS software V.24 (IBM SPSS, IBM Statistical Package for the Social Sciences (SPSS) Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp). The data was visually inspected for normal distribution which revealed that most variables were not normally distributed. Therefore, non-parametric statistical analysis was used. Independent samples Mann–Whitney U tests were used to determine whether or not VPT and proprioception differed between the post-ACL group and the control group. Next, Related-Samples Wilcoxon Signed Rank Tests were used to compare VPT and proprioception between the affected and unaffected sides of the ACL reconstruction group. Spearman correlations were used to determine if associations among VPT, proprioception (TDPM), function, strength and pain existed. Finally, associations between TDPM and VPT were examined by group.

3. Results

Twenty subjects were recruited into each of the groups with demographic information provided in Table 1. In the experimental group, 40% (eight) had undergone ACL reconstruction using a semitendinosus tendon graft, 50% (10) received a patellar tendon graft and 10% (two) received an allograft from a cadaver. Type of surgery was not found to be a factor in sensory findings. The control group subjects denied any pain at the time of testing, while 12/20 subjects in the experimental group admitted to

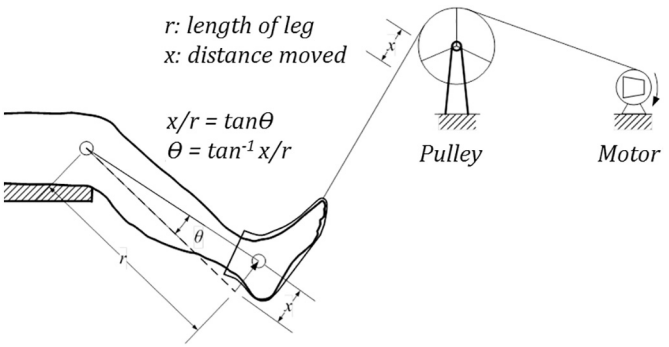

Figure 1. Measurement of threshold to detection of passive motion.

Table 1
Demographic information about participants.

Characteristics (standard deviation)	ACL reconstruction	Healthy controls
Age (years)	27 (5.9)	27 (5.5)
Months since surgery (months)	65 (47)	–
Body Mass Index	23 (1.7)	24 (2.0)
Pain at rest[a]	1.8 (1.7)	–
Knee Outcome Survey	85.1 (11.3)%	98.6 (2.3)%
Incidence of knee giving way	14/20	–
Isometric quadriceps strength[b]	95.5 (14.3)%	98.6 (5.4)%
Incidence of meniscal injury	11/20	–

[a] Numeric pain rating scale.
[b] Ratio of ACL reconstructed/contralateral limb or non-dominant/dominant limb.

some level of discomfort at their surgical knee. Average resting pain, as measured by the NPRS, was 1.8 ± 1.7 on a scale of 0–10 with 10 representing worst pain imaginable. Function, as measured by the KOS was significantly diminished in the ACL reconstructed group ($p < 0.001$) with an average of 85.1 ± 11.3% (15% deficit) as compared to the control group at 98.6 ± 2.3% (<2% deficit). Furthermore, 14/20 reported an incident of giving way during the post-operative period, however, a majority reported this occurred in the first three months post-surgery and not beyond. Quadriceps strength was not significantly different between groups ($p = 0.09$). In the ACL reconstruction group, 19 demonstrated at least 85% isometric quadriceps strength when compared to the unaffected limb, 15 of which had ≥90% (Table 1), and one individual had 54%. Meniscal damage was reported in 11/20 subjects with ACL reconstruction, with one subject reporting a lateral meniscus tear and all others reporting medial meniscus damage. In all cases, meniscal damage was repaired or debrided, depending on the extent of the injury.

Proprioceptive deficits were demonstrated in the ACL reconstructed knees as compared to the non-surgical and control group (dominant) knees ($p < 0.001$) (Table 2). TDPM was significantly higher on the affected (4.8 ± 1.6°) than non-surgical side (3.5 ± 1.3°), indicating that significantly greater knee ROM must occur on the ACL-reconstructed knee before the subject could detect it. Both knees showed higher TDPM than control group knees (2.1 ± 0.7°).

There were site-specific, and side-specific differences in vibration sense between the ACL reconstructed and control group subjects. Larger values meant that more stimulus was required for the subject to perceive the vibration, indicative of a relative hypoesthesia. Significant differences were found at all sites at the knee and ankle on the surgical knee compared to the same site on the control limb (Table 2). Although less extensive, similar differences were found between sites on the surgical knee and the mirror image sites on the contralateral limb (Table 2), but not at the ankle. VPT deficits at the lateral femoral condyle of the surgical knee were greater if comorbidity of meniscal damage was present (25.8 ± 4.3 vs. 20.9 ± 6.3, $p = 0.066$, effect size = 0.94).

A moderate correlation was found between proprioceptive and vibration perception deficits in the individuals with ACL reconstruction. In the ACL reconstruction group, higher (worse) TDPM was associated with higher (worse) VPT at the lateral femoral condyle (Spearman's $\rho = 0.462$, $p = 0.040$) and the lateral malleolus (Spearman's $\rho = 0.449$, $p = 0.047$) (Table 3). These associations were not seen in the control group (lateral femoral condyle $\rho = -0.042$, $p = 0.862$; lateral malleolus $\rho = 0.067$, $p = 0.778$). There were no associations between proprioception and VPT at the other sites in either group ($\rho \geq 0.153$).

Isometric strength ratios were significantly correlated to pain (Spearman's $\rho = -0.589$; $p = 0.006$), but not to function as measured by the KOS (Spearman's $\rho = 0.395$; $p = 0.085$), or with proprioception (Spearman's $\rho = -0.380$; $p = 0.099$) (Table 3). History of meniscal injury did not influence isometric strength ratio ($p = 0.245$). However, when subjects with and

Table 2
Median data with interquartile range for vibration perception threshold[a] and proprioception.

	ACL reconstructed knee	Contralateral knee	Control dominant knee	p value (Effect size)	
				Vs. Contralateral	Vs. Control
Medial femoral condyle	21.8 (8.3)	19.0 (7.3)[b]	18.7 (3.5)	p = 0.002 (0.71)	p = 0.007 (1.0)
Lateral femoral condyle	21.8 (9.1)	19.7 (8.2)[b]	17.9 (3.2)[b]	p = 0.002 (0.69)	p < 0.001 (1.4)
Tibial tuberosity	18.8 (8.3)	14.4 (4.0)[b]	15.0 (4.0)[b]	p < 0.001 (1.0)	p = 0.001 (1.1)
Medial malleolus	13.8 (6.6)	13.5 (4.3)	11.0 (2.0)[b]	p = 0.589 (0.05)	p < 0.001 (1.5)
Lateral malleolus	14.0 (4.3)	13.5 (4.7)	11.0 (3.0)[b]	p = 0.629 (0)	p = 0.004 (1.0)
Proprioception	4.8 (0.7)°	3.4 (1.2)°[b]	2.0 (1.4)[b]	p < 0.001 (0.90)	p < 0.001 (2.4)

[a] Vibration perception threshold (VPT) measured in bioesthesiometer units [25].
[b] Significantly different from ACL reconstructed limb.

Table 3
Correlations between sensory and isometric strength measures.

	Spearman's p	p value
Proprioception correlations		
VPT lateral femoral condyle	0.462	0.040
VPT lateral malleolus	0.449	0.047
Isometric strength		
Pain	−0.589	0.006
Function[a]	0.395	0.085
Proprioception	−0.380	0.099

[a] Measured via Knee Outcome Survey (KOS).

without meniscal damage were analyzed separately, isometric strength ratio was only correlated with pain in the intact meniscus group (Spearman's $\rho = -0.826$; $p = 0.006$). No other associations differed between the two groups.

4. Discussion

A main finding of this study was that individuals with ACL reconstruction demonstrated somatosensory deficits of two distinct modalities, proprioception (TDPM) and vibratory perception at the surgical knee, when compared to both the opposite limb and to knees of age and sex matched controls. This occurred even though time since surgery was greater than a year. Impairment in these sensory modalities is indicative of hypoesthesia, defined as a decreased sensitivity to a specific stimulus [31]. These two sensory modalities are neurophysiologically distinct, and deficits in both may indicate that neurological changes may extend beyond damaged sensory receptors in the torn ligament. A moderate correlation was demonstrated between the two and is suggestive of a singular influence, potentially centrally mediated, causing systematic change in both modalities. Hence, somatosensory changes following ACL injury may, in part, occur due to altered central neural mechanisms. While the research literature regarding proprioceptive and vibration perception deficits in knee patient populations is extensive, the two sensory modalities have not been investigated concurrently in individuals following ACL reconstruction. Furthermore, while strength was correlated to pain, it was not correlated to hypoesthesia, indicating that muscle inhibition and hypoesthesia in this population may be separate processes but both may potentially be related to altered nociceptive mechanisms.

Loss of proprioceptive acuity following ACL injury has been associated with altered joint loading [32], impaired postural control [33] and potentially may predispose an individual to further injury. While several studies have reported proprioceptive deficits following ACL rupture [11,12], fewer studies have examined proprioceptive acuity after reconstructive surgery. Moreover, the conclusions of these studies are conflicting. Bonfim et al. [13] reported proprioceptive deficits in both TDPM and joint reposition sense, while Reider et al. [14] failed to find deficits in either proprioceptive modality, specifically at greater than six months following surgery. Proprioceptive measures in the present study fell within a similar range to the findings of both of these studies, yet support the results of Bonfim and colleagues. Proprioceptive loss following ACL rupture may occur due to a peripheral damage of the neuraxis, i.e., loss of afferent input from sensory fibers embedded in the damaged ligament [4,11]. In fact, a recent study suggested that maintaining the ligament remnant stump during ACL reconstruction may improve postoperative functional outcomes [34], ostensibly through maintenance of neural input from the torn ligament. However, a systematic review found no significant benefit to this method [35], likely due to the fact that proprioception, while mediated through joint, muscle and cutaneous sensory input, is most contingent upon input from the muscle spindle [10]. Deficits in vibration perception, which are not typically attributed to loss of ligament sensory afferents, may support the notion of centrally medicated hypoesthesia.

The present study demonstrated decreased VPT acuity at the affected limb of subjects in the ACL reconstruction group. This means that significantly higher levels of vibration were required to perceive vibratory stimuli on their surgical knee. These vibratory deficits were greater at the lateral femoral condyle when meniscal damage, medial or lateral, had been sustained. Other studies have reported vibratory perceptual deficits in relation to various musculoskeletal conditions, including temporomandibular disorder [36], patellofemoral disorders [19], and knee osteoarthritis [18,20]. Our findings are in contrast to a previous study which examined VPT in a study sample of ACL deficient and reconstructed individuals [37]. No significant differences from control knees were noted in VPT at the knee and ankle in these groups. Future studies which serially capture sensory function longitudinally from time of injury and through the rehabilitative process and beyond are clearly indicated.

Various neurophysiological mechanisms may explain VPT impairments demonstrated in our study. Cutaneous sensory nerves may have been damaged during surgery, however, secondary analysis of ACL reconstruction subjects when sub-grouped by surgical procedure or stimulus site at the knee failed to reveal differences between groups. Alternatively, it is possible that articular injury, via pain or inflammatory mechanisms, may alter somatosensation, particularly in the region of the injury or most pain. Apkarian et al. [38], referred to this as a 'reverse pain gate,' or 'touchgate,' suggesting that pain may inhibit non-nociceptive sensation. Using an experimentally induced pain paradigm in healthy controls, these researchers found inhibition of vibrotactile sensitivity at the site of induced pain [38]. They concluded that perceptual deficits associated with pain may be due to central neural mechanisms rather than impairment at the receptor level. In contrast to the findings of Apkarian et al., we found hypoesthesia in areas outside of the surgical knee, including the ankle and the opposite limb. It is impossible to know whether sensory impairments found in the present study occurred as a result of knee joint damage, or were already present, prior to ACL rupture, and immutable. However, vibratory acuity has been found to improve following a knee OA intervention [18], suggesting that this sensory modality

is mutable. Surprisingly, vibratory deficits were greater at the lateral femoral condyle when meniscal damage, medial or lateral, had been sustained. However in a previous study of individuals with knee OA, higher VPTs were also demonstrated [20]. The functional ramifications of hypoesthesia after knee injury have not been investigated extensively [3], however it is clear that ACL-deficient and ACL-reconstructed populations are prone to further injury [39] and early OA [40]. Future studies which examine hypoesthesia, diminished function and the underlying mechanisms of these deficits would be beneficial.

Evidence to support central neurophysiological changes following ACL injury has expanded in recent years. Courtney et al. [41] demonstrated hyperexcitability of the flexor withdrawal reflex, an established experimental measure of central sensitization, in ACL deficient individuals. Altered somatosensory evoked potentials (SEPs) [12] and functional magnetic resonance imaging (fMRI) in individuals with ACL rupture [42], and following ACL reconstruction [43] have been reported. In general, these studies proposed that loss of afferent input from the damaged ACL causes a somatosensory reorganization, which ultimately, may result in altered motor control. Alternatively, altered nociceptive processing associated with inflammation, may induce alterations in somatosensation. Accordingly, associated meniscal injury may potentially further promote altered nociceptive processing. Our findings of greater VPT deficits when meniscal damage was present would support this notion. Joint injury related hypoesthesia may be associated with altered nociceptive mechanisms even when pain is not a salient factor in the patient presentation. Measures of pain other than resting pain, which was used in the present study, may be more relevant in studies of somatosensation, particularly in study populations, such as individuals with ACL reconstruction, where the potential for OA to develop is present. Accordingly, Courtney et al. [41] demonstrated heightened flexor withdrawal reflex excitability, an indicator of central sensitization, in ACL deficient individuals even though all individuals in the experimental group denied resting pain. Future studies, utilizing a longitudinal design, which examine more specific measures of pain and nociceptive mechanisms may aid in elucidating these mechanisms.

Isometric strength was negatively correlated to the experience of resting pain in the ACL reconstruction group, and interestingly, a particularly strong negative correlation ($\rho = -0.826$) existed in those without history of meniscal damage, meaning that greater quadriceps weakness was related to increased pain. While it is unknown whether knee pain resulted in muscle weakness or the converse, it is likely that minor pain is often discounted in the ACL reconstruction population. Researchers have argued that individuals with ACL reconstruction are often discharged and allowed to return to sport without meeting rehabilitation goals [44]. Thus, prevention and management of pain and inflammation during rehabilitation following ACL reconstruction are likely critical.

4.1. Limitations

The study had several limitations. First, measurement of sensation is based upon subjective perception of what represents a threshold input, thus this outcome can be variable. However, previous studies have demonstrated measures of TDPM and VPT to be reliable [20,27]. A second limitation was that function was measured only via written outcome tool. Future studies using physical measures would be beneficial in determining the functional consequences of altered somatosensory findings. It is unclear whether the somatosensory deficits found in this study were a consequence of the injury, or contributed to the ACL injury. Serial quantitative sensory testing may be beneficial as a component of pre-participation physical, and if injured, pre-surgery, post-surgery, and longitudinally. In addition, it may be valuable to examine the relationship between amount of joint laxity, measured via an arthrometer, and sensory findings. This study utilized a sample of convenience and it may be argued that the experimental group was heterogeneous in terms of surgical intervention. While a limitation of the study, interestingly, sensory deficits were demonstrated regardless of graft type. It is clear that these findings provide the basis for a larger more comprehensive future study.

5. Conclusion

The findings in this study of proprioceptive and vibratory hypoesthesia demonstrate that while structural stabilization may have been achieved with ACL reconstruction, somatosensory deficits may be present. Altered proprioception and VPT may contribute to undesirable outcomes that have been reported in this population including altered joint loading [5], re-rupture [45], and potentially early osteoarthritic changes [2]. Greater appreciation of somatosensation during the rehabilitation process may be beneficial. In addition, rehabilitative interventions that have been demonstrated to modulate central pain mechanisms may also be effective in joint conditions where pain is not the main clinical feature.

We declare that we have no conflicts of interest in the authorship or publication of this contribution.

References

[1] Fremerey RW, Lobenhoffer P, Zeichen J, Skutek M, Bosch U, Tscherne H. Proprioception after rehabilitation and reconstruction in knees with deficiency of the anterior cruciate ligament: a prospective, longitudinal study. J Bone Joint Surg Br 2000;82:801–6.
[2] Lohmander LS, Englund PM, Dahl LL, Roos EM. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. Am J Sports Med 2007;35:1756–69.
[3] Ingersoll CD, Grindstaff TL, Pietrosimone BG, Hart JM. Neuromuscular consequences of anterior cruciate ligament injury. Clin Sports Med 2008;27:383–404 [vii].
[4] Hurley MV. The effects of joint damage on muscle function, proprioception and rehabilitation. Man Ther 1997;2:11–7.
[5] Hosseini A, Van de Velde S, Gill TJ, Li G. Tibiofemoral cartilage contact biomechanics in patients after reconstruction of a ruptured anterior cruciate ligament. J Orthop Res 2012;30:1781–8.
[6] Wojtys EM, Huston LJ. Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. Am J Sports Med 1994;22:89–104.
[7] Hatton AL, Crossley KM, Clark RA, Whitehead TS, Morris HG, Culvenor AG. Between-leg differences in challenging single-limb balance performance one year following anterior cruciate ligament reconstruction. Gait Posture 2017;52:22–5.

[8] Keays SL, Newcombe PA, Bullock-Saxton JE, Bullock MI, Keays AC. Factors involved in the development of osteoarthritis after anterior cruciate ligament surgery. Am J Sports Med 2010;38:455–63.
[9] Oiestad BE, Holm I, Gunderson R, Myklebust G, Risberg MA. Quadriceps muscle weakness after anterior cruciate ligament reconstruction: a risk factor for knee osteoarthritis? Arthritis Care Res (Hoboken) 2010;62:1706–14.
[10] Proske U, Gandevia SC. The kinaesthetic senses. J Physiol 2009;587:4139–46.
[11] Barrack RL, Skinner HB, Buckley SL. Proprioception in the anterior cruciate deficient knee. Am J Sports Med 1989;17:1–6.
[12] Courtney C, Rine RM, Kroll P. Central somatosensory changes and altered muscle synergies in subjects with anterior cruciate ligament deficiency. Gait Posture 2005;22:69–74.
[13] Bonfim TR, Jansen Paccola CA, Barela JA. Proprioceptive and behavior impairments in individuals with anterior cruciate ligament reconstructed knees. Arch Phys Med Rehabil 2003;84:1217–23.
[14] Reider B, Arcand MA, Diehl LH, Mroczek K, Abulencia A, Stroud CC, et al. Proprioception of the knee before and after anterior cruciate ligament reconstruction. Art Ther 2003;19:2–12.
[15] Roberts D, Friden T, Stomberg A, Lindstrand A, Moritz U. Bilateral proprioceptive defects in patients with a unilateral anterior cruciate ligament reconstruction: a comparison between patients and healthy individuals. J Orthop Res 2000;18:565–71.
[16] Sharma L. Proprioceptive impairment in knee osteoarthritis. Rheum Dis Clin North Am 1999;25:299–314 [vi].
[17] Knoop J, Steultjens MP, van der Leeden M, van der Esch M, Thorstensson CA, Roorda LD, et al. Proprioception in knee osteoarthritis: a narrative review. Osteoarthr Cartil 2011;19:381–8.
[18] Courtney CA, Steffen AD, Fernandez-de-Las-Penas C, Kim J, Chmell SJ. Joint mobilization enhances mechanisms of conditioned pain modulation in individuals with osteoarthritis of the knee. J Orthop Sports Phys Ther 2016;46:168–76.
[19] Jensen R, Hystad T, Kvale A, Baerheim A. Quantitative sensory testing of patients with long lasting patellofemoral pain syndrome. Eur J Pain 2007;11:665–76.
[20] Shakoor N, Agrawal A, Block JA. Reduced lower extremity vibratory perception in osteoarthritis of the knee. Arthritis Rheum 2008;59:117–21.
[21] Moshourab R, Frenzel H, Lechner S, Haseleu J, Begay V, Omerbasic D, et al. Measurement of vibration detection threshold and tactile spatial acuity in human subjects. J Vis Exp 2016(115). https://doi.org/10.3791/52966.
[22] Irrgang JJ, Snyder-Mackler L, Wainner RS, Fu FH, Harner CD. Development of a patient-reported measure of function of the knee. J Bone Joint Surg Am 1998;80:1132–45.
[23] Keizer D, van Wijhe M, Post WJ, Wierda JM. Quantifying allodynia in patients suffering from unilateral neuropathic pain using Von Frey monofilaments. Clin J Pain 2007;23:85–90.
[24] Stohler CS, Kowalski CJ, Lund JP. Muscle pain inhibits cutaneous touch perception. Pain 2001;92:327–33.
[25] von Schlippe M, Fowler CJ, Harland SJ. Cisplatin neurotoxicity in the treatment of metastatic germ cell tumour: time course and prognosis. Br J Cancer 2001;85:823–6.
[26] Hurkmans EJ, van der Esch M, Ostelo RW, Knol D, Dekker J, Steultjens MP. Reproducibility of the measurement of knee joint proprioception in patients with osteoarthritis of the knee. Arthritis Rheum 2007;57:1398–403.
[27] Courtney CA, Rine R, Jenk DT, Collier PD, Waters A. Enhanced proprioceptive acuity at the knee in the competitive athlete. J Orthop Sports Phys Ther 2013;43:422–6.
[28] Wise AK, Gregory JE, Proske U. The effects of muscle conditioning on movement detection thresholds at the human forearm. Brain Res 1996;735:125–30.
[29] Gregory JE, Morgan DL, Proske U. Aftereffects in the responses of cat muscle spindles. J Neurophysiol 1986;56:451–61.
[30] Barrack RL, Skinner HB, Cook SD. Proprioception of the knee joint. Paradoxical effect of training. Am J Phys Med 1984;63:175–81.
[31] International Association for the Study of Pain Proposed Pain Terminology; 2011.
[32] 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.
[33] Howells BE, Ardern CL, Webster KE. Is postural control restored following anterior cruciate ligament reconstruction? A systematic review. Knee Surg Sports Traumatol Arthrosc 2011;19:1168–77.
[34] Bali K, Dhillon MS, Vasistha RK, Kakkar N, Chana R, Prabhakar S. Efficacy of immunohistological methods in detecting functionally viable mechanoreceptors in the remnant stumps of injured anterior cruciate ligaments and its clinical importance. Knee Surg Sports Traumatol Arthrosc 2012;20:75–80.
[35] Song GY, Zhang H, Zhang J, Li X, Chen XZ, Li Y, et al. The anterior cruciate ligament remnant: to leave it or not? Art Ther 2013;29:1253–62.
[36] Hollins M, Sigurdsson A, Fillingim L, Goble AK. Vibrotactile threshold is elevated in temporomandibular disorders. Pain 1996;67:89–96.
[37] Thorlund JB, Shakoor N, Ageberg E, Sandal LF, Block JA, Roos EM. Vibratory perception threshold in young and middle-aged patients at high risk of knee osteoarthritis compared to controls. Arthritis Care Res (Hoboken) 2012;64:144–8.
[38] Apkarian AV, Stea RA, Bolanowski SJ. Heat-induced pain diminishes vibrotactile perception: a touch gate. Somatosens Mot Res 1994;11:259–67.
[39] Nagelli CV, Hewett TE. Should return to sport be delayed until 2 years after anterior cruciate ligament reconstruction? Biological and functional considerations. Sports Med 2017;47:221–32.
[40] Culvenor AG, Collins NJ, Guermazi A, Cook JL, Vicenzino B, Khan KM, et al. Early knee osteoarthritis is evident one year following anterior cruciate ligament reconstruction: a magnetic resonance imaging evaluation. Arthritis Rheumatol 2015;67:946–55.
[41] Courtney CA, Durr RK, Emerson-Kavchak AJ, Witte EO, Santos MJ. Heightened flexor withdrawal responses following ACL rupture are enhanced by passive tibial translation. Clin Neurophysiol 2011;122:1005–10.
[42] Kapreli E, Athanasopoulos S, Gliatis J, Papathanasiou M, Peeters R, Strimpakos N, et al. Anterior cruciate ligament deficiency causes brain plasticity: a functional MRI study. Am J Sports Med 2009;37:2419–26.
[43] Grooms DR, Page SJ, Nichols-Larsen DS, Chaudhari AM, White SE, Onate JA. Neuroplasticity associated with anterior cruciate ligament reconstruction. J Orthop Sports Phys Ther 2017;47:180–9.
[44] Herrington L, Myer G, Horsley I. Task based rehabilitation protocol for elite athletes following anterior cruciate ligament reconstruction: a clinical commentary. Phys Ther Sport 2013;14:188–98.
[45] Wiggins AJ, Grandhi RK, Schneider DK, Stanfield D, Webster KE, Myer GD. Risk of secondary injury in younger athletes after anterior cruciate ligament reconstruction: a systematic review and meta-analysis. Am J Sports Med 2016;44:1861–76.