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Original Research

Monitoring Cortical and Neuromuscular Activity: Six-month Insights into Knee Joint Position Sense Following ACL Reconstruction

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Background

Changes in cortical activation patterns after rupture of the anterior cruciate ligament (ACL) have been described. However, evidence of these consequences in the early stages following the incident and through longitudinal monitoring is scarce. Further insights could prove valuable in informing evidence-based rehabilitation practices.

Purpose

To analyze the angular accuracy, neuromuscular, and cortical activity during a knee joint position sense (JPS) test over the initial six months following ACL reconstruction.
Study design: Cohort Study

Methods

Twenty participants with ACL reconstruction performed a JPS test with both limbs. The measurement time points were approximately 1.5, 3-4 and 6 months after surgery, while 20 healthy controls were examined on a single occasion. The active JPS test was performed seated with a target angle of 50° for two blocks of continuous angular reproduction (three minutes per block). The reproduced angles were recorded simultaneously by an electrogoniometer. Neuromuscular activity of the quadriceps muscles during extension to the target angle was measured with surface electromyography. Spectral power for theta, alpha-2, beta-1 and beta-2 frequency bands were determined from electroencephalographic recordings. Linear mixed models were performed with group (ACL or controls), the measurement time point, and respective limb as fixed effect and each grouping per subject combination as random effect with random intercept.

Results

Significantly higher beta-2 power over the frontal region of interest was observed at the first measurement time point in the non-involved limb of the ACL group in comparison to the control group ($p = 0.03$). Despite individual variation, no other statistically significant differences were identified for JPS error, neuromuscular, or other cortical activity.

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Conclusion

Variation in cortical activity between the ACL and control group were present, which is consistent with published results in later stages of rehabilitation. Both indicate the importance of a neuromuscular and neurocognitive focus in the rehabilitation.

Level of Evidence

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INTRODUCTION

An anterior cruciate ligament (ACL) rupture represents a significant and long-lasting sports injury. Surgical reconstruction is frequently selected as a means of restoring joint stability.[1] Nonetheless, neither reconstruction nor rehabilitation can fully restore knee function.[2] It has been stated that the ACL contains mechanoreceptors that are responsible for detecting position, movement, and force within the joint.[3] These contribute to the proprioceptive information that is provided for the central nervous system (CNS) in order to maintain function.[4,5] An evaluation of these proprioceptive senses is the joint position sense (JPS) test, which assesses the ability to reproduce actively or passively a previously presented angle by a body segment.[6] Systematic reviews and meta-analyses have demonstrated tendencies of greater knee JPS errors after ACL rupture in the injured limb compared to the non-injured side as well as to healthy controls,[7-10] however, the clinical significance of these errors is still a matter of debate.[11] In addition to changes in proprioception, sensory dysfunction can result in deficits in balance, coordination, and joint stability.[12,13] Neuromuscular activity, which encompasses both voluntary and involuntary muscle activation for the purpose of performing functional tasks and maintaining or restoring joint stability, can be quantified through the use of surface electromyography (EMG).[4] In patients with anterior cruciate ligament (ACL) injuries, there is evidence that neuromuscular activity is impaired.[14] Thus, proprioceptive changes might translate to errors in the coordination of movement, potentially leading to an elevated secondary injury risk.[11, 15]

The loss of mechanoreceptors resulting from an ACL rupture is likely to impact the adaptability of the CNS in regulating joint function.[15,16] Consequently, studies have examined brain activation following ACL rupture during a knee JPS test. In their functional magnetic resonance imaging (fMRI) study, Strong et al.[17] reported no significant differences in JPS error or brain response in the ACL group, with an average follow-up of 23 months post-surgery, compared to a healthy control group. However, greater JPS errors were significantly associated with higher brain activity in some brain regions (ipsilateral anterior cingulate, supramarginal gyrus and insula) in both groups.[17] Baumeister et al.[18] examined brain activity via electroencephalography (EEG) during a JPS test in ACL patients with an average follow-up of 12.5 months after reconstruction. The authors observed a higher JPS error in the ACL injured limb in comparison to the healthy controls, accompanied by a significantly higher theta-power recorded by the electrodes over the frontal cortical region of interest and a significantly higher alpha-2 power in the electrodes over the parietal cortical region of interest.[18] Additionally, they identified significant differences in brain activity between the contralateral limb and healthy controls. Moreover, a review article discusses that bilateral alterations in other tasks may be attributable to the functional reorganization of motor networks, which is not confined to somatosensory and mechanical deficiencies subsequent to unilateral injury.[15]

The aforementioned studies have only investigated participants with an ACL rupture and reconstruction cross-sectionally after completion of the standard rehabilitation programme. This leaves uncertainty regarding early adaptations and potential time points for specified interventions during the rehabilitation phase.[16,19] Therefore, the purpose of this investigation was to analyze the angular accuracy, neuromuscular, and cortical activity during a knee joint position sense (JPS) test over the initial six months following ACL reconstruction.

The hypothesis was that no significant proprioceptive deficiencies measured by the angular error would be detectable between the groups, measurement time points, and limbs[10] Although the performance outcome may remain unchanged, alterations in the underlying neuromuscular and cortical activity were assumed. It was hypothesized that there would be differences in the neuromuscular activity during the extension to the target angle.[20] Furthermore, higher theta frequency was expected in the frontal region of interest and lower alpha-2 power in the parietal region of interest. Conversely, no differences in the beta frequency were anticipated in the ACL group compared to the healthy control group, regardless of the region of interest.[18]

METHODS

PARTICIPANTS

In this prospective observational study, 20 participants with reconstructed ACL were recruited in collaboration with their corresponding orthopaedic surgeon between March 2022 and September 2023. Additionally, 20 healthy controls participated voluntarily. The participants were matched according to sex, age, height, weight and leg dominance. The subsequent analyses were conducted on a single leg of the healthy control group. This was identified as the leg corresponding to the involved limb of the ACL participant, representing the matched involved leg. General inclusion criteria were: age between 18 and 50 years, physically active (at least 2x 45 min per week[21]) currently or before the ACL rupture, and no former knee pathology. The ACL patient group was required to have a clinically

confirmed ACL rupture and have undergone reconstructive surgery with a quadriceps tendon graft within eight weeks of the incidence. No restriction regarding concomitant injuries was set. General exclusion criteria were: cardiac, neurological or peripheral vascular diseases, acute infection, alcohol abuse, current pain medication, other injuries of the lower extremity or trunk, back pain, thrombosis, pregnancy, dementia, or other musculoskeletal disorders limiting successful execution of the test protocol. An a priori sample size calculation was not feasible, due to missing data in a previous publication. Nonetheless, based on findings of previous studies examining EEG after ACL rupture, it was estimated that a sample size of 20 participants per group would be required.[18,22] The study complied with the criteria of the declaration of Helsinki and was ethically approved by the local legal authority Kantonale Ethikkommission für die Forschung (KEK Bern, CH, No. 2020-02200). All participants gave their informed consent before participation. The study was pre-registered at the German Clinical Trials Register (DRKS-ID: 00023002).

PROCEDURE

The ACL group was invited to participate in a series of measurements at the Bern Movement Lab (Bern University of Applied Sciences, Bern, Switzerland) at three defined time points: 1.5 months, 3-4 months and 6 months following the reconstructive surgery. The healthy control group was invited to participate in a single measurement session. Following clarification of the inclusion criteria, anthropometric data, leg dominance, the side of the ACL rupture and reconstruction and any concomitant injuries of the ACL group were recorded. Additionally, the Tegner activity score (TAS)[23] (before the injury in the ACL group) and the current Knee Osteoarthritis Outcome Score (KOOS)[24] were obtained at the first measurement. The ACL group reported if they received preoperative rehabilitation and provided details of their current main rehabilitation program content and the amount of rehabilitation they received at each measurement time point.

Next, the participants were prepared for the JPS test in the knee with recordings of the cortical activity using electroencephalography (EEG; see EEG recording and processing section), neuromuscular activity using electromyography (EMG; see EMG recording and processing section) as well as the angular error using an electrogoniometer (see electrogoniometer recording and processing). The procedure of the active-active JPS test as well as the recording and processing of the neuromuscular activity and angular deviation has been described in detail elsewhere.[25] In short, following preparation, the participants' actual general well-being and pain levels were assessed using a visual analogue scale with 100 mm in length.[26] Then, the participants completed five minutes of level walking on a treadmill (Quasar® med, h/p/cosomos sports & medical GmbH, Nussdorf-Traunstein, Germany) at a speed of 3 $km*h^{-1}$ as a standardized warm-up with additional recording of the neuromuscular activity during the final minute for later submaximal normalization of the EMG signals.[27] Subsequently, the resting EEG activity during the period of seated rest with the eyes open was recorded for three minutes and later used for the normalization of the EEG signals.[28] Following this, the active-active JPS test was performed. Participants were seated with a hip angle of 100° flexion and a gap of approx. 5 cm between the posterior aspect of the knee and the chair surface, in an open kinetic chain (Figure 1).

Figure 1. Study setup of the active knee joint position sense test.

During familiarization with the task, the participants actively flexed their knee into the starting position of 90° knee flexion (0° = full extension) and extended it to the target angle of 50° knee flexion (movement range of 40°) as indicated by the instruction of the examiner and visual feedback of the knee angle on a screen.[25] While the vision of the participants towards their leg was obstructed by a hanging curtain throughout the five familiarization trials, they were allowed to use the feedback displayed on a screen. During the actual testing, the visual feedback was no longer available. The testing phase comprised two blocks of 3-minute-long active continuous angular reproduction per leg at a self-selected pace. Between the blocks, a 3-minute rest was taken and the starting leg was randomised for each measurement time point. The participants were instructed to reproduce the target angle as accurately as possible, hold it for three seconds and then return to the starting position (90° flexion).[25] During the execution of the JPS test, the angular changes and neuromuscular and cortical electrical activity were recorded.

ELECTROGONIOMETER RECORDING AND PROCESSING

The electrogoniometer (Potentiometer RP20, Megatron Elektronik GmbH & Co.KG, Munich, Germany) was attached with the center of rotation placed at the knee joint

space, in the midline between the lateral femoral and tibial epicondyle of the participant's leg. The goniometer arms were affixed with Velcro strips in a superior/inferior orientation, aligned proximally with the greater trochanter and distally with the lateral malleolus. The electrogoniometer data were captured at a rate of 4000 Hz and then underwent analogue-to-digital conversion (NI PCI 6255 device from National Instruments®, Austin, USA; 1.25 MS/s, 16 bits). Subsequently, the signals were recorded ustilizing LabVIEW®-based software Imago Record (Pfitec®, Endingen, Germany). The electrogoniometer data were processed using Imago Process Master (Pfitec®, Endingen, Germany). The angle reproduced for each trial was determined as the midpoint of the 3-second holding phase, in accordance with the specified procedure. These angles were then exported to a Microsoft® Excel spreadsheet (Windows 10, Microsoft Corporation, Redmond, WA, USA). The angular error, defined as the difference between the targeted and reproduced angles, was calculated for each trial. Moreover, the constant angular error (CE) and absolute angular error (AE) were computed.[29] The CE quantifies the directional bias of the error, with positive or negative arithmetical differences considered in the calculation. Negative arithmetic differences indicate an underestimation of the reproduced angle in comparison to the targeted angle, which is characterised by lesser knee extension. Conversely, positive values signify an overestimation, indicating greater knee extension. Additionally, the variable error (VE) was calculated in order to assess the consistency of the constant error.[30]

NEUROMUSCULAR RECORDING AND PROCESSING

For the surface EMG measurements, the electrodes were positioned meticulously on the vastus medialis (VM), vastus lateralis (VL), and rectus femoris (RF) of both limbs, adhering to the guidelines provided by SENIAM.[31] Prior to electrode placement, the skin was prepared by shaving, sandpaper abrasion, and alcohol cleaning in order to optimize muscle signal detection. Bipolar electrodes (Type P-00-S, Blue Sensor®, Ambu, Ballerup, Denmark, interelectrode distance: 20 mm) were utilized to ensure that interelectrode impedance remained below 2 kΩ (Impedance meter: D175, Digitimer®, Hertfordshire, UK). The data acquisition methodology was consistent with that described previously. The neuromuscular activity during the JPS test was analyzed in the extension movement phases based on angular recordings obtained from the electrogoniometer. The extension phase spanned from the commencement of the extension until reaching the anticipated target angle. EMG data underwent processing using the Imago Process Master (Pfitec®, Endingen, Germany). The raw signals were subjected to full-wave rectification and band-pass filtering within the range of 10–500 Hz (Butterworth, 2nd order). The root mean squares (RMS) of the amplitudes were computed for each muscle and movement phase and exported to a Microsoft® Excel spreadsheet (Windows 10, Microsoft Corporation, Redmond, WA, USA). The submaximal normalization of neuromuscular activity involved utilising the mean gait cycle activity of each muscle during level walking.[27]

ELECTROENCEPHALOGRAPHY RECORDING AND PROCESSING

Cortical electrical activity was recorded continuously throughout each block of JPS testing using a dry electroencephalography-system (DSI-24, Wearable Sensing, San Diego, CA, USA) and the corresponding software (DSI-STREAMER–V.1.08.44), operating at a sampling rate of 300 Hz. The dry-EEG headset comprised 19 electrodes arranged in accordance with the international 10:20 system across the scalp and with two electrodes positioned at the ear lobules. The electrodes were mounted and secured following the instructions provided in the device manual to ensure optimal scalp contact.[32] The impedances of each electrode were carefully monitored to maintain levels below 5kΩ. The recorded cortical activity signals were processed in MATLAB (Version R2020a, Mathworks Inc., Natick, MA, USA) utilising the EEGLAB toolbox (eeglab2022.1),[33] following a standardized pipeline. Initially, the two blocks of JPS test per leg were combined. To mitigate line noise, the CleanLine software plugin[34] was employed. Subsequently, the cortical activity signals were band-pass filtered within the range of 1 to 30 Hz. An "Artifact Subspace Reconstruction bad burst correction"[35] was implemented to rectify any erroneous data periods. Following manual identification and rejection of undesirable episodes or channels, an adaptive mixture independent component analysis (AMICA)[36] was conducted to discern and eliminate non-brain signals originating from muscle activity, eye movements, or electrocardiogram interference. The EEG data were then re-referenced to a common average, with the reference channel Pz reinstated in the dataset. Power spectra for the following frequency bands – theta (4-7.5 Hz), alpha-1 (8-10 Hz), alpha-2 (10.5-12.5 Hz), beta-1 (13-18 Hz), and beta-2 (18.5-25 Hz) – were computed using Fast-Fourier-Transformation and extracted for each trial and participant. Regions of interest (ROI) were formed for the frontal (F3, Fz, F4), central (C3, Cz, C4), and parietal (P3, Pz, P4) brain areas, as per current literature.[18,28] The spectral power values per ROI were normalised to the resting cortical activity baseline for further statistical analysis.

STATISTICAL ANALYSIS

Statistical data analysis was carried out using R (R Core Team, Version 4.3.2, 2023).[37] Participants' characteristics were tested for significant group differences using a Mann-Whitney U test and a one-way repeated measures ANOVA to identify any significant differences in the general well-being and pain levels before and after each measurement time point performed. Post-hoc analysis was utilized for multiple comparisons. The electrogoniometer, neuromuscular, and cortical activity data were examined to ensure their plausibility. Individual values exceeding two standard deviations from the overall mean were identified and traced back to the original dataset. A comprehensive examination of the data processing procedures pertaining to these outliers was conducted, and adjustments were made if deviations from standard procedures were identified. Otherwise, they were excluded from the dataset in accordance with the

relevant criteria.[27] The results section presents descriptive statistics as mean, standard deviation (SD) and 95% confidence intervals (95% CI). Inferential statistics were conducted on the independent variable "Group", which consisted of the three factors: group (ACL patients or healthy controls), measurement time point, and whether the task was executed with the involved or non-involved leg. A linear mixed model was fitted to each dependent variable (namely the absolute error of the electrogoniometric data, normalized neuromuscular activity during extension to the target angle and normalised cortical activity for the defined frequency bands per ROI frontal theta, parietal alpha-2 and beta-1 and beta-2 within the frontal, central, and parietal ROI) with Group as the fixed effect and using different random effect structures, that is random intercept and slope per Group-subject combination, random intercept per Group-subject combination and random intercept for both Group and subject. Model comparison using the Akaike information criterion (AIC) indicated that the optimal model was the one with "Group:subject" as a random intercept, thereby allowing each Group-subject combination to have a random intercept. From the fitted model, contrasts of interest were computed, that is, the effects relative to the reference category (matched-involved leg of the healthy control group), with standard errors and 95% CI's including p-value adjustment for multiple comparisons using the Tukey's Honest Significance Difference method. A residual analysis was performed to ascertain the validity of the model assumptions. The R lme4 package[38] was used for model fitting and emmeans package[39] was used for computing contrasts.

RESULTS

PARTICIPANTS

A total of 40 participants were enrolled in this study (20 participants after ACL rupture and reconstruction and 20 healthy matched controls). The initial measurement time point (M1) was 1.5 ± 0.2 months post-surgery, the second (M2) was 3.5 ± 0.3 months post-surgery and the third measurement time point (M3) was 6.1 ± 0.3 months post-surgery. There were no dropouts. Table 1 provides an overview of the participants' characteristics. Half of the ACL participants had a rupture in their dominant leg. Only one participant reported no associated injuries. Four participants received preoperative rehabilitation, and all participants in the ACL group underwent standard rehabilitation procedures following surgery over the course of the six month measurement period. Details on associated injuries and the rehabilitation content and frequency can be found in the supplement. One ACL participant was unable to perform the JPS test at the first measurement time point due to discomfort in the targeted range of motion in the involved limb. Data of one non-involved limb of an ACL participant at the first measurement time point and the second block of the non-involved limb at the third measurement time point were excluded from the analysis due to technical issues. The pre- and post-measurement self-reported well-being scores were comparable between the two groups ($p = 0.39$; Table 1). Significant differences were observed in pain levels between the measurement time points ($p = 0.004$). A post-hoc analysis revealed that the ACL group exhibited significantly elevated pain levels following the first measurement time point when compared to both pre- and post-measurements in the control group ($p = 0.017$ and $p = 0.006$, respectively). The remaining mean pain levels were higher among the ACL group than the control group, though they did not reach a statistically significant difference ($0.22 < p < 1$).

ANGULAR ERROR

The mean number of repetitions performed by the participants was 48 repetitions (overall range of repetitions: 8-99; Con-matched-involved limb: 25-84; ACL-M1-involved limb range: 39-99; ACL-M1-non-involved limb range: 40-77; ACL-M2-involved limb range: 35-75; ACL-M2-non-involved limb range: 22-75; ACL-M3-involved limb range: 27-90; ACL-M3-non-involved limb range: 8-88). The highest mean absolute errors were observed in the control group with (9.1 ± 5.9°), while the lowest was noted in the non-involved leg of the ACL group at M3 (6.3 ± 4.2°). Conversely, the mean constant error was found to be lowest in the control group (4.9 ± 9.7°) and highest in the involved limb of the ACL group at M3 (7.5 ± 6.1°). The mean variable error did not exceed 1°. A comprehensive presentation of the descriptive statistics for the constant, variable and absolute error are presented in Table 2.

The linear mixed model revealed no statistically significant difference in the absolute error in the fixed effects estimates ($0.06 < p < 0.77$, AIC = 33943.0; $R^2 = 0.56$) (Table 3). The residual analysis provided no evidence against model assumptions.

NEUROMUSCULAR ACTIVITY

Descriptive statistics of the normalised neuromuscular activity during the extension to the target angle are displayed in Table 4. Residual analysis revealed no evidence to contradict the model assumptions. Vastus medialis presented no statistical difference in the fixed effect estimates ($0.80 < p < 0.98$; AIC = 52333.5; $R^2 = 0.84$). No statistically significant difference in the fixed effect estimates was found for the vastus lateralis ($0.64 < p < 1$; AIC = 48910.8; $R^2 = 0.85$). The rectus femoris demonstrated no statistically significant difference in the fixed effect estimates ($0.31 < p < 0.99$, AIC = 49927.3; $R^2 = 0.78$) (Table 4).

CORTICAL ELECTRICAL ACTIVITY

Descriptive statistics of the normalized cortical activity over the entire JPS test per frontal, central and parietal region of interest, as well as frequency bands, are displayed in Table 5. Residual analysis demonstrated no evidence against model assumptions. No significant differences were observed in the fixed effects estimates for frontal theta ($0.16 < p < 0.99$; AIC = 3613.4; $R^2 = 0.34$) and frontal beta-1 ($0.06 < p < 0.47$; AIC = 3550.5; $R^2 = 0.35$) (Table 5). A signif-

Table 1. Participant characteristics displayed in mean ± standard deviation.

Characteristic		ACL (n = 20)	Control (n = 20)	p-value
Age, years		26.9 ± 6	28.4 ± 7.6	0.597
Height, cm		172 ± 8.3	173 ± 8.3	0.903
Weight, kg		71.8 ± 12.1	69 ± 11.2	0.516
Female sex, %		40	40	-
(Preinjury) Tegner score (10 points max)		5.3 ± 1	4.6 ± 0.9	0.047
KOOS total score (168 points max)		86.8 ± 24.4	167 ± 1.4	< 0.001
VAS well-being (mm)	M1- PRE	9.05 ± 12.5	7.9 ± 13.6	-
	M1- POST	11.5 ± 14	5.3 ± 9.4	-
	M2 - PRE	13.0 ± 15.1	NA	-
	M2 - POST	10.5 ± 11.4		
	M3 - PRE	7.1 ± 9	NA	-
	M3 - POST	5.8 ± 9		
VAS pain (mm)	M1- PRE	15.2 ± 17.3	3.6 ± 6.7	-
	M1- POST	20.4 ± 17	2.3 ± 5.1	-
	M2 - PRE	13.5 ± 16.5	NA	-
	M2 - POST	14.2 ± 17.7		
	M3 - PRE	11.3 ± 14.8	NA	-
	M3 - PRE	12.5 ± 18.9		

ACL = anterior cruciate ligament group; n = number; KOOS = Knee injury and Osteoarthritis Outcome Score; max = maximum; VAS = visual analogue scale

Table 2. Constant, variable and absolute error of the JPS test per measurement time point and leg.

	Constant error *M ± SD*	Variable error *M ± SD*	Absolute error *M ± SD*
Con – involved	4.9±9.7	1±0.1	9.1±5.9
ACL – M1 – involved	6.6±5.2	1±0.1	7.2±4.3
ACL – M1 – non-involved	7.5±5.6	1±0.1	7.9±4.9
ACL – M2 – involved	6.2±4.7	1±0.1	6.6±4.1
ACL – M2 – non-involved	5.2±6.2	1±0.1	6.5±4.9
ACL – M3 – involved	7.5±6.1	1±0.1	8.1±5.3
ACL – M3 – non-involved	5.6±5.0	1±0.1	6.3±4.2

M = mean; SD = standard deviation; Con = control group; ACL = anterior cruciate ligament group; M1-M3 = measurement time point 1-3

Table 3. Linear mixed model output for the absolute error with Group as fixed effect and Group:subject as random effect with random intercept.

		Absolute Error Model output			
	Beta	SE	Lower 95% CI	Upper 95% CI	p-value
Con – involved	*Reference*				
ACL – M1 – involved	-2.10	1.20	-5.65	1.45	0.31
ACL – M1 – non-involved	-1.20	1.19	-4.73	2.32	0.77
ACL – M2 – involved	-2.94	1.18	-6.42	0.54	0.06
ACL – M2 – non-involved	-2.85	1.18	-6.34	0.63	0.07
ACL – M3 – involved	-1.39	1.21	-4.97	2.18	0.68
ACL – M3 – non-involved	-2.95	1.21	-6.53	0.63	0.07

SE = standard error; CI = confidence interval; Con = control group; ACL = anterior cruciate ligament group; M1-M3 = measurement time point 1-3

Table 4. Normalized neuromuscular activity during extension to the target angle. Descpritive statistic and linear mixed model output with Group as fixed effect and Group:subject as random effect with random intercept.

	Normalised RMS [% subMVC]	Model output				
Vastus medialis	M ± SD	Beta	SE	Lower 95% CI	Upper 95% CI	p-value
Con – involved	48.9±30.3	*Reference*				
ACL – M1 – involved	50.9±26.6	3.27	9.30	-24.1	30.7	0.98
ACL – M1 – non-involved	43.7±28.6	-3.69	9.30	-31.1	23.7	0.98
ACL – M2 – involved	56.2±31.1	8.65	9.18	-18.4	35.7	0.80
ACL – M2 – non-involved	48.4±29.6	3.35	9.18	-23.7	30.4	0.98
ACL – M3 – involved	48.5±29.3	3.68	9.30	-23.7	31.1	0.98
ACL – M3 – non-involved	53.8±33.7	6.20	9.31	-21.2	33.6	0.92
Vastus lateralis						
Con – involved	46.3±26.0	*Reference*				
ACL – M1 – involved	48.4±23.3	0.74	8.03	-22.9	24.4	0.99
ACL – M1 – non-involved	37.7±23.3	-9.57	7.92	-32.9	13.8	0.64
ACL – M2 – involved	56.3±28.3	8.73	7.83	-14.3	31.8	0.70
ACL – M2 – non-involved	47.0±26.7	0.18	7.83	-22.9	23.3	1
ACL – M3 – involved	52.0±22.8	3.01	7.92	-20.3	26.4	0.98
ACL – M3 – non-involved	46.3±24.5	-1.16	7.93	-24.55	22.2	0.99
Rectus femoris						
Con – involved	62.3±32.0	*Reference*				
ACL – M1 – involved	68.3±22.1	4.67	6.79	-15.35	24.67	0.91
ACL – M1 – non-involved	59.5±21.3	-3.68	6.76	-23.62	16.27	0.95
ACL – M2 – involved	76.4±25.7	11.65	6.68	-8.04	31.33	0.31
ACL – M2 – non-involved	70.3±18.0	5.22	6.68	-14.47	24.90	0.88
ACL – M3 – involved	63.3±22.6	-1.14	6.76	-21.08	18.81	0.99
ACL – M3 – non-involved	66.8±23.6	5.26	6.77	-14.70	25.22	0.88

RMS = root mean square; subMVC = submaximal voluntary construction; M = mean; SD = standard deviation; SE = standard error; CI = confidence interval; Con = control group; ACL = anterior cruciate ligament group; M1-M3 = measurement time point 1-3

icantly different fixed effect estimate was found for frontal beta-2 when comparing the ACL-M1-non-involved leg to the control group ($p = 0.03$). The remaining comparison of frontal beta-2 did not yield a statistically significant difference ($0.32 < p < 0.75$, AIC = 3468.2; $R^2 = 0.27$) (Table 5).

No statistically significant differences were detected in the fixed effects estimates for central beta-1 ($0.06 < p < 0.47$; AIC = 3550.5; $R^2 = 0.35$) and central beta-2 ($0.81 < p < 1$, AIC = 3595.9; $R^2 = 0.46$) (Table 5).

Furthermore, no statistically significant differences were evident in the fixed effects estimates for parietal alpha-2 ($0.15 < p < 0.99$; AIC = 3623.3; $R^2 = 0.49$), parietal beta-1 ($0.50 < p < 0.95$, AIC = 3391.1; $R^2 = 0.50$) and parietal beta-2 ($0.62 < p < 0.99$, AIC = 3365.3; $R^2 = 0.54$) (Table 5).

DISCUSSION

This is the first study to examine the angular accuracy, neuromuscular, and cortical activity during a JPS test longitudinally over the first six months following ACL reconstruction. No significant differences were observed between the measurement time points or limb in comparisons to the healthy control group, with the exception of the beta-2 frequency band in the frontal region of interest for the non-involved leg of the ACL group, which demonstrated a difference at the first measurement time point in comparison to the control group.

ANGULAR ERROR

The mean constant error was observed to be the smallest in the control group, although this was accompanied by a high standard deviation and no statistically significant differences when compared to the ACL group. It could be suggested that the mean precision presented by the constant error is marginally better in the control group when compared to the ACL group. Nevertheless, the mean absolute errors were found to be higher in the healthy control group than in the ACL group for each measurement time point and limb, with no significant difference observed. The non-significant difference confirms the authors' hypotheses. Furthermore, a systematic review focusing on the active JPS test among ACL reconstructed participants in the early phase following surgery demonstrated nonhomoge-

Table 5. Normalized cortical activity during joint position sense test. Descpritive statistic and linear mixed model output with Group as fixed effect and Group:subject as random effect with random intercept.

	Normalised EEG [% resting EEG]	Model output				
Frontal Theta	M ± SD	Beta	SE	Lower 95% CI	Upper 95% CI	p-value
Con – involved	93.5±31.5	*Reference*				
ACL – M1 – involved	104.2±30.9	10.74	6.82	-9.7	31.2	0.41
ACL – M1 – non-involved	107.5±25.7	14.34	6.86	-6.2	34.9	0.16
ACL – M2 – involved	105.6±27.4	12.38	7.02	-8.7	33.4	0.31
ACL – M2 – non-involved	103.2±31.4	10.24	6.76	-10.0	30.5	0.45
ACL – M3 – involved	93.3±32.0	-1.67	6.91	-22.4	19.0	0.99
ACL – M3 – non-involved	96.3±28.3	2.50	7.00	-18.5	0.4	0.98
Parietal Alpha-2						
Con – involved	80.9±29.3	*Reference*				
ACL – M1 – involved	80.9±37.5	0.60	9.20	-27.0	28.2	0.99
ACL – M1 – non-involved	78.4±36.2	-1.17	9.22	-28.8	26.4	0.99
ACL – M2 – involved	86.1±28.5	4.72	9.34	-23.3	32.7	0.96
ACL – M2 – non-involved	86.3±27.8	6.19	9.09	-21.0	33.4	0.91
ACL – M3 – involved	92.2±31.5	10.61	9.22	-17.0	38.2	0.68
ACL – M3 – non-involved	91.9±29.3	10.87	9.33	-17.1	38.8	0.67
Frontal Beta-1						
Con – involved	93.1±26.3	*Reference*				
ACL – M1 – involved	105.9±28.3	12.47	6.65	-7.5	23.4	0.25
ACL – M1 – non-involved	110.1±25.1	16.57	6.65	-3.4	36.5	0.06
ACL – M2 – involved	109.8±26.7	15.86	6.75	-4.4	36.1	0.09
ACL – M2 – non-involved	104.9±23.7	11.47	6.56	-8.2	31.1	0.32
ACL – M3 – involved	103.7±32.1	9.77	6.59	-9.9	29.5	0.47
ACL – M3 – non-involved	106.8±28.7	13.28	6.75	-6.9	33.5	0.21
Frontal Beta-2						
Con – involved	91.0±28.3	*Reference*				
ACL – M1 – involved	102.9±33.0	11.38	7.66	-11.6	34.4	0.47
ACL – M1 – non-involved	112.6±30.6	20.92	7.70	-2.2	44.0	**0.03**
ACL – M2 – involved	100.4±29.9	8.03	7.74	-15.2	33.1	0.75
ACL – M2 – non-involved	104.2±33.3	13.04	7.52	-9.5	35.6	0.32
ACL – M3 – involved	101.4±34.8	9.25	7.54	-13.4	31.9	0.63
ACL – M3 – non-involved	99.2±36.9	8.49	7.78	-14.8	31.8	0.72
Central Beta-1						
Con – involved	95.9±24.4	*Reference*				
ACL – M1 – involved	100.4±24.3	5.10	6.22	-13.5	23.8	0.86
ACL – M1 – non-involved	97.9±23.5	2.93	6.26	-15.8	21.7	0.97
ACL – M2 – involved	93.4±20.1	-2.11	6.31	-21.0	16.8	0.98
ACL – M2 – non-involved	95.0±25.5	-0.57	6.14	-19.0	17.8	0.99
ACL – M3 – involved	103.8±27.9	6.57	6.17	-11.9	25.1	0.73
ACL – M3 – non-involved	98.5±23.3	2.49	6.30	-16.4	21.4	0.98
Central Beta-2						
Con – involved	95.6±28.5	*Reference*				
ACL – M1 – involved	95.9±29.9	-0.33	7.85	-23.9	23.2	1
ACL – M1 – non-involved	96.9±33.7	1.32	7.90	-22.3	25.0	0.99
ACL – M2 – involved	89.4±29.2	-7.39	7.93	-31.2	16.4	0.81
ACL – M2 – non-involved	92.5±31.8	-3.92	7.74	-27.1	20.0	0.96

	Normalised EEG [% resting EEG]	Model output				
ACL – M3 – involved	91.6±29.2	-5.60	7.83	-29.1	17.9	0.90
ACL – M3 – non-involved	90.6±26.5	-5.07	7.95	-28.9	18.7	0.93
Parietal Beta-1						
Con – involved	92.2±18.1	*Reference*				
ACL – M1 – involved	99.8±23.2	7.21	5.39	-8.9	23.4	0.56
ACL – M1 – non-involved	98.8±21.3	6.19	5.39	-9.9	22.3	0.68
ACL – M2 – involved	99.4±19.6	6.69	5.48	-9.7	23.1	0.64
ACL – M2 – non-involved	95.6±19.2	3.02	5.33	-12.9	19.0	0.95
ACL – M3 – involved	99.2±20.9	6.55	5.40	-9.6	22.7	0.64
ACL – M3 – non-involved	100.4±20.1	7.82	5.47	-8.5	24.2	0.50
Parietal Beta-2						
Con – involved	88.2±21.1	*Reference*				
ACL – M1 – involved	91.7±18.9	3.02	5.38	-13.1	19.1	0.95
ACL – M1 – non-involved	95.4±22.4	6.71	5.38	-9.4	22.8	0.62
ACL – M2 – involved	90.4±19.4	1.58	5.47	-14.8	18.0	0.99
ACL – M2 – non-involved	91.6±17.7	3.05	5.32	-11.7	20.5	0.94
ACL – M3 – involved	93.1±22.0	4.38	5.38	-14.9	17.7	0.86
ACL – M3 – non-involved	92.8±17.2	4.15	5.46	-12.2	20.5	0.88

EEG = electroencephalography; M = mean; SD = standard deviation; SE = standard error; CI = confidence interval; Con = control group; ACL = anterior cruciate ligament group; M1-M3 = measurement time point 1-3

neous results.[10] Contrary, in the literature, JPS errors have most often been reported to be higher among the ACL patient groups compared to the contralateral side or a healthy control group,[7-9] however, no clear consensus exists.[40]

The psychometric properties of the present study setup were investigated within a healthy control group, and a minimal detectable change of 6.8° was identified.[25] Therefore, presented differences found within the current study are likely to be the result of systematic error and demonstrate no clear influence of the measurement time point or limb in the ACL group. Moreover, it has been proposed that differences should exceed 5° to be considered clinically meaningful.[8,9,11] The absolute errors presented exceed the minimal clinically important difference, yet no significant differences were observed between the ACL group and the healthy control group.

NEUROMUSCULAR ACITVITY

No significant differences were found between the healthy control group and the ACL group at each time point and for each limb. In the literature, only a few studies investigating the neuromuscular activity in the early postoperative period following ACL reconstruction exist. A general reduction in voluntary quadriceps activation has been reported in the literature,[14,20,41] as well as alterations in the neural pathways responsible for this activation.[42] Studies have discussed whether the quadriceps deficits can be attributed to arthrogenic muscle inhibition.[14,43] In the present study, it can be hypothesized that the open-chain and non-weight-bearing JPS task may not have elicited differences in the neuromuscular activity due to the absence of sensory feedback and task functionality.

CORTICAL ELECTRICAL ACTIVITY

A higher mean frontal theta power was observed in both limbs of the ACL group in the first two measurement time points, although this did not reach statistical significance when compared to a healthy control group. Baumeister et al.[18] reported significantly higher theta power over the frontal cortical areas in both limbs of participants on average one year after ACL reconstruction compared to healthy controls.[18] Elevated frontal theta power might represent a higher need for attention during the JPS test execution.[44,45]

No significantly different alpha-2 power was detected in recordings from the electrodes over the parietal region at both limbs of the ACL group in comparison to the healthy control group. The mean alpha-2 power over the parietal region demonstrated a slight increase over the measurement time points in the ACL group. Higher alpha-2 power over parietal cortical brain regions indicates less task-specific demands.[46] It can be hypothesized that the slight increase in alpha-2 power may be influenced by a learning effect with the repeated measures, reflecting a reduction in the need for cortical activation during the task.[46] However, this hypothesis was not supported by the findings of the present study. The study by Baumeister et al.[18] presented a significantly lower alpha-2 power from the parietal electrodes during JPS testing with the involved limb of the ACL group in comparison to healthy controls.[18] Reduced activity within the alpha-2 frequency band in sensorimotor areas has been found to be associated with an increase in movement-related information and sensory processes.[47]

The literature suggests that frontal theta and parietal alpha-2 activities play an important role in the executive functions of working memory.[46,48] The findings of the present study did not reveal any alterations in these executive functions within the ACL group while maintaining performance in the JPS test. It is worth considering, whether the study protocol, which included an open-chain JPS test, creates a sufficiently challenging environment for typically physically active, young participants. Moreover, the primary focus of attention during the execution of a JPS test is internally. It is possible that the ACL group has undergone a process of training whereby they have focused predominantly on their knees and limbs over the past six months of rehabilitation. which could result in a different basis in comparison to a healthy control group. It would be of interest to examine whether similar results would be found if the JPS test was conducted with external visual cues or under dual-task conditions. Herewith, it may be beneficial to investigate attention control in addition to working memory.[49] Furthermore, recent clinical commentaries have recommended adapting ACL rehabilitation to allow for motor re-learning and support neuroplasticity, during which an external focus during the execution of movements is emphasized.[50,51]

Beta oscillations are thought to be involved in top-down processing and sensorimotor integration and are linking different brain regions, including the pre-motor and somatosensory cortex, the supplementary motor areas and the cerebellum.[44,52] The literature indicates that fluctuations in beta oscillations can be divided into movement-based reductions, with a decrease in beta power slightly prior to and during movement, and post-movement beta rebound, which is characterized by an elevation of beta power following movement.[52] It should be noted that the referenced study did not distinguish between beta-1 and beta-2. Apart from frontal beta-2 in one comparison, no other significant results were found in the present study.[51] A detailed analysis regarding the movement phase would provide valuable insights into the differences in movement planning, execution and the integration of sensory information. Moreover, examining cortical activity in repetitions with higher angular error could prove beneficial. One fMRI study reported a significantly positive correlation between angular error and brain activity.[17] Further investigation could clarify how proprioceptive error detection and integration contribute to the execution of precise movements and the association with cortical activities. This could be used to evaluate the high re-injury rates among patients with ACL reconstruction.

It is possible that the statistically significant differences, which were not observed during the initial six month period following ACL reconstruction, may become apparent at a later stage.[14] This is supported by a systematic review, reporting neurocognitive changes and alterations to the central nervous system that persist after athletes return to sport.[53] It is further recommended that a neurocognitive test battery be implemented in the return to sport testing process to enhance the detection of neurocognitive reliance in patients returning to sport following ACL reconstruction.[54] Nevertheless, it remains ambiguous whether differences in cortical activities and functional connectivity precede ACL injury as a cause or emerge as a consequence thereof.[55,56]

METHODOLOGICAL CONSIDERATIONS AND LIMITATIONS

The characteristics of the participants were not significantly different between the groups, with the exception of the (preinjury) Tegner score and the current KOOS total score. Both groups exhibited a mean Tegner score of greater than 4, indicating that they were a physically active cohort in total. Nevertheless, the preinjury Tegner score of the ACL group was slightly higher than that of the healthy control group. The influence of physical activity on proprioceptive capacity is a topic that has been discussed in the literature.[57,58] It is possible that a less active ACL patient group would yield more distinct results.[17] Moreover, the presence of joint effusion and potential pain, particularly at the first measurement time point for the ACL group, may have exerted additional influence on the outcomes. This is, however, unavoidable in measurements taken during the early phase following injury and reconstruction. Nonetheless, it is possible that the observed effects within the ACL group may have been influenced by the rehabilitation process, which was not controlled. A sub-analysis of patients with a higher frequency of rehabilitative appointments in comparison to a lower frequency might provide further insights into the potential effect on the neuromuscular or cortical activity. Nevertheless, the number of patients included in the present study was insufficient for the performance of meaningful sub-analyses. The influence of limb dominance on JPS quality has been the subject of previous research.[25,57,58] A recent fMRI study has proposed the existence of a right hemisphere lateralization of proprioceptive processing test in healthy persons during a JPS.[59] Although the present study included only one participant per group defined as left-footed and ACL patients with equally paired ACL injuries in their non-dominant limb, future research should evaluate the potential influence of limb dominance.

The self-selected pace during testing resulted in different repetitions per execution of the JPS test. The use of a metronome would have ensured greater standardisation of the procedure, although it is possible that this would have resulted in alterations to individual movement preferences. Nonetheless, the range of repetitions was comparable between the two groups and measurement time points, particularly in order to minimize the impact of potential pain or discomfort during the first measurement time points in the involved limb of the ACL group. Moreover, the rather long duration of angular reproduction may have resulted in a decline in motivation or focus. Yet, the variable error was minimal and the numerous repetitions ensured an accurate assessment of angular reproduction performance.[60] As previously stated, the clinical relevance of the JPS test is a topic of ongoing debate.[6,11] The limitation of the test to a single joint does not accurately reflect the planning and execution of movement in functional tasks, which often involve multiple joints and movement directions.[60] Accordingly, a test simulating weight-bearing movement or ob-

stacle clearance test has been proposed.[61,62] However, the feasibility of these tests within a study population in the early stages after surgery, with the measurement of brain activity using EEG, remains unknown and would be subject to further investigation. Moreover, the JPS test enables the explicit measurement of the participant's ability to perceive the position of the knee joint. A comprehensive understanding of the changes observed during the execution of this test may provide valuable insights into the various affected parts of the sensorimotor system. This could offer a differentiated view of the observed alterations during functional tasks and assist in determining the original causes of the changes.

It should be noted that this study is not without limitations. The rehabilitation process of the ACL group was not subject to control, but was documented. While the primary rehabilitation content was largely consistent across the participants, there were also notable differences in terms of frequencies. Furthermore, the degree of independent training varied considerably between participants, depending on their motivation. This may have had an impact on the results and a more structured and balanced rehabilitation scheme may facilitate more effective clinical implications. Nevertheless, the recruited patients represent patients in their regular therapy setting, which lends to a high external validity to the study and corresponds well with procedures in everyday practice. The target angle consisted of solely 50° knee flexion. Despite this angle representing the range of typical daily activities and having been used in previous studies,[63,64] a variation of movement directions and angles would provide more comprehensive insight.[25]

CONCLUSION

No statistically significant differences in acuity, neuromuscular, or cortical activity were observed during the first six months after ACL reconstruction, except for the beta-2 frequency band in the frontal region of interest for the non-involved leg in comparison to the control group at the first measurement time point. Differences might become evident in later phases following ACL reconstruction and neurocognitive testing should be incorporated into the return to sport evaluation process. In an active study population, the JPS test may not align with the requirements of functionality or difficulty, particularly in terms of eliciting observable changes in neuromuscular activity. An extension with visual cues or under dual-task conditions may yield a distinct evaluation.

CONFLICT OF INTEREST

The authors report no conflicts of interest.

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